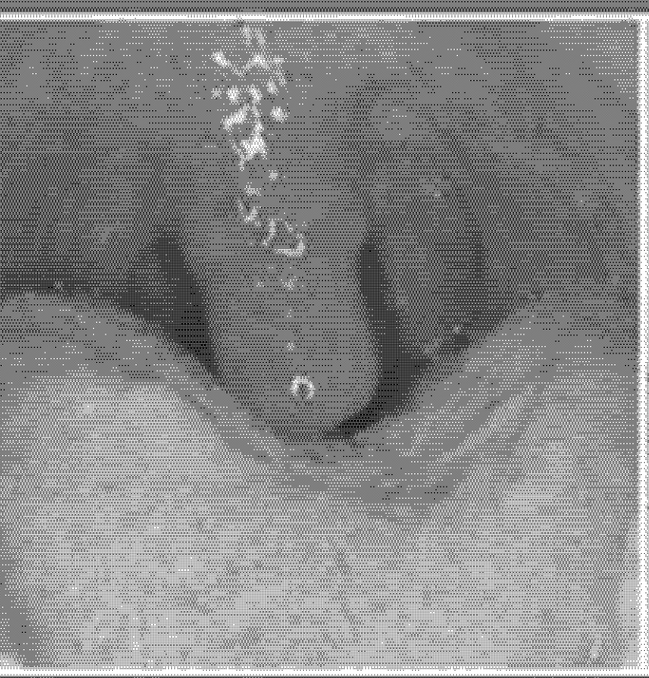
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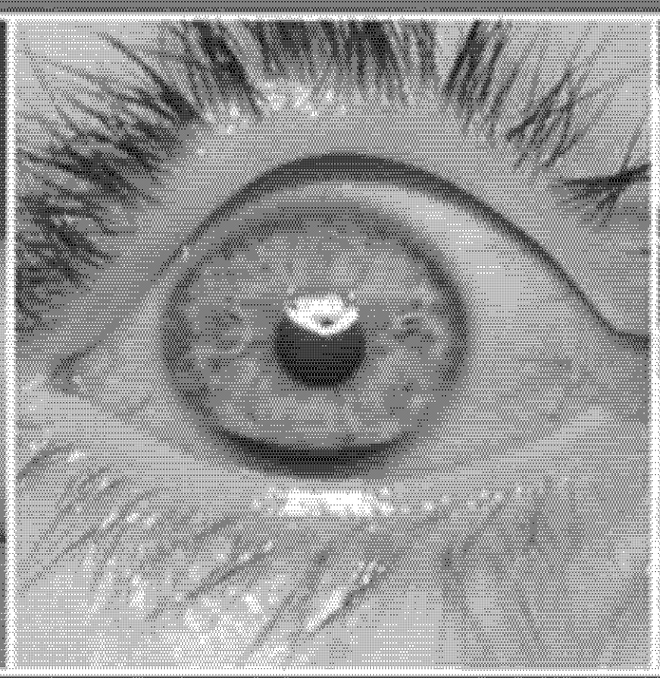
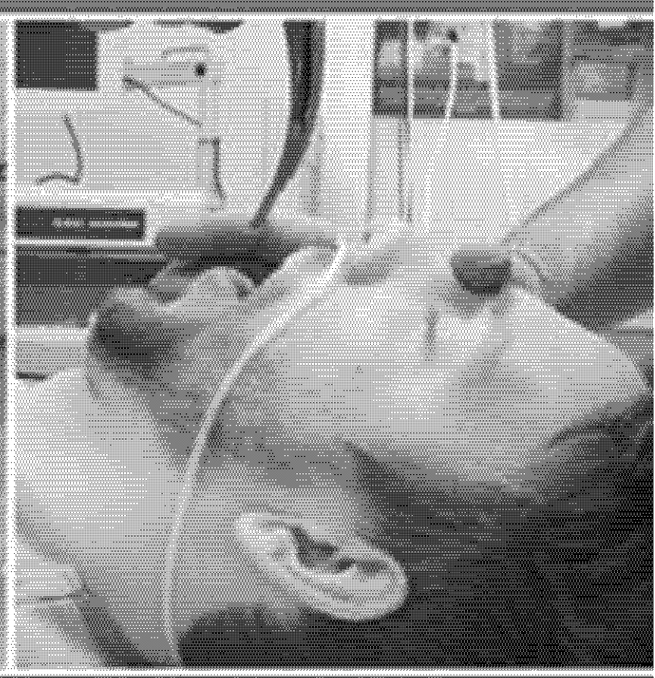
Lawrence B. Stack

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THE ATLAS OF EMERGENCY MEDICINE



Mc Graw Hill Education

4TH EDITION

The Atlas of Emergency Medicine

FOURTH EDITION

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The Atlas of Emergency Medicine, Fourth Edition

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1 2 3 4 5 6 7 8 9 0 DSS/DSS 20 19 18 17 16

ISBN 978-0-07-179725-2
MHID 0-07-179725-4

This book was set in Minion pro by Cenveo® Publisher Services.
The editors were Brian Belval and Christie Naglieri.
The production supervisor was Catherine H. Saggese.
Project management was provided by Raghavi Khullar, Cenveo Publisher Services.
RR Donnelley was the printer and binder.

Library of Congress Cataloging-in-Publication Data

T e atlas of emergency medicine / editors, Kevin J. Knoop, Lawrence B. Stack, Alan B. Storrow, R. Jason T urman.—Fourth edition.
p. ; cm.
Includes index.
ISBN 978-0-07-179725-2 (hardcover : alk. paper)—ISBN 0-07-179725-4 (hardcover : alk. paper)
I. Knoop, Kevin J., editor. II. Stack, Lawrence B., editor. III. Storrow, Alan B., editor. IV. T urman, R. Jason (Robert Jason), editor.
[DNLM: 1. Emergency Medicine—methods—Atlases. 2. Critical Care—methods—Atlases. 3. Emergencies—Atlases. WB 17]
RC86.7
616.02′5—dc23 2015011035

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To MJ, Mimi and Stephen for your love and support of this and all life's endeavors. To the providers who deliver emergency care 24/7 throughout the world for inspiring me to be a lifelong learner. To our patients for sharing their malady for others to learn and then use in improving care.

KJK

To the patients and families who have allowed us to photography their examination findings of themselves and their family members — I pray for God's grace and blessing on you for allowing others to learn from your illness and injuries for the benefit of caring for others.

LBS

To Julia, Robert, and Allison with deep gratitude for your patience, kindness, and love.

ABS

To my wife Lauren, and children Kate and Ben: You are the greatest blessings of my life. To all those who have generously provided mentoring over the years, especially Drs. Knoop, Stack, and Storrow: I am deeply honored and humbled to have had the opportunity to work alongside each of you.

RJT

CONTENTS

Contributors / xv
Media Credits / xvix
Foreword / xxxiii
Preface / xxxv

Part 1 REGIONAL ANATOMY 1

Chapter 1
HEAD AND FACIAL TRAUMA 3

David W. Munter

BASILAR SKULL FRACTURE 4
DEPRESSED SKULL FRACTURE 7
NASAL INJURIES 9
ZYGOMA FRACTURES 11
LEFORT FACIAL FRACTURES 12
ORBITAL WALL FRACTURES 13
MANDIBULAR FRACTURES 16
EXTERNAL EAR INJURIES 19
FRONTAL SINUS FRACTURE 21
TRAUMATIC EXOPHTHALMOS 23
PENETRATING FACIAL TRAUMA 25
UNCAL HERNIATION SYNDROME 26

Chapter 2
OPHTHALMIC CONDITIONS 29

Frank Birinyi ■ Thomas F. Mauger ■ Andrew J. Hendershot

NEONATAL CONJUNCTIVITIS (OPHTHALMIA NEONATORUM) 30
BACTERIAL CONJUNCTIVITIS 32
VIRAL CONJUNCTIVITIS 33
ALLERGIC CONJUNCTIVITIS 35
DACRYOCYSTITIS 36
DACRYOADENITIS 37
PTERYGIUM/PINGUECULA 38
HORDEOLUM/CHALAZION 39
SCLERITIS 41
EPISCLERITIS 42
ACUTE ANGLE-CLOSURE GLAUCOMA 43
ANTERIOR UVEITIS (IRITIS) 45
SYMPATHETIC OPHTHALMIA 47
ANISOCORIA 48
HERPES ZOSTER OPHTHALMICUS 49
OCULAR HERPES SIMPLEX 50
CORNEAL ULCER 52
AFFERENT PUPILLARY DEFECT (MARCUS GUNN PUPIL) 55
HORNER SYNDROME 57
THIRD-NERVE PALSY 58
SIXTH-NERVE PALSY 60

Chapter 3
FUNDUSCOPIC FINDINGS 61

David Effron ■ Beverly C. Forcier ■ Richard E. Wyszynski

NORMAL FUNDUS 62
AGE-RELATED MACULAR DEGENERATION 63
EXUDATE 64
ROTH SPOTS 65
EMBOLI 66
CENTRAL RETINAL ARTERY OCCLUSION 67
CENTRAL RETINAL VEIN OCCLUSION 69
HYPERTENSIVE RETINOPATHY 70
DIABETIC RETINOPATHY 71
VITREOUS HEMORRHAGE 73
RETINAL DETACHMENT 74
CYTOMEGALOVIRUS RETINITIS 75
PAPILLEDEMA 76
OPTIC NEURITIS 77
ANTERIOR ISCHEMIC OPTIC NEUROPATHY 78
GLAUCOMA 79
SUBHYALOID HEMORRHAGE IN SUBARACHNOID HEMORRHAGE 80

Chapter 4
OPHTHALMIC TRAUMA 81

Christopher S. Weaver ■ Kevin J. Knoop

CORNEAL ABRASION 82
SUBCONJUNCTIVAL HEMORRHAGE 84
CORNEAL FOREIGN BODY 85
HYPHEMA 86
INTRAOCULAR FOREIGN BODY 88
IRIDODIALYSIS 89
LENS DISLOCATION 90
PENETRATING GLOBE INJURY 92
GLOBE RUPTURE 94
TRAUMATIC CATARACT 95
EYELID LACERATION 96
CHEMICAL EXPOSURE 98

Chapter 5
EAR, NOSE, AND THROAT CONDITIONS 99

Edward C. Jauch ■ David R. White ■ Kevin J. Knoop

OTITIS MEDIA 100
BULLOUS MYRINGITIS 103
CHOLESTEATOMA 104
TYMPANIC MEMBRANE PERFORATION 105
OTITIS EXTERNA 106
PREAURICULAR SINUS ABSCESS 107
MASTOIDITIS 108
AURICULAR PERICHONDRITIS 109
HERPES ZOSTER OTICUS (RAMSAY HUNT SYNDROME) 111
FACIAL NERVE PALSY 113
ANGIOEDEMA 115
PHARYNGITIS 117
NASAL SEPTAL CONDITIONS 119
NASAL CELLULITIS 120
DIPHTHERIA 122
PERITONSILLAR ABSCESS 123
EPIGLOTTITIS 125
UVULITIS 127
MUCOCELE (RANULA) 128
SIALOADENITIS 129
SINUSITIS 132

Chapter 6
ORAL CONDITIONS 135

Edward C. Jauch ■ James W. Howell

TOOTH SUBLUXATION 136
TOOTH IMPACTION (INTRUSIVE LUXATION) 137
TOOTH AVULSION 138
TOOTH FRACTURES 139
ALVEOLAR RIDGE FRACTURE 141
TEMPORAL MANDIBULAR JOINT DISLOCATION 142
TONGUE LACERATION 144
VERMILION BORDER LIP LACERATION 145

Odontogenic infections 146

GINGIVAL ABSCESS (PERIODONTAL ABSCESS) 146
PERIAPICAL ABSCESS 147
PERICORONAL ABSCESS 149
BUCCAL SPACE ABSCESS 150
CANINE SPACE ABSCESS 151
LUDWIG ANGINA 152
PARAPHARYNGEAL SPACE ABSCESS 153

Oral Conditions 154

TRENCH MOUTH (ACUTE NECROTIZING ULCERATIVE GINGIVITIS) 154
ACID TOOTH EROSION (BULIMIA) 155
THRUSH (ORAL CANDIDIASIS) 156
ORAL HERPES SIMPLEX VIRUS (COLD SORES) 157
APHTHOUS ULCERS (CANKER SORES) 158
STRAWBERRY TONGUE 159
TORUS PALATINUS 160
GINGIVAL HYPERPLASIA 161
BLACK HAIRY TONGUE 162
ORAL CAVITY PIERCING COMPLICATIONS 163

Chapter 7
CHEST AND ABDOMEN 165

Stephen W. Corbett ■ Lawrence B. Stack ■ Kevin J. Knoop

Chest and Abdominal Trauma 166

TRAUMATIC ASPHYXIA 166
FLAIL CHEST 167
STERNOCLAVICULAR DISLOCATION 168
CLAVICLE FRACTURE 169
TENSION PNEUMOTHORAX WITH NEEDLE THORACENTESIS 171
CARDIAC TAMPONADE WITH PERICARDIOCENTESIS 172
EMERGENCY DEPARTMENT THORACOTOMY 173
SEAT BELT INJURY 174
GREY TURNER SIGN AND CULLEN SIGN 175
IMPALED FOREIGN BODY 176
ABDOMINAL EVISCERATION 177
TRAUMATIC ABDOMINAL HERNIA 178
PELVIC FRACTURE 179

Nontraumatic Chest and Abdominal Conditions 180

RESPIRATORY RETRACTIONS 180
SUPERIOR VENA CAVA SYNDROME 181
SUPRACLAVICULAR ADENOPATHY 182
JUGULOVENOUS DISTENSION 183
CAPUT MEDUSAE 184
ABDOMINAL HERNIAS 185

UMBILICAL HERNIA 187
PATENT URACHAL DUCT 188
SISTER MARY JOSEPH NODE (NODULAR UMBILICUS) 189
ABDOMINAL DISTENSION 190
ABDOMINAL WALL HEMATOMA 193
PECTUS DEFORMITIES 195
STOMA PROLAPSE 197
PRUNE BELLY (EAGLE-BARRETT) SYNDROME 198

Chapter 8
UROLOGIC CONDITIONS 201
Jeffery D. Bondesson

TESTICULAR TORSION 202
TORSION OF A TESTICULAR OR EPIDIDYMAL APPENDIX 204
ACUTE EPIDIDYMITIS 205
ORCHITIS 206
HYDROCELE 208
TESTICULAR TUMOR 209
SCROTAL ABSCESS 210
FOURNIER GANGRENE 211
PARAPHIMOSIS 213
PRIAPISM 214
URETHRAL RUPTURE 215
FRACTURE OF THE PENIS 216
PENILE TOURNIQUET 217
STRADDLE INJURY 218
BALANOPOSTHITIS 219
PENILE ZIPPER INJURY 221
VARICOCELE 222

Chapter 9
SEXUALLY TRANSMITTED INFECTIONS AND ANORECTAL CONDITIONS 223
Suzanne Dooley-Hash ■ Brian R. Sharp

Sexually Transmitted Infections 224
SYPHILIS 224
GONORRHEA 227
CHLAMYDIA 229
GENITAL HERPES 230
LYMPHOGRANULOMA VENEREUM 232
CHANCROID 233
CONDYLOMA ACUMINATA (GENITAL WARTS) 234
PEDICULOSIS 236
ANAL FISSURE 237
ANORECTAL ABSCESSES 238
HEMORRHOIDS 240
PROLAPSED RECTUM 243
PILONIDAL ABSCESS 244
RECTAL FOREIGN BODY 245
MELENA 247

Chapter 10
GYNECOLOGIC AND OBSTETRIC CONDITIONS 249
Suzanne Dooley-Hash ■ Sean A. Lisse ■ Kevin J. Knoop

Gynecologic Conditions 250
VAGINITIS 250
CERVICAL POLYPS 252
BARTHOLIN GLAND ABSCESS 253
SPONTANEOUS ABORTION 254
GENITAL TRAUMA AND SEXUAL ASSAULT 255
LICHEN SCLEROSUS 257
LICHEN PLANUS 259
VULVAR CANCER 260
URETHRAL PROLAPSE 261
UTERINE PROLAPSE 262
CYSTOCELE 263
RECTOCELE 264
IMPERFORATE HYMEN 265

Obstetric Conditions 266
ECTOPIC PREGNANCY 266
MOLAR PREGNANCY (HYDATIDIFORM MOLE) 269
FAILED INTRAUTERINE PREGNANCY 270
THIRD-TRIMESTER BLUNT ABDOMINAL TRAUMA 271
EMERGENCY DELIVERY: IMMINENT VERTEX DELIVERY—CROWNING 273
EMERGENCY DELIVERY: NORMAL DELIVERY SEQUENCE 274
BREECH DELIVERY 277
UMBILICAL CORD PROLAPSE IN EMERGENCY DELIVERY 278
NUCHAL CORD IN EMERGENCY DELIVERY 279
SHOULDER DYSTOCIA IN EMERGENCY DELIVERY 280
POSTPARTUM PERINEAL LACERATIONS 281
INTRAUTERINE FETAL DEMISE 283

Chapter 11
EXTREMITY TRAUMA 285

Neha P. Raukar ■ George J. Raukar ■ Daniel L. Savitt

ACROMIOCLAVICULAR JOINT SEPARATION 286
SHOULDER DISLOCATION 288
ELBOW FRACTURES 290
ELBOW DISLOCATION 292
BICEPS TENDON RUPTURE 293
FOREARM FRACTURES 294
FRACTURES OF THE DISTAL RADIUS 296
CARPAL AND CARPOMETACARPAL DISLOCATIONS 298
SCAPHOID FRACTURE 301
CLENCHED FIST INJURY (FIGHT BITE) 302
BOXER'S FRACTURE 303
PERIPHERAL NERVE INJURY 304
BENNETT AND ROLANDO FRACTURES 305
BOUTONNIÈRE AND SWAN-NECK DEFORMITIES 307
HIGH-PRESSURE INJECTION INJURY 308
GAMEKEEPER'S THUMB (SKIER'S THUMB) 309
PHALANGEAL DISLOCATIONS 310
MALLET FINGER AND JERSEY FINGER 312
COMPARTMENT SYNDROME 314
SUBUNGUAL HEMATOMA 316
HIP DISLOCATION 317
HIP FRACTURE 318
KNEE EXTENSOR INJURIES 319
PATELLAR DISLOCATIONS 321
KNEE DISLOCATION 322
FEMUR FRACTURE 323
TIBIAL-FIBULAR FRACTURES 324
FRACTURE BLISTERS 326
ACHILLES TENDON RUPTURE 327
ANKLE DISLOCATION 328
ANKLE FRACTURE 330
ANKLE SPRAIN 332
CALCANEUS FRACTURE 333
FRACTURES OF THE FIFTH METATARSAL BASE 334
LISFRANC FRACTURE-DISLOCATION 336
AMPUTATIONS 338

Chapter 12
EXTREMITY CONDITIONS 341

Robert J. Tubbs ■ Daniel L. Savitt ■ Selim Suner

CELLULITIS 342
FELON 343
GANGRENE 344
GAS GANGRENE (MYONECROSIS) 345
NECROTIZING FASCIITIS 346
CRYSTAL-INDUCED SYNOVITIS (GOUT AND PSEUDOGOUT) 348
INGROWN TOENAIL (ONYCHOCRYPTOSIS) 350
LYMPHANGITIS 351
LYMPHEDEMA 352
OLECRANON AND PREPATELLAR BURSITIS 353
PALMAR SPACE INFECTION 354
TENOSYNOVITIS 355
THROMBOPHLEBITIS 357
PARONYCHIA 358
SUBCLAVIAN VEIN THROMBOSIS 359
CERVICAL RADICULOPATHY 360
DIGITAL CLUBBING 361
PHLEGMASIA DOLENS 362
DEEP VENOUS THROMBOSIS 363
DUPUYTREN CONTRACTURE 365
ACHILLES TENDONITIS 366
GANGLION (SYNOVIAL) CYST 367
RAYNAUD DISEASE 368
ARTERIAL EMBOLUS 369

Chapter 13
CUTANEOUS CONDITIONS 371

J. Matthew Hardin

STEVENS-JOHNSON SYNDROME/TOXIC EPIDERMAL NECROLYSIS 372
ERYTHEMA MULTIFORME 374
DRUG ERUPTIONS 376
FIXED DRUG ERUPTION 377
DISSECTING CELLULITIS OF THE SCALP 378
HIDRADENITIS SUPPURATIVA 379
PYOGENIC GRANULOMA 380
PYODERMA GANGRENOSUM 382
SEBORRHEIC DERMATITIS 383
PSORIASIS 384
PITYRIASIS ROSEA 386
ATOPIC DERMATITIS 388
NUMMULAR/XEROTIC ECZEMA 390
DYSHIDROTIC ECZEMA 391
ID REACTION (DISSEMINATED ECZEMA) 392
STASIS DERMATITIS 393
LIVEDO RETICULARIS 394

VASCULITIS (LEUKOCYTOCLASTIC VASCULITIS) 396
IDIOPATHIC THROMBOCYTOPENIC PURPURA 398
THROMBOTIC THROMBOCYTOPENIC PURPURA 399
URTICARIA AND DERMATOGRAPHISM 400
ALLERGIC CONTACT DERMATITIS 402
ERYTHEMA NODOSUM 403
ERYSIPELAS 404
INFECTIVE ENDOCARDITIS 405
ERYTHEMA CHRONICUM MIGRANS 407
ROCKY MOUNTAIN SPOTTED FEVER 408
DISSEMINATED GONOCOCCUS 409
HOT TUB FOLLICULITIS/PSEUDOMONAS HOT-FOOT SYNDROME 410
ECTHYMA GANGRENOSUM 411
NONGENITAL HERPES SIMPLEX INFECTIONS 412
HERPES ZOSTER 413
DERMATOPHYTE INFECTIONS 414
TINEA (PITYRIASIS) VERSICOLOR 416
ONYCHOMYCOSIS 417
INTERTRIGO 418
SCABIES 419
JAUNDICE 420
MELASMA 421
ABDOMINAL STRIAE (STRIAE ATROPHICAE) 422
VITILIGO 423
UREMIC FROST 424
AUTOIMMUNE BULLOUS DISEASES 425
SYSTEMIC LUPUS ERYTHEMATOSUS 426
SWEET SYNDROME 427
PORPHYRIA CUTANEA TARDA 428
SQUAMOUS CELL CARCINOMA 429
BASAL CELL CARCINOMA 430
MELANOMA 431
LIP LICKER'S DERMATITIS 432

Part 2 SPECIALTY AREAS 433

Chapter 14
PEDIATRIC CONDITIONS 435

Brad Sobolewski ■ Matthew R. Mittiga ■ Javier A. Gonzalez del Rey

Newborn Conditions 436
ERYTHEMA TOXICUM NEONATORUM 436
SALMON PATCHES (NEVUS SIMPLEX) 437
NEONATAL JAUNDICE 438
NEONATAL MILK PRODUCTION (WITCH'S MILK) 439
NEONATAL MASTITIS 440
UMBILICAL GRANULOMA 441
HYPERTROPHIC PYLORIC STENOSIS 442
INTESTINAL MALROTATION WITH VOLVULUS 444

Rashes and Lesions 446
ERYTHEMA INFECTIOSUM (FIFTH DISEASE) 446
ROSEOLA INFANTUM (EXTHANTHEM SUBITUM) 447
IMPETIGO 448
MEASLES 449
VARICELLA (CHICKENPOX) 450
HERPES ZOSTER 451
HAND, FOOT, AND MOUTH SYNDROME 452
COLD PANNICULITIS (POPSICLE PANNICULITIS) 453
HERPETIC GINGIVOSTOMATITIS 454
MENINGOCOCCEMIA 455
URTICARIA 456
SUMMER PENILE SYNDROME 457
STAPHYLOCOCCAL SCALDED SKIN SYNDROME 458
SCARLET FEVER (SCARLATINA) 459
BLISTERING DISTAL DACTYLITIS 461
HENOCH-SCHÖNLEIN PURPURA 462
SERUM SICKNESS–LIKE REACTION 463
STRAWBERRY HEMANGIOMA 464
ORBITAL AND PRESEPTAL (PERIORBITAL) CELLULITIS 466
BRANCHIAL CLEFT CYST 469
THYROGLOSSAL DUCT CYST 470
CYSTIC HYGROMA 471
CAT-SCRATCH DISEASE 472

General Conditions 473
EPIGLOTTITIS 473
RETROPHARYNGEAL ABSCESS 475
BUTTON (DISC) BATTERY INGESTION 477
MEMBRANOUS (BACTERIAL) TRACHEITIS 478
NASAL FOREIGN BODY 479
PHYSEAL FRACTURES: SALTER-HARRIS CLASSIFICATION 480
TODDLER'S FRACTURE 481
DACTYLITIS (HAND-FOOT SYNDROME) 482
HAIR TOURNIQUET 483
FAILURE TO THRIVE 484
NURSING BOTTLE CARIES 485
NURSEMAID'S ELBOW (RADIAL HEAD SUBLUXATION) 486
ILEOCOLIC INTUSSUSCEPTION 488

INGUINAL HERNIA 490
PINWORM INFECTION (ENTEROBIASIS) 492
LICE 493
TRICHOTILLOMANIA 494
OCULOGYRIC CRISIS 495
SETTING-SUN PHENOMENON (SUNDOWNING) 496
KAWASAKI DISEASE 497

Chapter 15
CHILD ABUSE 501
Kathi L. Makoroff ■ Daniel M. Lindberg ■ Marguerite M. Care

Physical Abuse 502
CUTANEOUS FINDINGS 502
CRANIAL FINDINGS 508
FACIAL FINDINGS 511
SKELETAL FINDINGS 514
VISCERAL FINDINGS 517

Sexual Abuse 519
CHILD SEXUAL ABUSE EXAM AND GENITAL FINDINGS 519
INJURIES AND FINDINGS INDICATIVE OF GENITAL OR ANAL TRAUMA 524
MIMICS OF TRAUMA: STRADDLE INJURY 527
MIMICS OF TRAUMA: TOILET SEAT INJURIES 528
MIMICS OF TRAUMA: URETHRAL PROLAPSE 529
MIMICS OF ERYTHEMA/BRUISING: LICHEN SCLEROSIS 530
MIMICS OF ERYTHEMA/BRUISING: LABIAL ADHESIONS 531
MIMICS OF ERYTHEMA/BRUISING: PERIANAL STREPTOCOCCAL DISEASE 532
MIMICS OF ERYTHEMA/BRUISING: VENOUS POOLING 533

Chapter 16
ENVIRONMENTAL CONDITIONS 535
Ken Zafren ■ R. Jason Thurman ■ Ian D. Jones

HIGH-ALTITUDE RETINAL HEMORRHAGE 536
HIGH-ALTITUDE PULMONARY EDEMA 537
HIGH-ALTITUDE CEREBRAL EDEMA 538
HYPOTHERMIA 539
FROSTBITE 540
PERNIO 542
IMMERSION INJURY (TRENCH FOOT) 543
ULTRAVIOLET RADIATION EXPOSURE 544
LIGHTNING INJURIES 547
LARGE TERRESTRIAL ANIMAL ATTACKS 549
CORAL SNAKE ENVENOMATION 550
PIT VIPER ENVENOMATION 551
TROPICAL SNAKE ENVENOMATIONS 554
GILA MONSTER ENVENOMATION 556
BLACK WIDOW SPIDER ENVENOMATION 557
BROWN RECLUSE SPIDER ENVENOMATION 558
SCORPION ENVENOMATION 560
TICKS 562
HYMENOPTERA ENVENOMATION 564
CATERPILLAR, MITE, AND CENTIPEDE ENVENOMATION 566
MIDDLE EAR SQUEEZE 568
MASK SQUEEZE 569
STINGRAY ENVENOMATION 570
SEA URCHIN ENVENOMATION 572
COELENTERATE ENVENOMATION 574
MARINE DERMATITIS 577
SCORPION FISH STING 578
CONE SNAIL ENVENOMATION 579
SEA SNAKE ENVENOMATION 580
CEPHALOPOD ENVENOMATION 581
ERYSIPELOID 582
TOXICODENDRON AND OTHER PLANT EXPOSURES 583
SPOROTRICHOSIS 586

Chapter 17
TOXICOLOGICAL CONDITIONS 587
Saralyn R. Williams ■ Matthew D. Sztajnkrycer ■ R. Jason Thurman

AMPHETAMINE TOXICITY 588
NEW DESIGNER DRUGS: "BATH SALTS" AND "SPICE" 590
COCAINE TOXICITY 591
ANTICHOLINERGIC (ANTIMUSCARINIC) TOXIDROME 594
CHOLINERGIC TOXIDROME 595
OPIOID TOXICITY 597
ACETAMINOPHEN POISONING 599
SALICYLATE POISONING 600
TOXIC ALCOHOL INGESTION 602

TRICYCLIC ANTIDEPRESSANT POISONING 604
POISONING BY B-BLOCKER AND CALCIUM CHANNEL BLOCKER AGENTS 606
INHALANT ABUSE 608
METHEMOGLOBINEMIA 610
CELLULAR ASPHYXIANTS 612
VANCOMYCIN-INDUCED RED MAN SYNDROME 614
BOTULISM 615
ANTICOAGULANTS 616
CAUSTIC INGESTION 618
HYDROFLUORIC ACID BURNS 620
ARSENIC POISONING 622
IRON POISONING 624
LEAD POISONING 625
MERCURY POISONING 627
MUSHROOM INGESTION 629
CARDIAC GLYCOSIDE PLANT INGESTION 631
HOUSEPLANTS WITH CALCIUM OXALATE CRYSTALS 633
JIMSONWEED INGESTION 634
PEYOTE INGESTION 635
TOXALBUMIN INGESTION 636
LEVAMISOLE-INDUCED VASCULITIS 637

Chapter 18
WOUNDS AND SOFT TISSUE INJURIES 639
Chan W. Park ■ Michael L. Juliano ■ Dana Woodhall

WOUND CLEANING AND IRRIGATION 640
WOUND ASSESSMENT 642
WOUND FOREIGN BODIES 644
TRAUMATIC WOUNDS 646
EAR LACERATIONS 650
LIP LACERATIONS 651
TENDON LACERATIONS 652
ANIMAL BITE WOUNDS 654
FISHHOOK INJURIES 656
SIMPLE WOUND CLOSURES 658
COMPLEX WOUND CLOSURES 661
WOUND CARE COMPLICATIONS 664
BURNS 666
ELECTRICAL INJURY 668
DECUBITUS ULCERS 670

Chapter 19
CLINICAL FORENSIC MEDICINE 671
William S. Smock ■ Lawrence B. Stack

GUNSHOT WOUNDS 672
Pattern Injuries of Domestic Violence, Assault, and Abuse 679
SHARP-FORCE-PATTERN INJURIES 679
BLUNT-FORCE-PATTERN INJURIES 682
STRANGULATION INJURIES 684
THERMAL-PATTERN INJURIES 686

Chapter 20
HIV CONDITIONS 689
Stephen P. Raffanti ■ Anna K. Person

PRIMARY HIV INFECTION 690
IMMUNE RECONSTITUTION INFLAMMATORY SYNDROME (IRIS) 691
ORAL HAIRY LEUKOPLAKIA 692
CANDIDIASIS ASSOCIATED WITH HIV 693
KAPOSI SARCOMA 695
TOXOPLASMA GONDII INFECTION 697
PNEUMOCYSTIS 699
CRYPTOCOCCAL INFECTIONS 700
HISTOPLAMOSIS 702
CYTOMEGALOVIRUS INFECTIONS 703
HERPES ZOSTER 704
EOSINOPHILIC FOLLICULITIS 706
HERPES SIMPLEX VIRUS 707
SCABIES 708
MOLLUSCUM CONTAGIOSUM 710
DERMATOPHYTE (TINEA) INFECTIONS 711
THROMBOCYTOPENIA 712
DRUG REACTIONS 713
ACUTE NECROTIZING ULCERATIVE GINGIVITIS 714

Chapter 21
TROPICAL MEDICINE 715
Seth W. Wright ■ Andrew N. Pfeffer

FREE-LIVING AMEBA INFECTION 716
ANEMIA IN THE TROPICS 717
ANTHRAX 718
ASCARIASIS 720
CHAGAS DISEASE 721

CHOLERA 722
CUTANEOUS LARVA MIGRANS 724
CYSTICERCOSIS 725
DENGUE FEVER 727
DRACUNCULIASIS 729
ELEPHANTIASIS 730
EPIDEMIC MENINGITIS 732
GOITER 733
HYDATID CYST 734
LEECH BITES 735
LEISHMANIASIS 737
LEPROSY 739
LEPTOSPIROSIS 741
MALARIA 742
MUMPS 744
MYCETOMA 745
MYIASIS 747
NONTUBERCULOSIS MYCOBACTERIA INFECTIONS 749
PROTEIN-ENERGY MALNUTRITION—KWASHIORKOR AND MARASMUS 750
RABIES 752
SCHISTOSOMIASIS 753
TETANUS 755
TRACHOMA 757
TRADITIONAL MEDICINE IN THE TROPICS 758
TROPICAL SPOROTRICHOSIS 760
TUBERCULOSIS 761
TUNGIASIS 764

Chapter 22
AIRWAY 765

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AIRWAY MAXIM ONE 766
AIRWAY MAXIM TWO 767
AIRWAY MAXIM THREE 768
AIRWAY MAXIM FOUR 769
AIRWAY MAXIM FIVE 770
LARYNGOSCOPIC OROTRACHEAL INTUBATION 772
BOUGIE-ASSISTED INTUBATION 781
VIDEO-ASSISTED INTUBATION—GLIDESCOPE 784
VIDEO-ASSISTED INTUBATION—STORZ C-MAC 785
VIDEO-ASSISTED INTUBATION—McGRATH MAC EDL 786
LARYNGEAL MASK AIRWAY 787
LIGHTED STYLETS AND WANDS 789
DIGITAL (TACTILE) INTUBATION 792
INTUBATING LARYNGEAL AIRWAY (ILA) 793
KING LARYNGEAL TUBE 795
BLIND NASOTRACHEAL INTUBATION 796
NASAL FLEXIBLE FIBEROPTIC INTUBATION 799
CRICOTHYROTOMY (SELDINGER TECHNIQUE) 801
OPEN CRICOTHYROTOMY 803

Chapter 23
ECG ABNORMALITIES 805

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Part 1: St-T Abnormalities 806
ACUTE ANTERIOR MYOCARDIAL INFARCTION 806
ACUTE INFERIOR MYOCARDIAL INFARCTION 807
ACUTE RIGHT VENTRICULAR MYOCARDIAL INFARCTION 808
ACUTE POSTERIOR MYOCARDIAL INFARCTION 809
LEFT MAIN LESION 810
SGARBOSSA CRITERIA FOR AMI IN SETTING OF LBBB 811
SUBENDOCARDIAL ISCHEMIA 812
HYPERACUTE T WAVES 813
WELLENS WAVES 814
"CEREBRAL" T WAVES 815
EARLY REPOLARIZATION 816
LEFT VENTRICULAR ANEURYSM 817
PERICARDITIS 818

Part 2: Conduction Disturbances 819
FIRST-DEGREE AV BLOCK 819
TYPE 1 SECOND-DEGREE AV BLOCK (MOBITZ I, WENCKEBACH) 820
TYPE 2 SECOND-DEGREE AV BLOCK (MOBITZ II) 821
THIRD-DEGREE (COMPLETE) AV BLOCK 822
QT INTERVAL PROLONGATION 823
RIGHT BUNDLE BRANCH BLOCK 824
LEFT BUNDLE BRANCH BLOCK 825
LEFT ANTERIOR FASCICULAR BLOCK 826
LEFT POSTERIOR FASCICULAR BLOCK 827
TRIFASCICULAR BLOCK 828
ASHMAN PHENOMENON 829

Part 3: Rhythm Disturbances 830
JUNCTIONAL RHYTHM 830

VENTRICULAR RHYTHM 831
PACED RHYTHM 832
ATRIAL FIBRILLATION 833
ATRIAL FLUTTER 834
MULTIFOCAL ATRIAL TACHYCARDIA 835
SUPRAVENTRICULAR TACHYCARDIA (SVT) 836
SVT WITH ABERRANCY 837
VENTRICULAR TACHYCARDIA 838
VENTRICULAR FLUTTER 839
TORSADES DE POINTES 840

Part 4: Structural Abnormalities 841
DEXTROCARDIA 841
LEFT VENTRICULAR HYPERTROPHY 842
RIGHT VENTRICULAR HYPERTROPHY 843
LEFT ATRIAL HYPERTROPHY 844
RIGHT ATRIAL HYPERTROPHY 845
HYPERTROPHIC CARDIOMYOPATHY 846
BRUGADA SYNDROME 847
WOLFF-PARKINSON-WHITE SYNDROME 848

Part 5: ECG Abnormalities of Noncardiac Origin 849
HYPOTHERMIA 849
HYPOKALEMIA 850
HYPERKALEMIA 851
PULMONARY EMBOLISM 853
PERICARDIAL EFFUSION 854
DIGOXIN EFFECT, TOXICITY 855
TRICYCLIC ANTIDEPRESSANT EFFECT 856
LIMB LEAD REVERSAL 857
LOW VOLTAGE 858

Chapter 24
EMERGENCY ULTRASOUND 859
Jeremy S. Boyd ■ Jordan D. Rupp ■ Robinson M. Ferre

EMERGENCY ULTRASOUND: INTRODUCTION 860
TRAUMA ULTRASOUND 863
PERICARDIAL (SUBXIPHOID VIEW) 864
RIGHT UPPER QUADRANT (POUCH OF MORISON) 866
LEFT UPPER QUADRANT (PERISPLENIC VIEW) 868
SUPRAPUBIC 870
THORAX (RIGHT AND LEFT) 873
CARDIAC ULTRASOUND 876
SUBXIPHOID CARDIAC (SUBCOSTAL VIEW) 877
PARASTERNAL LONG-AXIS VIEW (PSLA) 879
PARASTERNAL SHORT-AXIS VIEW (PSSA) 881
APICAL FOUR-CHAMBER VIEW (AP4) 883
SUBXIPHOID LONG-AXIS (IVC) 885
LUNG ULTRASOUND 887
ABDOMINAL AORTA ULTRASOUND 890
DEEP VENOUS THROMBOSIS ULTRASOUND 894
ULTRASOUND-GUIDED RESUSCITATION 897
GALLBLADDER ULTRASOUND 899
RENAL AND BLADDER ULTRASOUND 903
PREGNANCY ULTRASOUND 907
PROCEDURAL GUIDANCE: ULTRASOUND-GUIDED VENOUS ACCESS 911
PROCEDURAL GUIDANCE: THORACENTESIS/PARACENTESIS 914
OCULAR ULTRASOUND 916
SOFT TISSUE ULTRASOUND 920

Chapter 25
MICROSCOPIC FINDINGS 923
Brett Bechtel

URINALYSIS 924
SYNOVIAL FLUID ANALYSIS FOR CRYSTALS 926
GRAM STAIN 928
DARK-FIELD EXAMINATION 929
VAGINAL FLUID WET MOUNT 930
POTASSIUM HYDROXIDE MOUNT 931
STOOL EXAMINATION FOR FECAL LEUKOCYTES 932
SKIN SCRAPING FOR DERMATOSES AND INFESTATIONS 933
CEREBROSPINAL FLUID EXAMINATION: INDIA INK PREPARATION 934
WRIGHT STAIN—THIN SMEAR FOR MALARIA 935
FERNING PATTERN FOR AMNIOTIC FLUID 936
PERIPHERAL BLOOD SMEAR FOR SCHISTOCYTES AND BLAST CELLS 937
TAPE TEST FOR ENTEROBIAS VERMICULARIS EGGS 938
TZANCK PREPARATION 940

INDEX 941

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FOREWORD

Within a given medical specialty, typically two or three major textbooks help define the breadth and depth of a discipline. This Atlas of Emergency Medicine, for the field of emergency care, is one of these defining works. First appearing in 1997, the 4th Edition of the Atlas is now published in multiple foreign languages and available electronically, which makes it universally available and important for patient care across the globe. As many acute diagnoses are made visually, and many physicians are visual learners, the Atlas of Emergency Medicine serves as an outstanding educational and reference tool for the medical student, resident, and particularly the practicing emergency physician. It is a useful adjunct in preparation for the pictorial section of certification tests. This Atlas also serves as a valuable resource for other ambulatory care specialties such as pediatricians, general internists, family physicians, physician assistants, and nurse practitioners, by providing an approach designed by emergency physicians for patients with emergent conditions.

The 4th Edition of the Atlas of Emergency Medicine has been completely revised by the four editors as well as expert emergency physician contributors from across the specialty. It now contains 1500 full color photographs, electrocardiograms, and radiographs, 500 newly chosen for this 4th Edition. The chapters are arranged logically by organ system, special populations, and clinical problems. Environmental conditions, toxicology, electrocardiographic abnormalities, forensic medicine, airway procedures, tropical medicine, microscopic diagnoses, and emergency ultrasound are some of the special sections that greatly expand the usefulness of this Atlas for the practicing clinician caring for patients with emergencies. A clinical summary, management and disposition approaches, and pearls for photographic stimuli of a particular diagnosis make this text a full purpose reference. The impressive Table of Contents and exhaustive Index of the 4th Edition provides a comprehensive ability to access the important photographs, diagrams, and radiographs necessary to quickly help care for the patient presenting with an emergent condition. This expansion of topics and diagnoses has made this textbook a major resource in multiple medical environments for clinicians worldwide.

This 4th edition of the Atlas, for the first time, also provides access to videos to help improve the diagnostic capabilities of the clinician and complement the static photographic images. The editors are to be congratulated for continuing to expand the scope and media of this major work, while continuing to search for better photographic representations of disease processes for this latest edition.

For me personally, this Atlas of Emergency Medicine **4th Edition** represents the incredibly successful germination of an idea started as a conversation between an emergency medicine attending physician and a very talented emergency medicine resident (the senior editor of this Atlas, Dr. Knoop) during an overnight shift in the University of Cincinnati Medical Center emergency department, now over 20 years ago. The critical gap in the Emergency Medicine textbooks of the early 1990's was the lack of a comprehensive and practical pictorial atlas available for real-time diagnosis of patients presenting to emergent care settings. This book has not only bridged this gap, but also now provides the concise diagnostic and treatment information necessary for the emergency physician or other practitioners to manage the patient and carefully disposition them in real time, at the bedside. I believe this important Atlas of Emergency Medicine, now a classic in its 4th Edition, will remain a mainstay for the hospital emergency department and outpatient clinic libraries, as well as have a major presence in the savvy clinician's personal reference collection long into the future.

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PREFACE

The Atlas of Emergency Medicine, 4th Edition Preface
"The life so short, the craft so long to learn"
Hippocrates

We have a passion for improving patient care. Our journey with The Atlas of Emergency Medicine began with superb mentors who instilled in us a drive to become excellent clinicians and educators. We discovered imaging was a powerful tool to take the learner "to the bedside" and establish permanence, in a fashion unlike any other didactic technique. In 1994, much by chance, collegial networking brought three of us together to pursue an aggressive goal of producing the most comprehensive source of high-quality emergency care images available. While there were some initial detractors, our first three editions received widespread praise and have been translated into three foreign languages, flash cards, and electronic versions. We are deeply humbled and honored to present our fourth iteration.

Emergency care is defined by time and the emergency department is the most diverse melting pot of acute conditions in the hospital. Diagnostic accuracy, prognostic prediction, and the treatment pathways rely heavily on visual clues. We desire to maximize this skill for the benefit of our patients. We also strongly believe the visual experience, while sometimes downplayed within the hectic and time-pressured environment of modern medicine, is critical to ideal education. Images can teach faster and with greater impact than many pages of text or hours of lecture.

We continue our pursuit of these goals with a substantially updated, expanded, and improved fourth edition of The Atlas of Emergency Medicine. Nearly all of our changes and additions come from reader suggestions and criticisms, all received with sincere gratitude.

First, we have changed the format to greatly reduce text and allow for more images. Hence, the text is more concise, providing essential information. Each chapter item is now organized into: **Clinical Summary**, including a differential diagnosis where appropriate, followed by **Management and Disposition,** and, finally, **Pearls**. We have, as in the past, endeavored to provide relevant "pearls" representing tips for diagnosis, management, or unique aspects of a condition difficult to find in a typical text.

Second, after extensive review and critique, hundreds of new and replacement images have been added. We have been fortunate to draw upon new sources of photographs available on the Internet or through electronic channels unavailable to us previously. This has substantially strengthened the content and is a reminder of how much the world of media has changed. Gone are the days of manually sorting through thousands of slides or prints. There have been radical changes in the way we access medical knowledge over the past two decades. Regardless of the form, however, an image maintains a potent means to teach and learn.

Third, we are making an initial move into video. Many chapters include links to short videos further elucidating findings and providing a powerful medium to expand upon static images.

The audience for this text is all who provide emergency medical care, including clinicians, educators, residents, nurses, prehospital caregivers, and medical students. Many have also found it extremely useful as a review for written board examinations containing pictorial questions. Other healthcare workers, such as internists, family physicians, pediatricians, nurse practitioners, and physician assistants will find the Atlas a useful guide in identifying and treating many acute conditions, where visual clues significantly guide, improve, and expedite diagnosis as well as treatment.

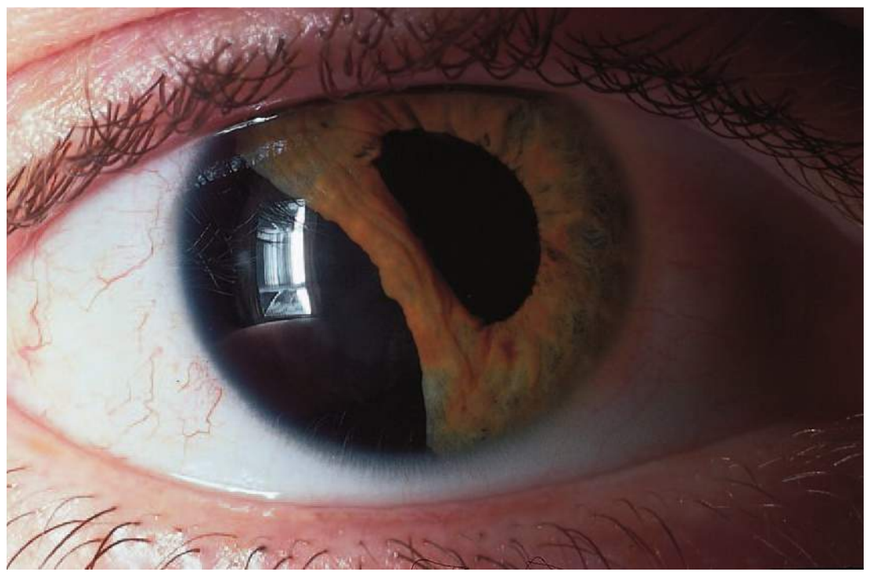
We thank the many contributors and readers who have helped make this edition possible. We are especially grateful for the many contributions from two great educators in emergency medicine who share our passion: David Effron, MD, and Larry Mellick, MD. Also, special thanks to Anne Sydor, PhD, our McGraw-Hill editor for her tireless commitment, organizational ability and focus. Lastly, and most importantly, we express our deepest gratitude to our patients who were willing to be a "great case" in the Atlas, thus ultimately paving the way for improved emergency care.

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PART 1

Regional Anatomy





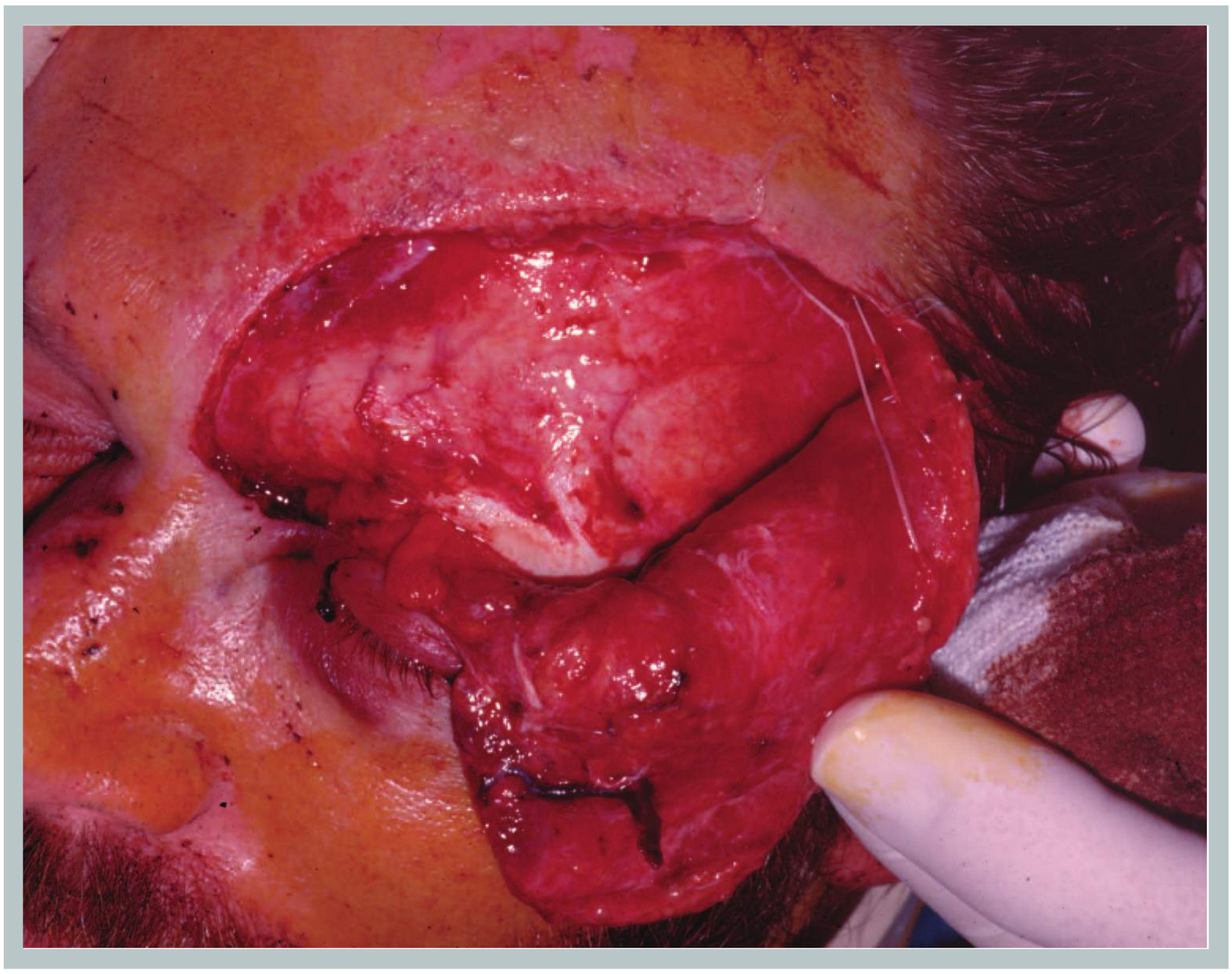


Chapter 1

HEAD AND FACIAL TRAUMA

David W. Munter

Scan QR code or go to www.ematlas.com for video related to chapter



Exposed Galea. Partial forehead degloving injury after a motor vehicle crash. Galea overlying the frontal bone is demonstrated just prior to repair. (Photo contributor: Lawrence B. Stack MD)

BASILAR SKULL FRACTURE

Clinical Summary

The skull "base" comprises the frontal bone, occiput, occipital condyles, clivus, carotid canals, petrous portion of the temporal bones, and the posterior sphenoid wall. A basilar skull fracture is basically a linear fracture of the skull base. Trauma resulting in fractures to this area typically does not have localizing symptoms. Indirect signs of the injury may include visible evidence of bleeding from the fracture into surrounding soft tissue, such as a Battle sign or "raccoon eyes." Bleeding into other structures—including hemotympanum or blood in the sphenoid sinus seen as an air-fluid level on computed tomography (CT)—may also be seen. Cerebrospinal fluid (CSF) leaks may also be evident and noted as clear or pink rhinorrhea. If CSF is present, a dextrose stick test may be positive. The fluid can be placed on filter paper and a "halo" or double ring may be seen.

Management and Disposition

Identify underlying brain injury, which is best accomplished by CT. CT is also the best diagnostic tool for identifying the fracture site, but fractures may not always be evident. Evidence of open communication, such as a CSF leak, mandates neurosurgical consultation and admission. Otherwise, the decision for admission is based on the patient's clinical condition, other associated injuries, and evidence of underlying brain injury as seen on CT. The use of antibiotics in the presence of a CSF leak is controversial because of the possibility of selecting resistant organisms.



FIGURE 1.1 ■ **Battle Sign.** Ecchymosis in the postauricular area develops when the fracture line communicates with the mastoid air cells, resulting in blood accumulating in the cutaneous tissue. This patient had sustained injuries several days prior to presentation. (Photo contributor: Frank Birinyi, MD.)



FIGURE 1.2 ■ **Battle Sign.** A striking Battle sign is seen in this patient with head trauma. This finding may take hours to days to develop. (Photo contributor: David Effron, MD.)

Pearls

1. Clinical manifestations of basilar skull fracture may take several hours to fully develop.
2. There should be a low threshold for head CT in any patient with head trauma, loss of consciousness, change in mental status, severe headache, visual changes, or nausea or vomiting.
3. The use of filter paper or a dextrose stick test to determine if CSF is present in rhinorrhea is not 100% reliable.



FIGURE 1.3 ■ **Raccoon Eyes.** Acute periorbital ecchymosis seen in this patient with a basilar skull fracture. These findings may also be caused by facial fractures. (Photo contributor: Shannon Koh, MD.)



FIGURE 1.4 ■ **Early Raccoon Eyes.** Subtle periorbital ecchymosis manifests 1 hour after a blast injury. (Photo contributor: Kevin J. Knoop, MD, MS.)

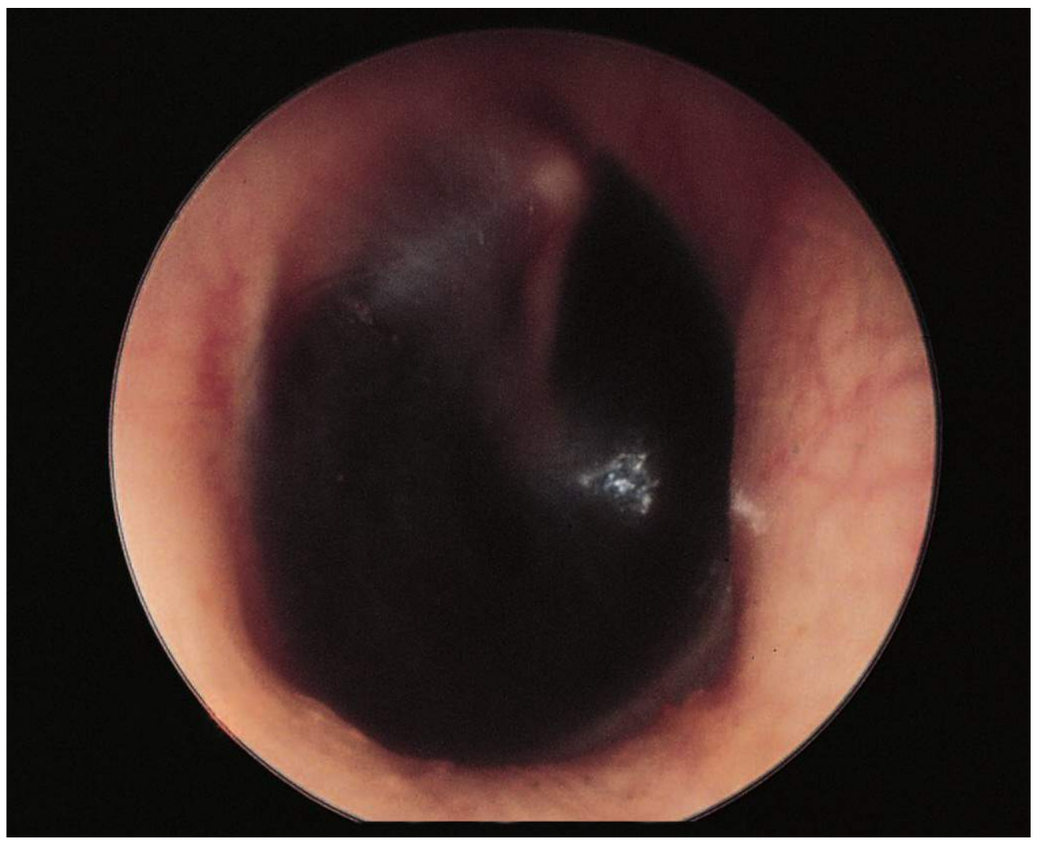


FIGURE 1.5 ■ **Hemotympanum.** Seen in a basilar skull fracture when the fracture line communicates with the auditory canal, resulting in bleeding into the middle ear. Blood can be seen behind the tympanic membrane. (Photo contributor: Richard A. Chole, MD, PhD.)



FIGURE 1.6 ■ **CT of Basilar Skull Fracture.** CT bone window demonstrates a fracture of the posterior wall left sphenoid sinus (arrow) and an air-fluid level. (Photo contributor: Jared McKinney, MD.)



FIGURE 1.7 ■ **Halo Sign—Cerebrospinal Fluid Leak.** Otorrhea on bed sheet demonstrating a halo sign from a patient with severe head trauma. The distinctive double-ring sign, seen here, comprises blood (inner ring) and CSF (outer ring). The reliability of this test has been questioned. (Photo contributor: Suzanne Bryce, MD.)

Clinical Summary

Depressed skull fractures typically occur when a large force is applied over a small area. They are classified as open if the skin above them is lacerated. Abrasions, contusions, and hematomas may also be present over the fracture site. The patient's mental status is dependent upon the degree of underlying brain injury. Direct trauma can cause abrasions, contusions, hematomas, and lacerations without an underlying depressed skull fracture. Evidence of other injuries such as a basilar fracture, facial fractures, or cervical spinal injuries may also be present.

Management and Disposition

Explore all scalp lacerations to exclude a depressed fracture. CT should be performed in all suspected depressed skull fractures to determine the extent of underlying brain injury. Depressed skull fractures require immediate neurosurgical consultation. Treat open fractures with antibiotics and tetanus prophylaxis as indicated. The decision to observe or operate immediately is made by the neurosurgeon. Children below 2 years of age with skull fractures can develop leptomeningeal cysts, which are extrusion of CSF or brain through dural defects. For this reason, children below age 2 with skull fractures require close follow-up or admission.



FIGURE 1.8 ■ **Depressed Skull Fracture.** A scalp laceration overlying a depressed skull fracture. Scalp lacerations should undergo sterile exploration for skull fracture. (Photo contributor: David W. Munter, MD.)

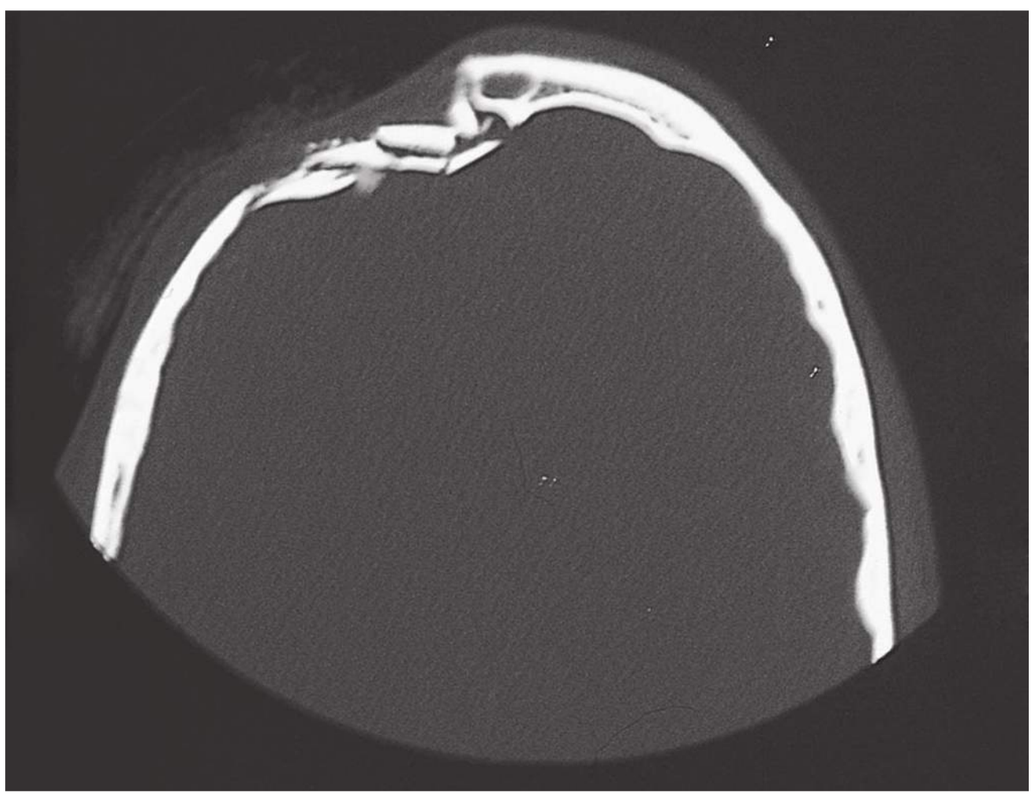


FIGURE 1.9 ■ **Depressed Skull Fracture.** CT demonstrating depressed skull fracture. (Photo contributor: David W. Munter, MD.)

Pearls

1. Examine all scalp injuries including lacerations for evidence of fractures or depression. When fragments are depressed 5 mm below the inner table, penetration of the dura and injury to the cortex are more likely.
2. Children with depressed skull fractures are more likely to develop epilepsy.
3. Ping pong ball skull fractures can occur from a forceps delivery or from compression by mother's sacral promontory during delivery.
4. Patients with head injuries must be evaluated for cervical spine injuries.

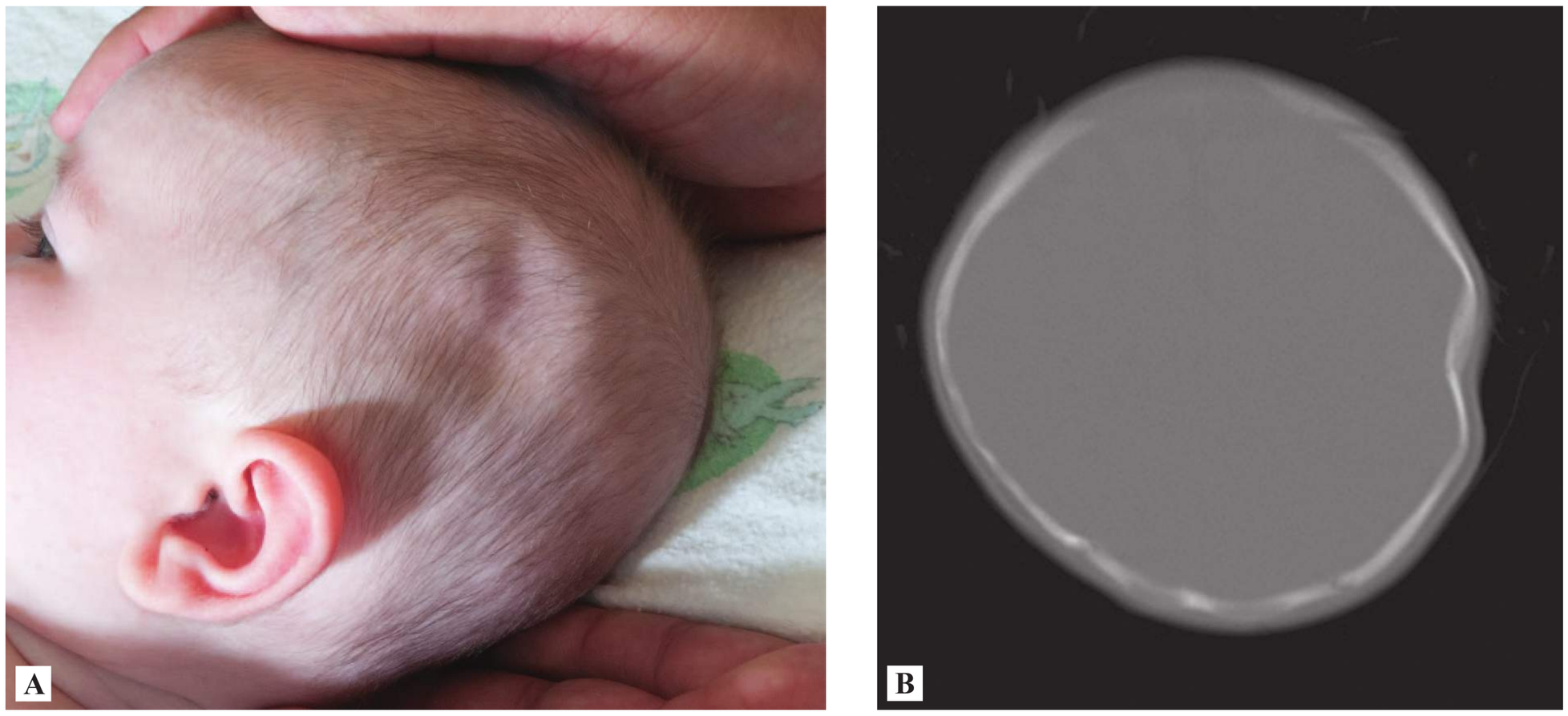


FIGURE 1.10 ■ **Ping Pong Ball Skull Fracture.** (**A**) Akin to the greenstick fracture, a ping pong ball fracture occurs when a newborn or infant's relatively soft skull is indented by the corner of a table or similar object without causing a frank break in the bone. (**B**) CT demonstrates the ping pong ball effect. (Photo contributor: Johnny Wei, BS.)

Clinical Summary

Clinically significant nasal fractures are almost always evident with deformity, swelling, and ecchymosis. Fractures to adjacent bony structures, including the orbit, frontal sinus, or cribriform plate, often occur. Epistaxis may be due to a septal or turbinate laceration but can also be seen with fractures of adjacent bones, including the cribriform plate. Septal hematoma is a rare but important complication that, if untreated, may result in necrosis of the septal cartilage and a resultant "saddle-nose" deformity. A frontonasoethmoid fracture has nasal or frontal crepitus and may have telecanthus or obstruction of the nasolacrimal duct.

Management and Disposition

Look for more serious injuries first. Patients with associated facial bone deformity or tenderness may require CT to rule out facial fractures. Isolated nasal fractures rarely require radiographs. Refer obvious deformities within 2 to 5 days for reduction, after the swelling has subsided. Nasal fractures with mild angulation and without displacement may be reduced in the emergency department (ED). Nasal injuries without deformity need only conservative therapy with an analgesic and possibly a nasal decongestant. Immediately drain septal hematomas, with packing placed to prevent reaccumulation. Uncontrolled epistaxis may require nasal packing. Vigorously irrigate and suture lacerations overlying a simple nasal fracture and place the patient on antibiotic coverage. Complex nasal lacerations with underlying fractures should be repaired by a facial surgeon.

Pearls

1. Control epistaxis to perform a good intranasal examination. If obvious deformity is present, including a new septal deviation or deformity, treat with ice and analgesics and provide ENT referral in 2 to 5 days for reduction.
2. Although the effectiveness of prophylactic antibiotics to prevent toxic shock syndrome is unproven, patients discharged with nasal packing should be placed on antistaphylococcal antibiotics and referred to ENT in 2 to 3 days.
3. Consider cribriform plate fractures in patients with clear rhinorrhea after nasal injury, with the understanding that this finding may be delayed.
4. Patients with facial trauma should be examined for a septal hematoma.



FIGURE 1.11 ■ **Nasal Fracture.** Deformity is evident on examination. Note periocular ecchymosis indicating the possibility of other facial fractures (or injuries). The decision to obtain radiographs is based on clinical findings. A radiograph is not indicated for an isolated simple nasal fracture. (Photo contributor: David W. Munter, MD.)



FIGURE 1.12 ■ **Septal Hematoma.** A bluish, grapelike mass on the nasal septum. If untreated, this can result in septal necrosis and a saddle-nose deformity. An incision, drainage, and packing are indicated. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 1.13 ■ **Saddle-Nose Deformity.** Nasal septal necrosis resulting in saddle-nose deformity. (Photo contributor: David Effron, MD.)



FIGURE 1.14 ■ **Open Nasal Fracture.** Lacerations with underlying fracture that require multilayer closure that should be repaired by a facial surgeon and require antibiotics. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 1.15 ■ **Minimally Displaced Nasal Fracture.** Plain radiograph of a fracture of the nasal spine. (Photo contributor: Lorenz F. Lassen, MD.)



FIGURE 1.16 ■ **Comminuted Nasal Fracture.** CT demonstrates a comminuted nasal bone fracture. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

The zygoma bone has two major components, the zygomatic arch and the body. The arch forms the inferior and lateral orbit, and the body forms the malar eminence of the face. Direct blows to the arch can result in isolated arch fractures. These present clinically with pain on opening the mouth secondary to the insertion of the temporalis muscle at the arch or impingement on the coronoid process. More extensive trauma can result in the "tripod fracture," which consists of fractures through three structures: the frontozygomatic suture; the maxillary process of the zygoma including the inferior orbital floor, inferior orbital rim, and lateral wall of the maxillary sinus; and the zygomatic arch. Clinically, patients present with a flattened malar eminence and edema and ecchymosis to the area, with a palpable step-off on examination. Injury to the infraorbital nerve may result in infraorbital paresthesia, and gaze disturbances may result from injury to orbital contents. Subcutaneous emphysema may be present.

Management and Disposition

Maxillofacial CT best identifies zygoma fractures. Treat simple zygomatic arch or tripod fractures without eye injury with ice and analgesics and refer for delayed operative consideration in 5 to 7 days. Refer extensive tripod fractures or those with eye injuries urgently. Decongestants and broad-spectrum antibiotics are generally recommended for tripod fractures, since the fracture crosses into the maxillary sinus. Fractures with blood in the sinus should also be treated with antibiotics.



FIGURE 1.17 ■ **Zygomatic Arch Fracture.** Axial cut of a facial CT which reveals a minimally depressed zygomatic arch fracture. (Photo contributor: Lawrence B. Stack, MD.)

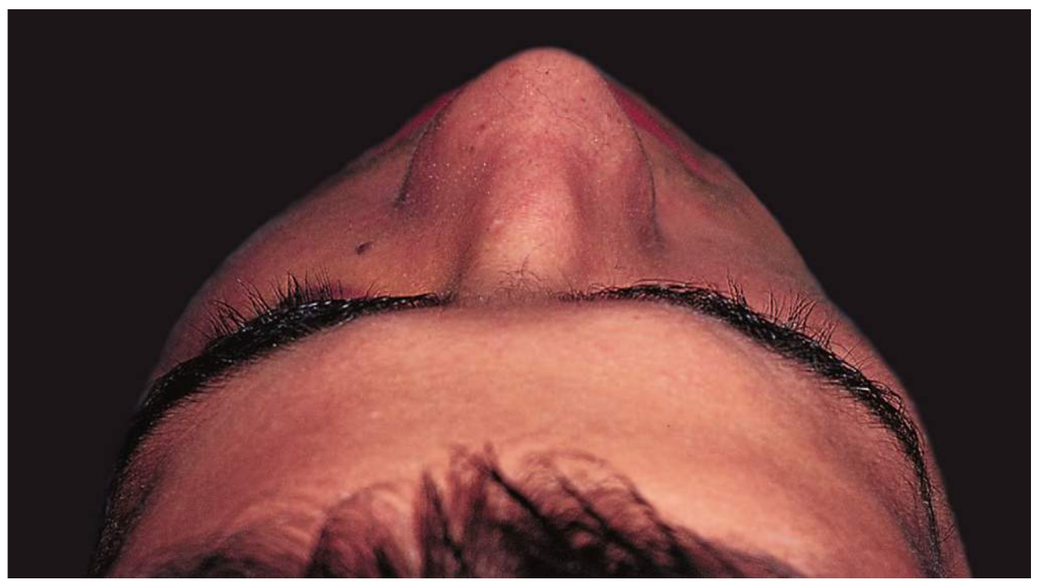


FIGURE 1.18 ■ **Zygomatic Arch Fracture.** Patient with blunt trauma to the zygoma. Flattening of the right malar eminence is evident. (Photo contributor: Edward S. Amrhein, DDS.)

Pearls

1. Tripod fractures are often associated with orbital and ocular trauma. Palpate the zygomatic arch and orbital rims carefully for a step-off deformity.
2. Examine for eye findings such as diplopia, hyphema, or retinal detachment. Check for infraorbital paresthesia indicating injury or impingement of the second division of cranial nerve V.
3. Visual inspection of the malar eminence from several angles (especially by viewing the area from over the head of the patient in the coronal plane) allows detection of a subtle abnormality.

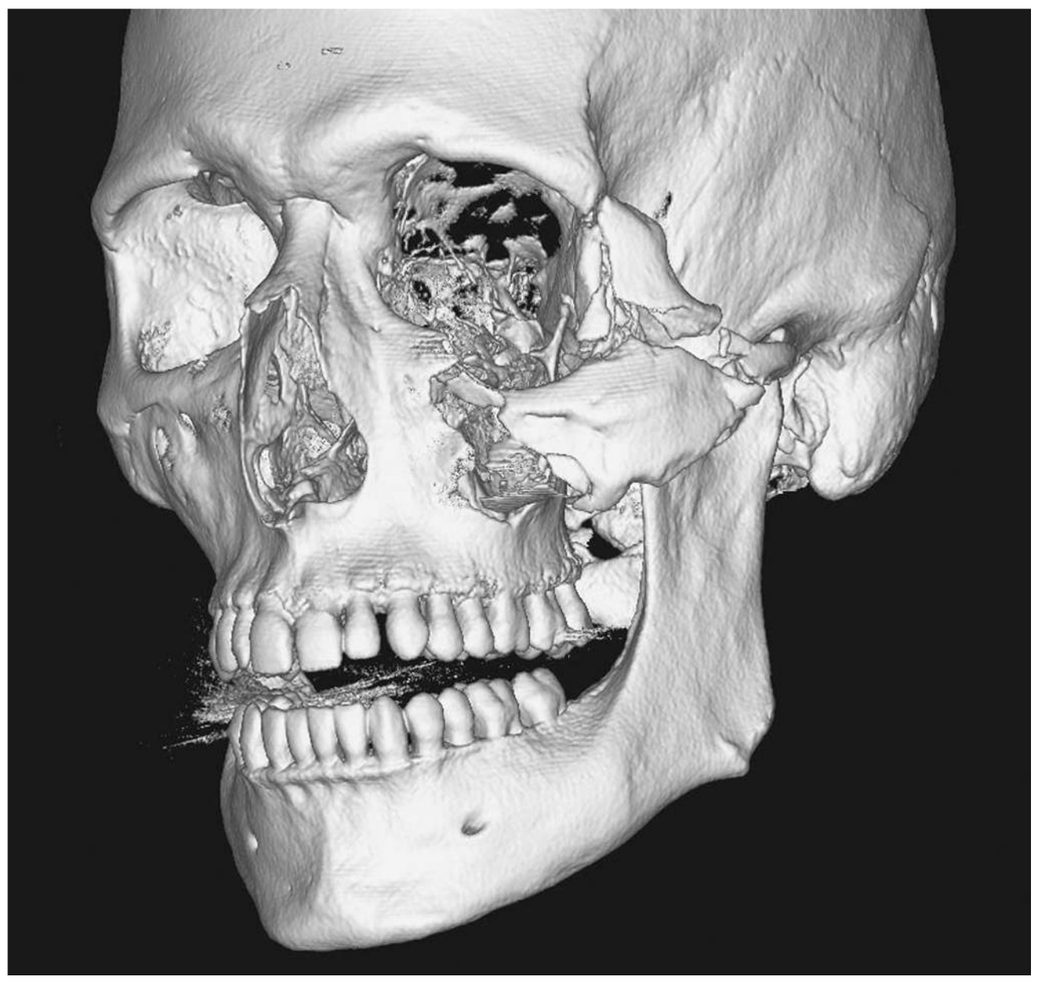


FIGURE 1.19 ■ **Tripod Fracture.** The fracture lines involved in a tripod fracture are demonstrated in this three-dimensional CT reconstruction. (Photo contributor: Patrick W. Lappert, MD.)

LEFORT FACIAL FRACTURES

Clinical Summary

All LeFort facial fractures involve the maxilla. Clinically, the patient has facial injuries, swelling, and ecchymosis. LeFort I fractures are those involving an area under the nasal fossa. LeFort II fractures involve a pyramidal area including the maxilla, nasal bones, and medial orbits. LeFort III fractures, sometimes described as craniofacial dissociation, involve the maxilla, zygoma, nasal and ethmoid bones, and the bones of the base of the skull. LeFort IV fractures have been described as a LeFort III fracture that also involves the frontal bone. Patients may have different LeFort fractures on each side of their face. Airway compromise may be associated with LeFort II and III fractures. Physical examination is sometimes helpful in distinguishing the four. The examiner places fingers on the bridge of the nose and tries to move the central maxillary incisors with the other hand. If only the maxilla moves, a LeFort I is present; movement of the upper jaw and nose indicates a LeFort II; and movement of the entire midface and zygoma indicates a LeFort III. Because of the extent of LeFort II and III fractures, they may be associated with cribriform plate fractures and CSF rhinorrhea. The force required to sustain a LeFort II or III fracture is considerable, and associated brain or cervical spine injuries or other facial fractures are common.

Management and Disposition

Attend to the airway and life-threatening injuries first. Maxillofacial CT best identifies LeFort injuries. Management of LeFort I fractures may involve only dental splinting and oral surgery referral. Consult on all LeFort II and III fractures for admission because of the need for operative repair. Epistaxis may be difficult to control in LeFort II and III fractures, in rare cases requiring intraoperative arterial ligation.

Pearls

1. Attention should be focused on immediate airway management, since the massive edema associated with LeFort II and III fractures may quickly lead to airway compromise.
2. Avoid nasotracheal intubation because of the possibility of intracranial passage.
3. Serious facial trauma is often associated with cervical spine injuries.
4. Associated cranial injuries are common, obtain head CT.
5. If not recognized, an occult CSF leak may result in significant morbidity. Suspected CSF leaks require neurosurgical consultation.



FIGURE 1.21 ■ **LeFort Facial Fractures.** Patient with blunt facial trauma. Note the ecchymosis and edema. This patient sustained a left LeFort II fracture and a right LeFort III, and intracranial hemorrhages. (Photo contributor: Stephen Corbett, MD.)

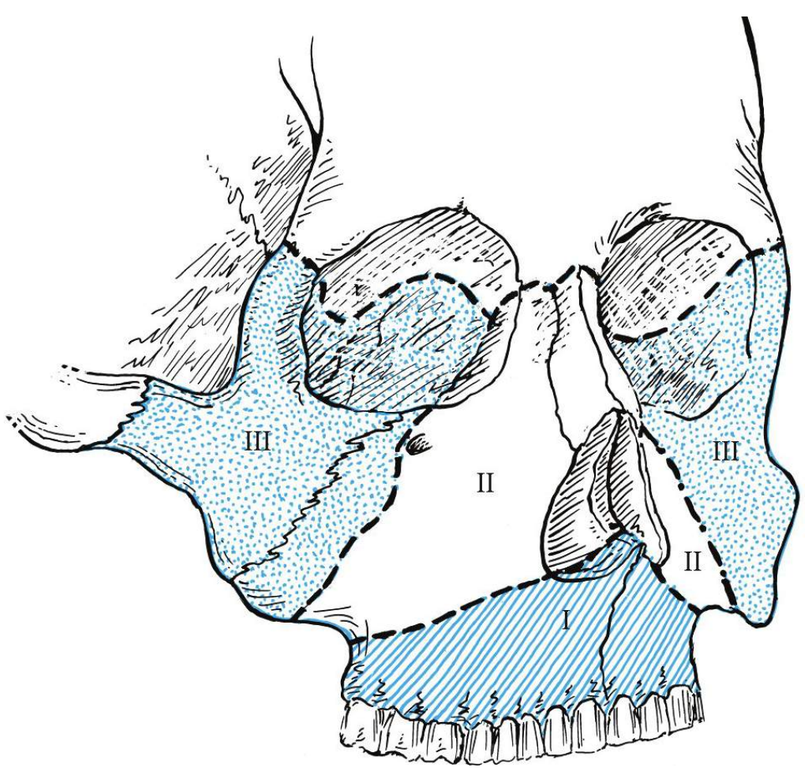


FIGURE 1.20 ■ **LeFort Fractures.** Illustration of the fracture lines of LeFort I (alveolar), LeFort II (zygomatic maxillary complex), and LeFort III (cranial facial dysostosis) fractures.



FIGURE 1.22 ■ **LeFort Facial Fractures.** Patient with blunt facial trauma who demonstrates the classic "dish face" deformity (depressed midface) associated with bilateral LeFort III fractures. (Photo contributor: Robert Schnarrs, MD.)

Clinical Summary

Orbital floor fractures are produced by two distinct mechanisms. The first is a true "blowout" fracture where all the energy is transmitted from a blunt object to the globe, causing increased orbital pressure blowing out either the orbital floor (most frequently) or medial wall. Fists and balls are the most common causative agents. This mechanism of injury is more likely to cause entrapment and globe injury. The second mechanism of injury occurs when the energy from the blow is transmitted to the infraorbital rim, causing a buckling of the orbital floor. Patients with blowout fractures have periorbital ecchymosis and lid edema but may also sustain globe injuries, including chemosis, subconjunctival hemorrhage, or infraorbital numbness from injury to the infraorbital nerve. Other globe injuries seen with orbital wall fractures include corneal abrasion, hyphema, enophthalmos, proptosis, iridoplegia, dislocated lens, retinal tear, retinal detachment, and ruptured globe. Diplopia with upward gaze suggests entrapment of the inferior rectus or its supporting structures. Diplopia with lateral gaze suggests entrapment of the medical rectus muscle. Periorbital subcutaneous emphysema is frequently seen with orbital wall fractures because of the proximity to the sinuses.

Management and Disposition

Maxillofacial CT best identifies orbital wall fractures. Treat patients without eye injury or entrapment conservatively with ice and analgesics, and refer for follow-up in 2 to 3 days. Treat patients with blood in the maxillary sinus with antibiotics. Strongly consider an ophthalmology consultation in patients with a true blowout fracture, as 30% of these patients sustain a significant globe injury. Immediately refer patients with entrapment, as muscle necrosis may occur if muscle blood supply is compromised by the entrapment.

Pearls

1. Enophthalmos, limited upward gaze, diplopia with upward gaze, or infraorbital anesthesia from entrapment or injury to the infraorbital nerve should heighten suspicion of an orbital floor fracture.
2. Compare the pupillary level on the affected side with the unaffected side, since it may be lower from prolapse of the orbital contents into the maxillary sinus. Subtle abnormalities may be appreciated as an asymmetric corneal light reflex (Hirschberg reflex).
3. Subcutaneous emphysema, a soft-tissue teardrop along the roof of the maxillary sinus on plain film, or an air-fluid level in the maxillary sinus on plain film should also be interpreted as evidence of an orbital floor fracture.
4. Patients with orbital wall fracture may present with subcutaneous emphysema after blowing their nose or air bubbles emanating from the tear duct.
5. Carefully examine the eye for visual acuity, hyphema, or retinal detachment, and nose for septal hematoma.



FIGURE 1.23 ■ **Orbital Floor Fracture.** Sustained from blunt trauma to infraorbital rim causing buckling of the orbital floor. Facial CT is similar to that in Fig. 1.25. Infraorbital hypesthesias and lack of entrapment suggest the buckling mechanism of injury. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 1.24 ■ **Inferior Rectus Entrapment.** The right inferior rectus muscle is entrapped within this orbital floor fracture limiting upward gaze. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 1.26 ■ **Medial Wall Orbital Fracture.** Periorbital ecchymosis and swelling is seen in this patient with a medial wall orbital fracture. The patient blew her nose after the injury and the swelling became more prominent. (Photo contributor: Lawrence B. Stack, MD.)

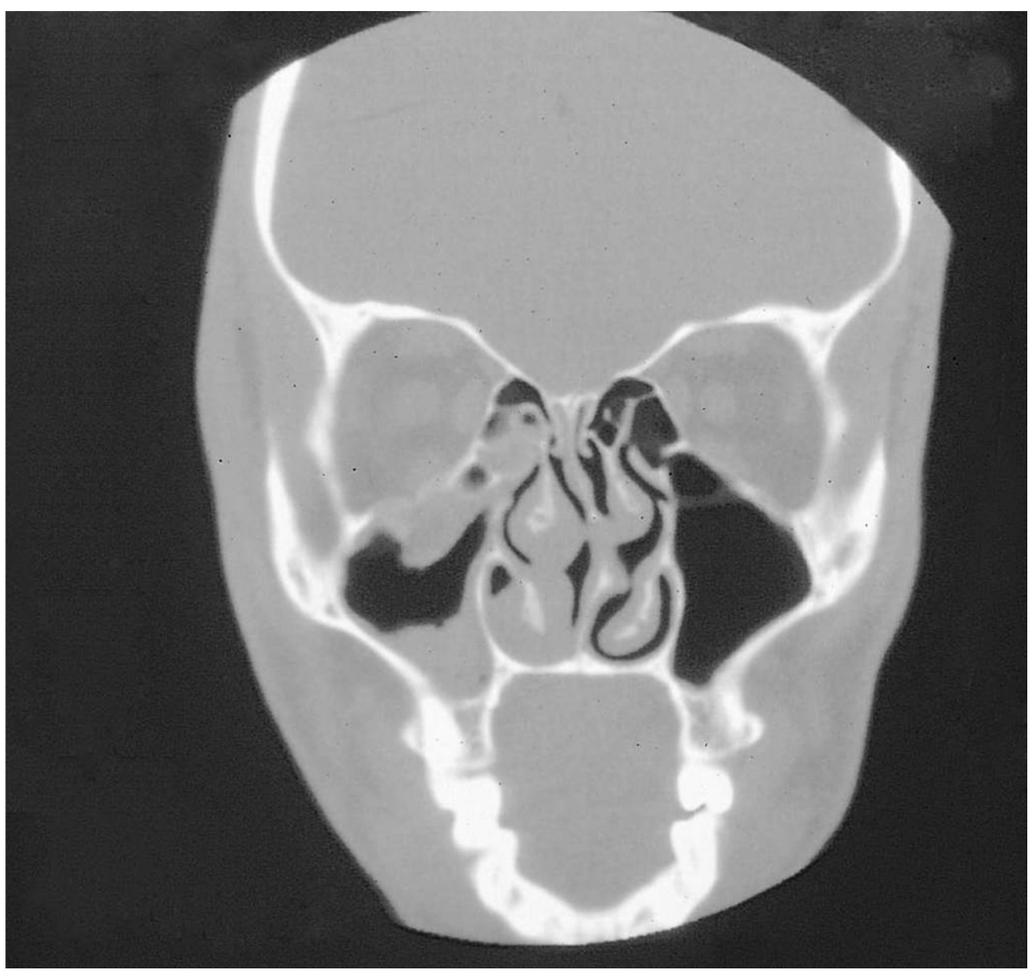


FIGURE 1.25 ■ **Orbital Floor Fracture with Entrapment.** Coronal CT of the patient in Fig. 1.24 demonstrating the entrapped muscle extruding into the maxillary sinus. (Photo contributor: Lawrence B. Stack, MD.)

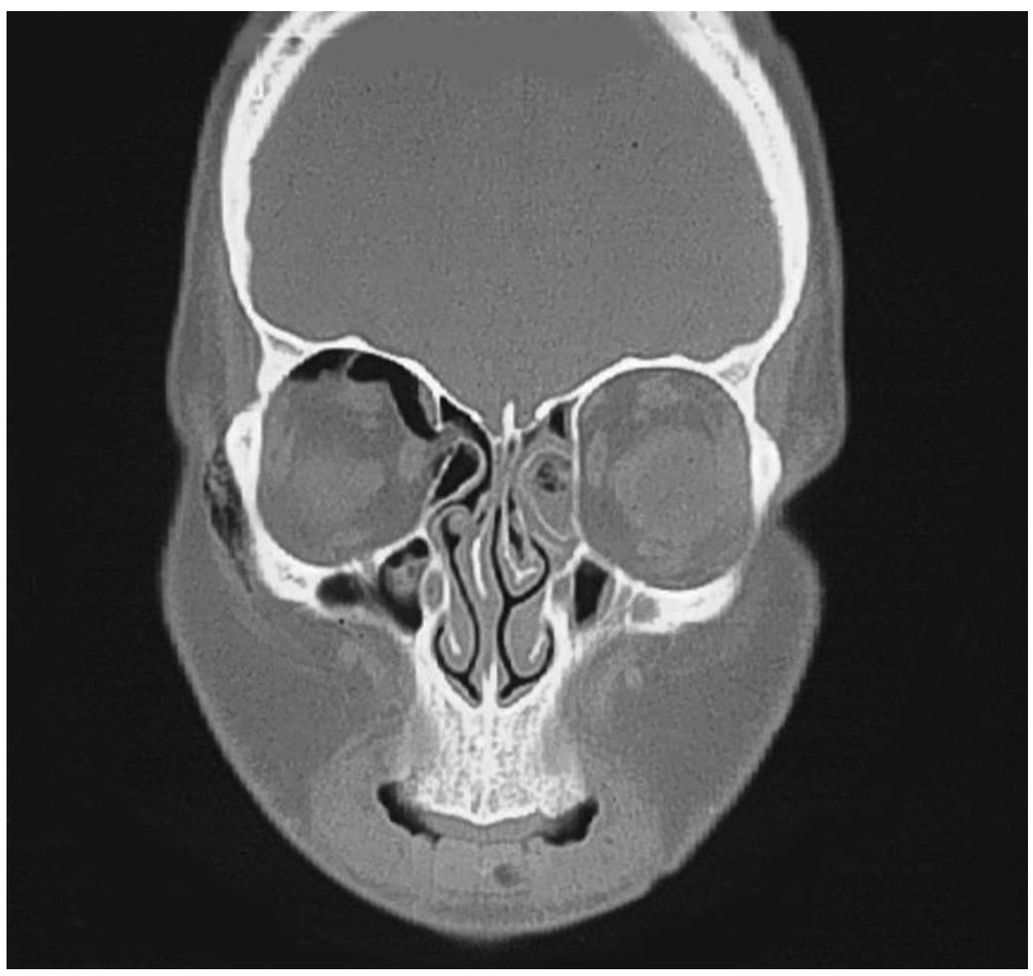


FIGURE 1.27 ■ **CT of Medial Wall Orbital Fracture.** Coronal CT of the patient in Fig. 1.26. Subcutaneous emphysema and orbital air is seen. An opening between the orbit and ethmoid air cells can be seen. (Photo contributor: Lawrence B. Stack, MD.)

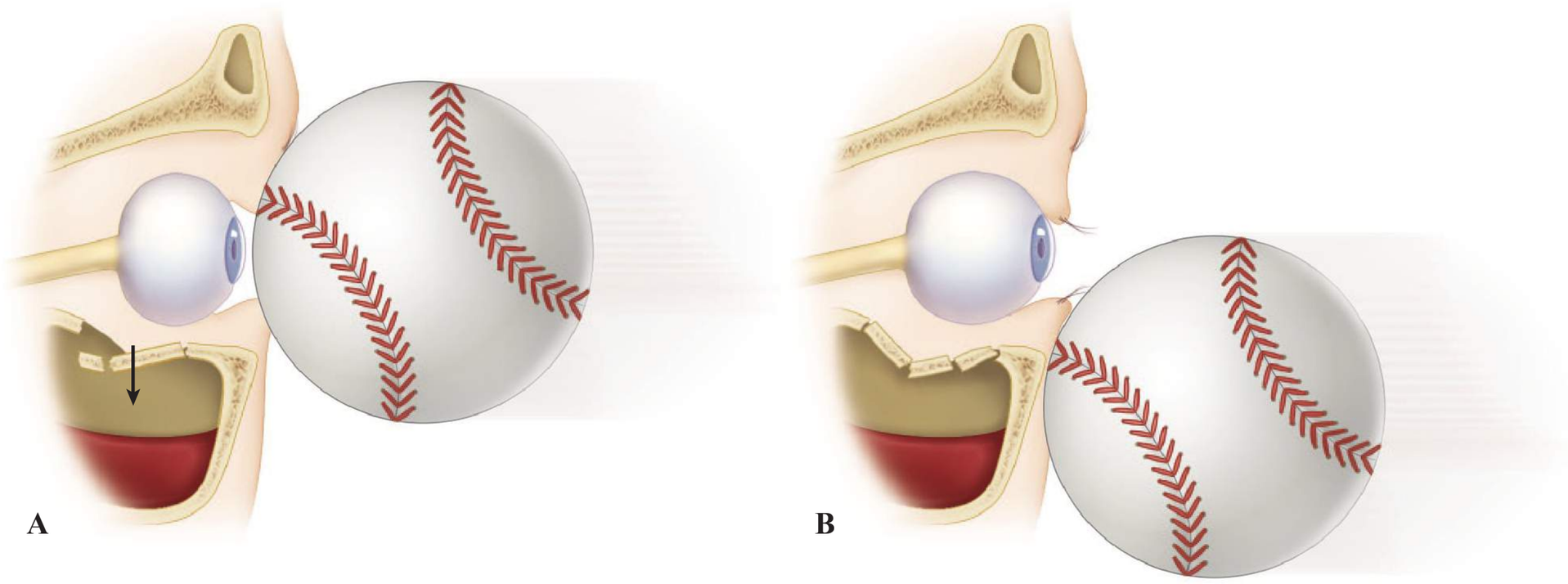


FIGURE 1.28 ■ **Mechanisms of Orbital Wall Injury.** (**A**) True "blowout" mechanism where all energy is transmitted to globe. (**B**) Buckle injury where energy is transmitted to inferior orbital rim, causing a buckling of the orbital floor.

MANDIBULAR FRACTURES

Clinical Summary

Blunt trauma, mandibular pain, and malocclusion are typically seen in patients with mandibular fractures. A step-off in the dental line or ecchymosis or hematoma to the floor of the mouth is often present. Mandibular fractures may be open to the oral cavity, as manifested by gum lacerations. Dental trauma is frequently seen. Other clinical features include inferior alveolar or mental nerve paresthesia, loose or missing teeth, dysphagia, trismus, or ecchymosis of the floor of the mouth (considered pathognomonic). Multiple mandibular fractures are present in more than 50% of cases because of the ringlike structure of the mandible. Mandibular fractures are often classified as "favorable" or "unfavorable." Fractures displaced by masseter muscle contraction are unfavorable and inevitably require fixation, whereas fractures that are not displaced by masseter contraction are favorable and in most cases will not require fixation. Injuries creating unstable mandibular fractures may create airway obstruction because the support for the tongue is lost. Mandibular fractures are also classified based on the anatomic location of the fracture. Dislocation of the mandibular condyles may also result from blunt trauma and will always have associated malocclusion, typified by an inability to close the mouth.

Management and Disposition

A dental panoramic film is the best diagnostic image for evaluating mandibular trauma. If only plain films are available, obtain anteroposterior, bilateral oblique, and Towne views to evaluate the condyles. Treat nondisplaced fractures with analgesics, soft diet, and referral to oral surgery in 1 to 2 days. Displaced fractures, open fractures, and fractures with associated dental trauma need more urgent referral. Treat all mandibular fractures with antibiotics effective against anaerobic oral flora (clindamycin, amoxicillin/clavulanate) and give tetanus prophylaxis if needed. The Barton bandage has been suggested to immobilize the jaw in the ED.

Pearls

1. The most sensitive sign of a mandibular fracture is malocclusion. The jaw will deviate toward the side of a unilateral condylar fracture on maximal opening of the mouth.
2. A nonfractured mandible should be able to hold a tongue blade between the molars tightly enough to break it off. There should be no pain in attempting to rotate the tongue blade between the molars.
3. Bilateral parasymphyseal fractures may cause acute airway obstruction in the supine patient. This is relieved by pulling the subluxed mandible and soft tissue forward and, in patients in whom the cervical spine has been cleared, by elevating the patient to a sitting position.



FIGURE 1.29 ■ **Open Mandibular Fracture.** An open fracture is suggested by the misaligned teeth and gingival disruption. (Photo contributor: Lawrence B. Stack, MD.)

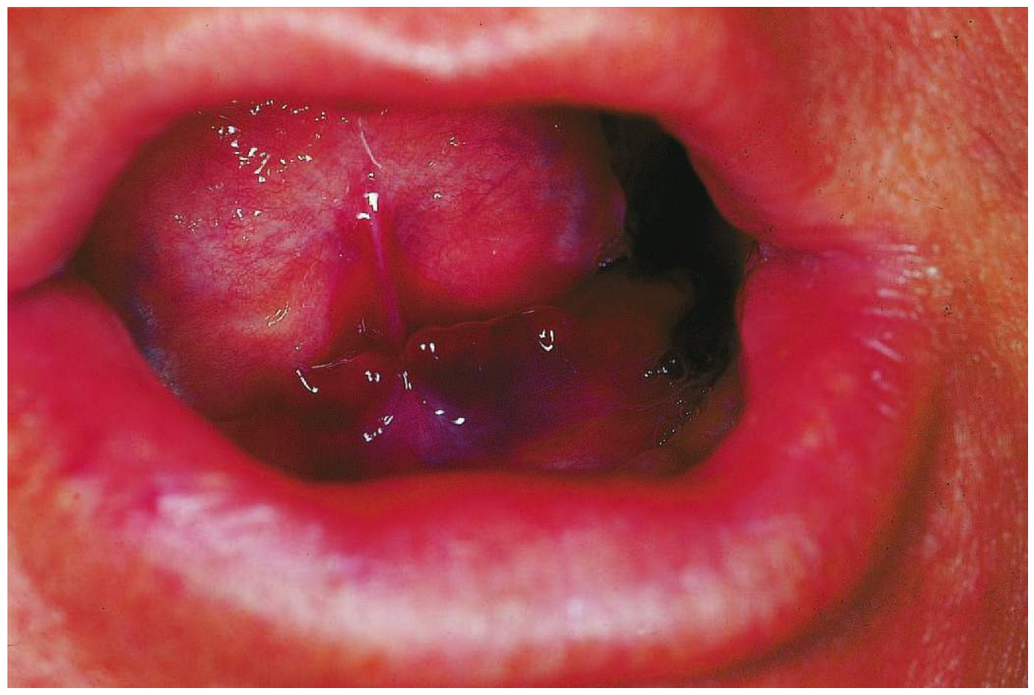


FIGURE 1.30 ■ **Sublingual Hemorrhage.** Hemorrhage or ecchymosis in the sublingual area is pathognomonic for mandibular fracture. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 1.31 ■ **Bilateral Mandibular Fracture.** The diagnosis is suggested by the bilateral ecchymosis seen in this patient. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 1.32 ■ **Favorable Mandibular Fracture.** Dental panoramic view showing two nondisplaced mandibular fractures that are amenable to conservative therapy. (Photo contributor: David W. Munter, MD.)

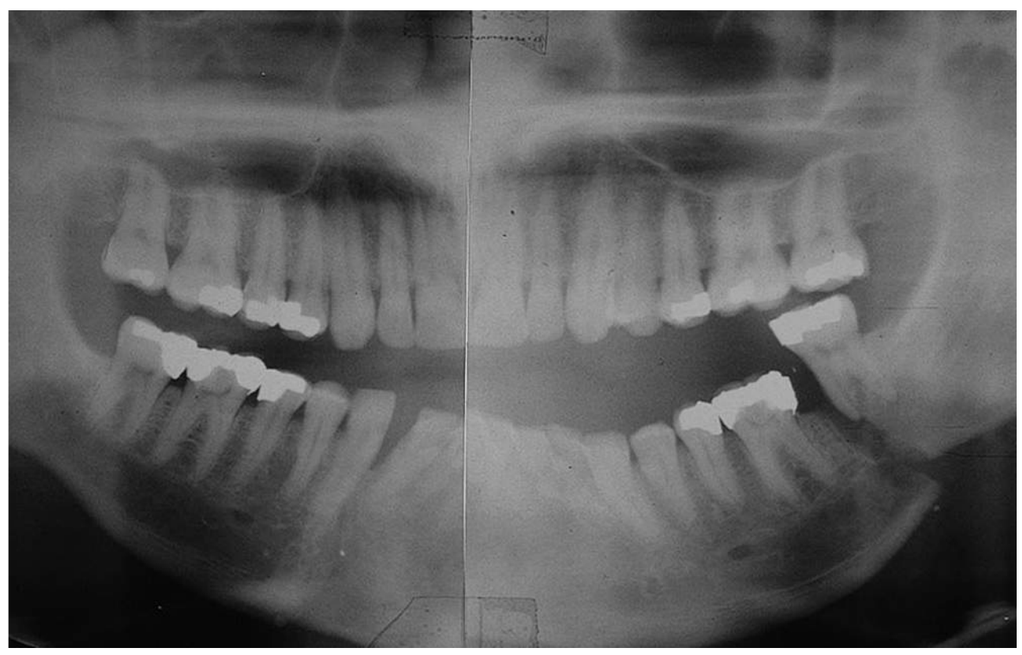


FIGURE 1.33 ■ **Unfavorable Mandibular Fracture.** Dental panoramic view demonstrating a mandibular fracture with obvious misalignment due to the distracting forces of the masseter muscle. (Photo contributor: Edward S. Amrhein, DDS.)

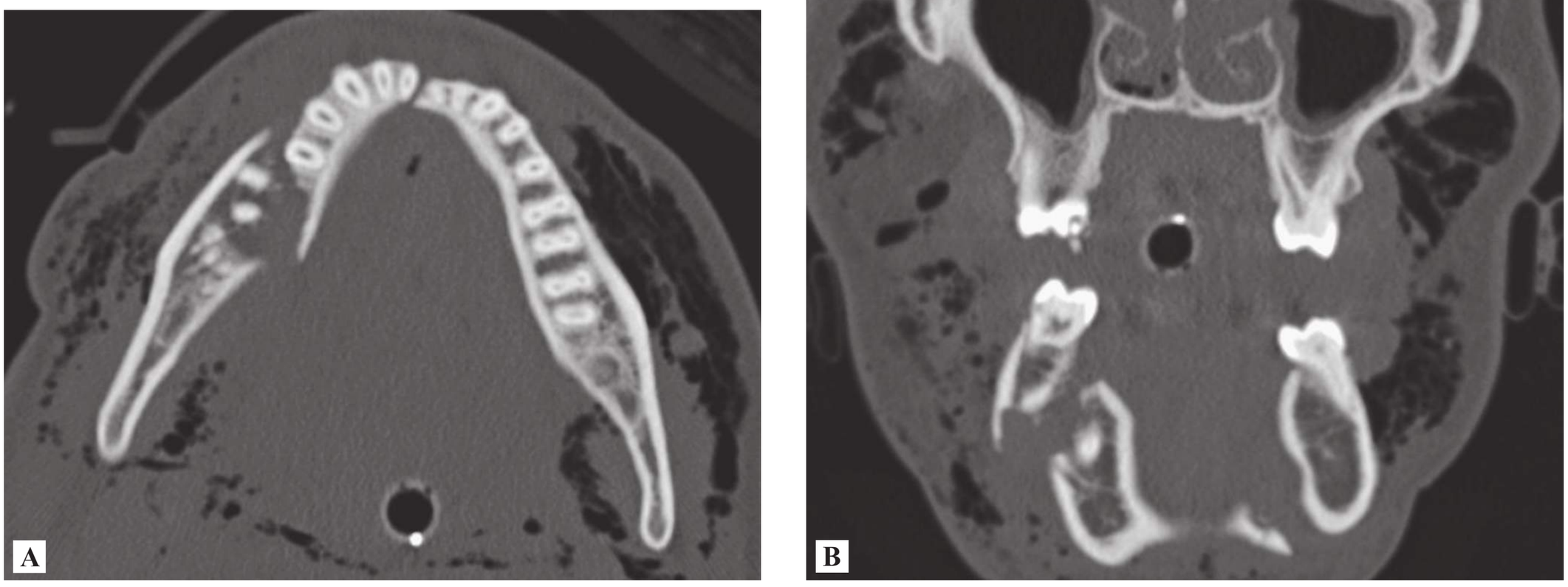


FIGURE 1.34 ■ **Mandibular Fractures.** Axial (**A**) and coronal (**B**) views of a maxillofacial CT reveal a right mandibular body and a parasymphyseal fracture. (Reproduced with permission from Block J, Jordanov MI, Stack, LB, and Thurman RJ (eds). The Atlas of Emergency Radiology. New York: McGraw-Hill; 2013; Fig. 1-99A and B.)

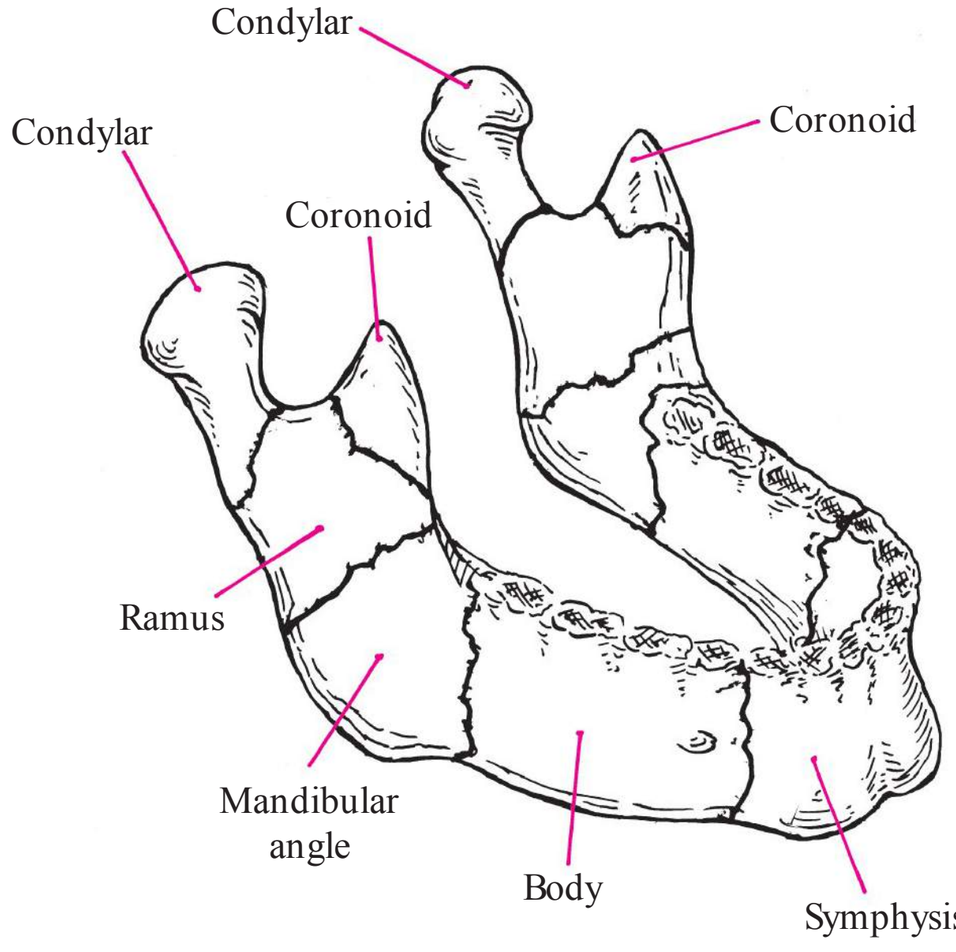


FIGURE 1.35 ■ **Classification of Mandibular Fractures**. Classification based on anatomic location of the fracture.

Clinical Summary

Injuries to the external ear may be open or closed. Blunt external ear trauma may cause a hematoma (otohematoma) of the pinna, which, if untreated, may result in cartilage necrosis and chronic scarring or further cartilage formation and permanent deformity ("cauliflower ear"). Open injuries include lacerations (with and without cartilage exposure) and avulsions.

Management and Disposition

Pinna hematomas must undergo incision and drainage or large needle aspiration using sterile technique, followed by a pressure dressing to prevent reaccumulation of the hematoma. This procedure may need to be repeated several times; hence, after ED drainage, the patient is treated with antistaphylococcal antibiotics and referred to ENT or plastic surgery for follow-up in 24 hours. Lacerations must be carefully examined for cartilage involvement; if this is present, copious irrigation, closure, and postrepair oral antibiotics covering skin flora are



FIGURE 1.37 ■ **Pinna Hematoma.** A hematoma has developed, characterized by swelling, discoloration, ecchymosis, and flocculence. Immediate incision and drainage or aspiration is indicated, followed by an ear compression dressing. (Photo contributor: C. Bruce MacDonald, MD.)



FIGURE 1.36 ■ **Pinna Contusion.** Contusion without hematoma is present. Reevaluation in 24 hours is recommended to ensure a drainable hematoma has not formed. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 1.38 ■ **Cauliflower Ear.** Repeated trauma to the pinna or undrained hematomas can result in cartilage necrosis and subsequent deforming scar formation. (Photo contributor: Timothy D. McGuirk, DO.)



FIGURE 1.39 ■ **Complete Avulsion of Partial Pinna.** This ear injury, sustained in a fight, resulted when the pinna was bitten off. Plastic repair is needed. The avulsed part was wrapped in sterile gauze soaked with saline and placed in a sterile container on ice. (Photo contributor: David W. Munter, MD.)

indicated. Simple skin lacerations may be repaired primarily with nonabsorbable 6-0 sutures. The dressing after laceration repair is just as important as the primary repair. If a compression dressing is not placed, hematoma formation can occur. Complex lacerations or avulsions normally require ENT or plastic surgery consultation.

Pearls

1. Pinna hematomas may take hours to develop, so give patients with blunt ear trauma careful discharge instructions, with a follow-up in 12 to 24 hours to check for hematoma development.
2. Failure to adequately drain a hematoma, reaccumulation of the hematoma owing to a faulty pressure dressing, or inadequate follow-up increases the risk of infection of the pinna (perichondritis) or of a disfiguring cauliflower ear.
3. Copiously irrigate injuries with lacerated cartilage, which can usually be managed by primary closure of the overlying skin.



FIGURE 1.40 ■ **Partial Avulsion of Pinna.** Partial avulsion of the pinna seen at its superior junction with the scalp. Cartilage exposure and injury prompts ENT consultation for repair. (Photo contributor: Eric Wu, MD.)

Direct closure of the cartilage is rarely necessary and is indicated only for proper alignment, which helps lessen later distortion. Use a minimal number of absorbable 5-0 or 6-0 sutures through the perichondrium.

4. Lacerations to the lateral aspect of the pinna should be minimally debrided because of the lack of tissue at this site to cover the exposed cartilage.
5. In the case of an avulsion injury, the avulsed part should be cleansed, wrapped in saline-moistened gauze, placed in a sterile container, and then placed on ice to await reimplantation by ENT.



FIGURE 1.41 ■ **Complete Avulsion of Entire Pinna.** This injury occurred as a result of a motor vehicle crash. The pinna was not found. (Photo contributor: Ian T. McClure, MD.)

Clinical Summary

Blunt trauma to the frontal area may result in a depressed frontal sinus fracture. Often, there is an associated laceration. Isolated frontal fractures normally do not have the associated features of massive blunt facial trauma such as seen in LeFort II and III fractures. Careful nasal speculum examination may reveal blood or CSF leak high in the nasal cavity. Posterior table involvement can lead to mucopyocele or epidural empyema as late sequelae. Involvement of the posterior wall of the frontal sinus may occur and result in cranial injury or dural tear. Frontal fractures may be part of a complex of facial fractures, as seen in frontonasoethmoid fractures, but generally more extensive facial trauma is required.

Pearls

1. Explore every frontal laceration digitally before repair. Digital palpation is sensitive for identifying frontal fractures, although false positives from lacerations extending through the periosteum can occur.
2. Communication of irrigating solutions with the nose or mouth indicates a breach in the frontal sinus.
3. For serious injuries, a CT scan is mandatory to assess the posterior aspect of the sinus and for possible intracranial injury.

Management and Disposition

Obtain head CT with bone windows on patients suspected of frontal sinus fractures. Fractures involving only the anterior table of the frontal sinus can be treated conservatively with referral to ENT or plastic surgery in 1 to 2 days. Fractures involving the posterior table require urgent neurosurgical consultation. Treat frontal sinus fractures with broad-spectrum antibiotics against both skin and sinus flora. ED management also includes control of epistaxis, application of ice packs, and analgesia.



FIGURE 1.42 ■ **Frontal Laceration.** Any laceration over the frontal sinuses should be explored to rule out a fracture. This laceration was found to have an associated frontal fracture. (Photo contributor: David W. Munter, MD.)

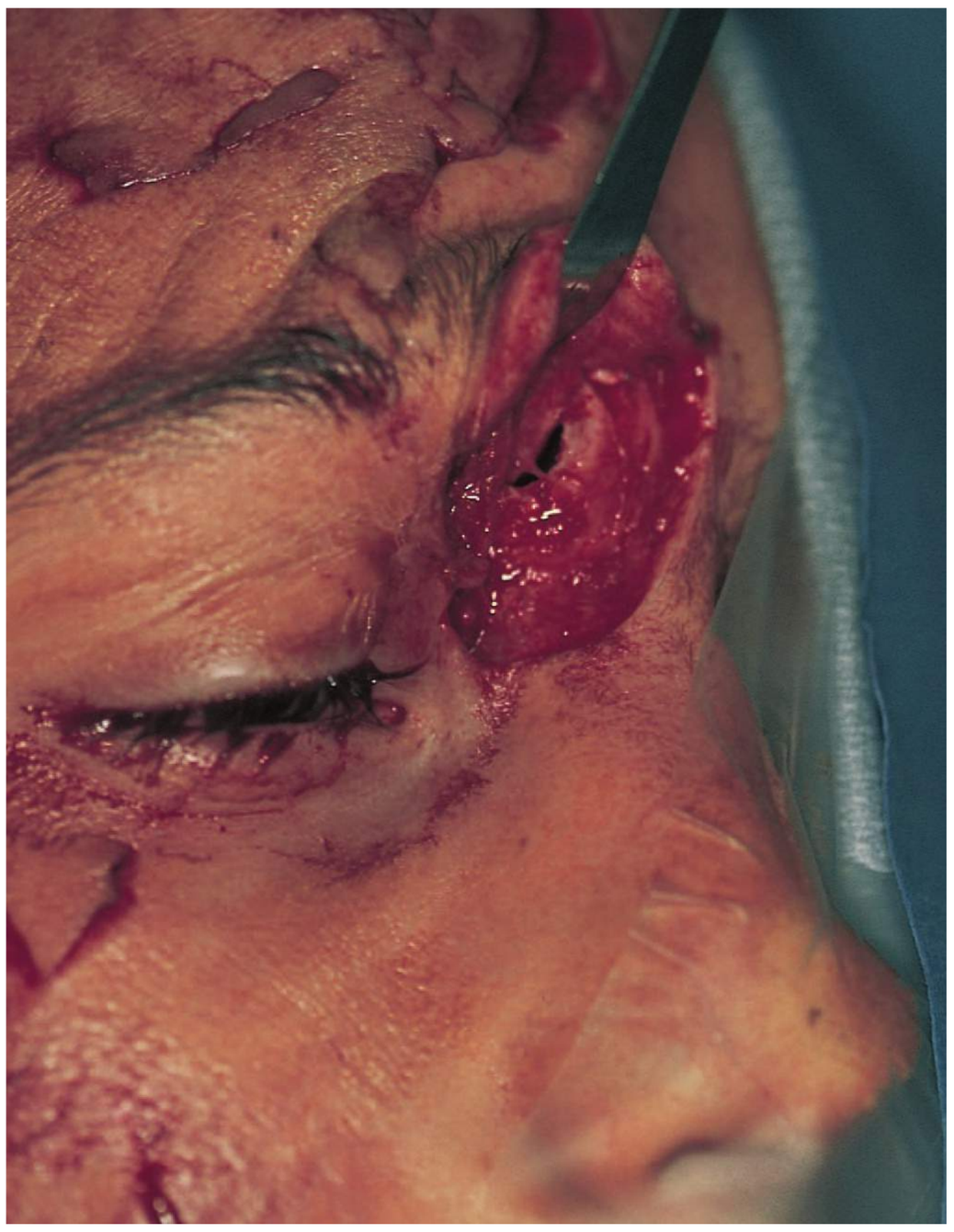


FIGURE 1.43 ■ **Frontal Sinus Fracture.** Fracture defect seen at the base of a laceration over the frontal sinus. (Photo contributor: Jeffrey Kuhn, MD.)



FIGURE 1.44 ■ **Frontal Sinus Fracture.** Fracture of the outer table of the frontal sinus is seen under this forehead laceration. (Photo contributor: Lawrence B. Stack, MD.)

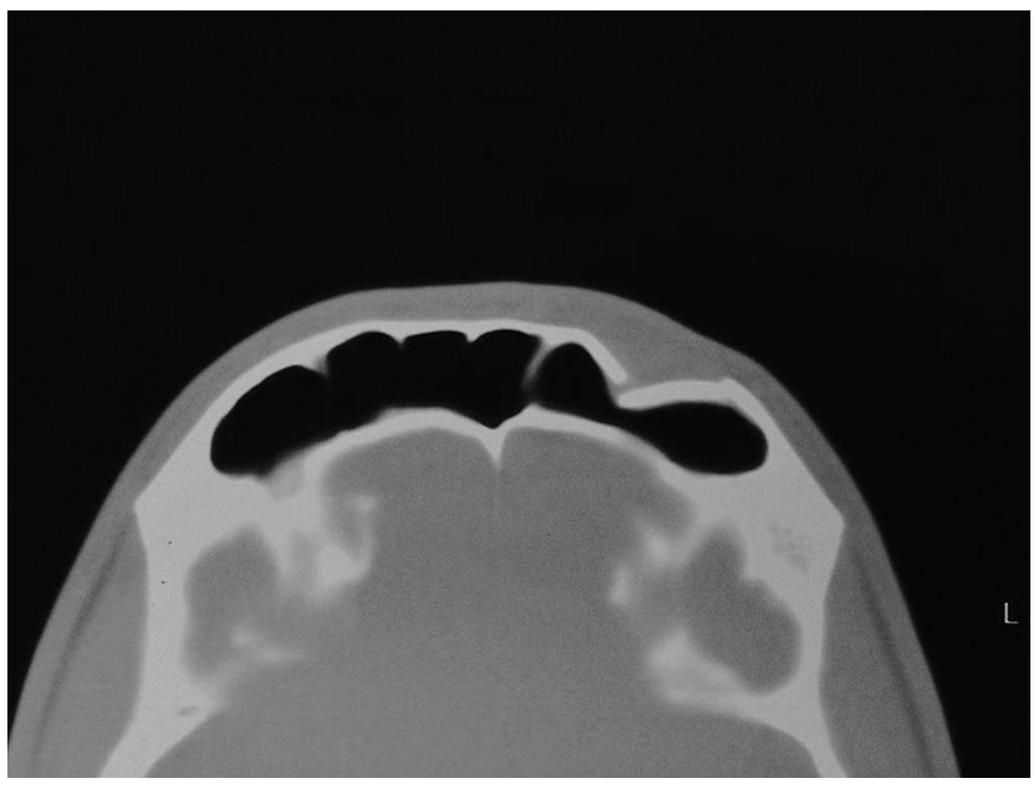


FIGURE 1.45 ■ **Frontal Sinus Fracture.** CT of the patient in Fig. 1.42 demonstrating a fracture of the anterior table of the frontal sinus. (Photo contributor: David W. Munter, MD.)

TRAUMATIC EXOPHTHALMOS

Clinical Summary

Typically the result of blunt orbital trauma, traumatic exophthalmos develops as a retrobulbar hematoma pushes the globe anteriorly. Patients present with periorbital edema, ecchymosis, a marked decrease in visual acuity, and an afferent pupillary defect in the involved eye. The exophthalmos, which may be obscured by periorbital edema, can be better appreciated from a superior view. Visual acuity may be affected by the direct trauma to the eye (retinal detachment, hyphema, globe rupture), compression of the retinal artery, or, more rarely, neuropraxia of the optic nerve.

Management and Disposition

Maxillofacial or orbital CT best delineates the presence and extent of a retrobulbar hematoma and associated facial or orbital fractures. Emergent ENT and ophthalmology consultation is indicated. Emergent lateral canthotomy and cantholysis to decompress the orbit can be sight-saving.

Pearls

1. The retrobulbar hematoma and exophthalmos may not develop for hours after the injury.
2. A subtle exophthalmos may be detected by looking down over the head of the patient and viewing the eye from the coronal plane.
3. Elevated intraocular pressure, relative afferent pupillary defect, and diminished visual acuity in patients with traumatic exophthalmos should strongly be considered for emergent lateral canthotomy and cantholysis.

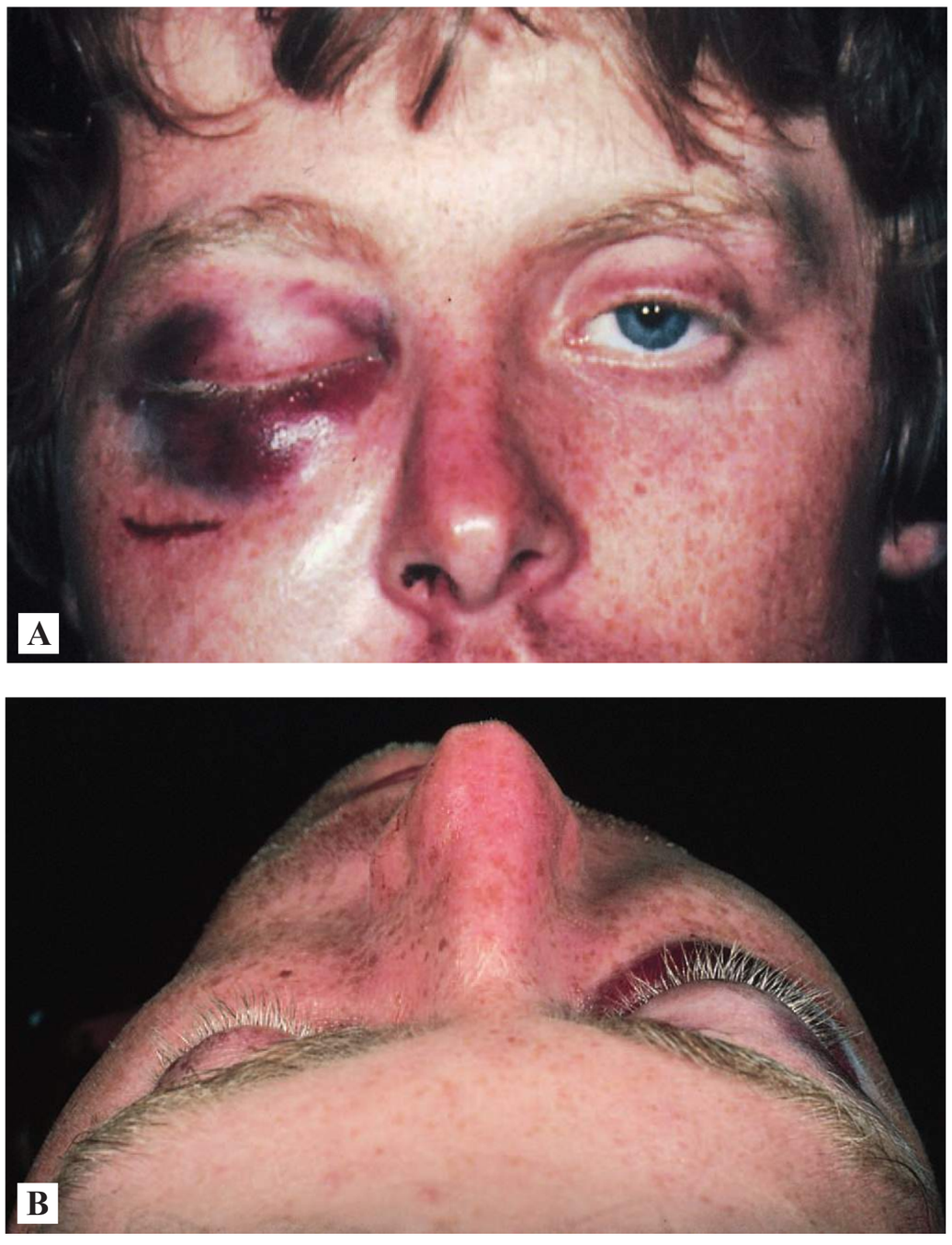


FIGURE 1.46 ■ **Traumatic Exophthalmos.** (**A**) Blunt trauma resulting in periorbital edema and ecchymosis, which obscures the exophthalmos in this patient. The exophthalmos is not obvious in the AP view and can therefore be initially unappreciated. (**B**) The same patient viewed in the coronal plane from over the forehead demonstrating right eye exophthalmos. (Photo contributor: Frank Birinyi, MD.)



FIGURE 1.47 ■ **Retrobulbar Hematoma.** CT of the patient in Fig. 1.46 with right retrobulbar hematoma and traumatic exophthalmos. (Photo contributor: Frank Birinyi, MD.)



FIGURE 1.48 ■ **Traumatic Exophthalmos.** Proptosis, hyphema, periorbital ecchymosis, and marked swelling in the patient with a retrobulbar hematoma from severe head and face trauma. Examination findings are more obvious than Fig. 1.46. (Photo contributor: David Effron, MD.)

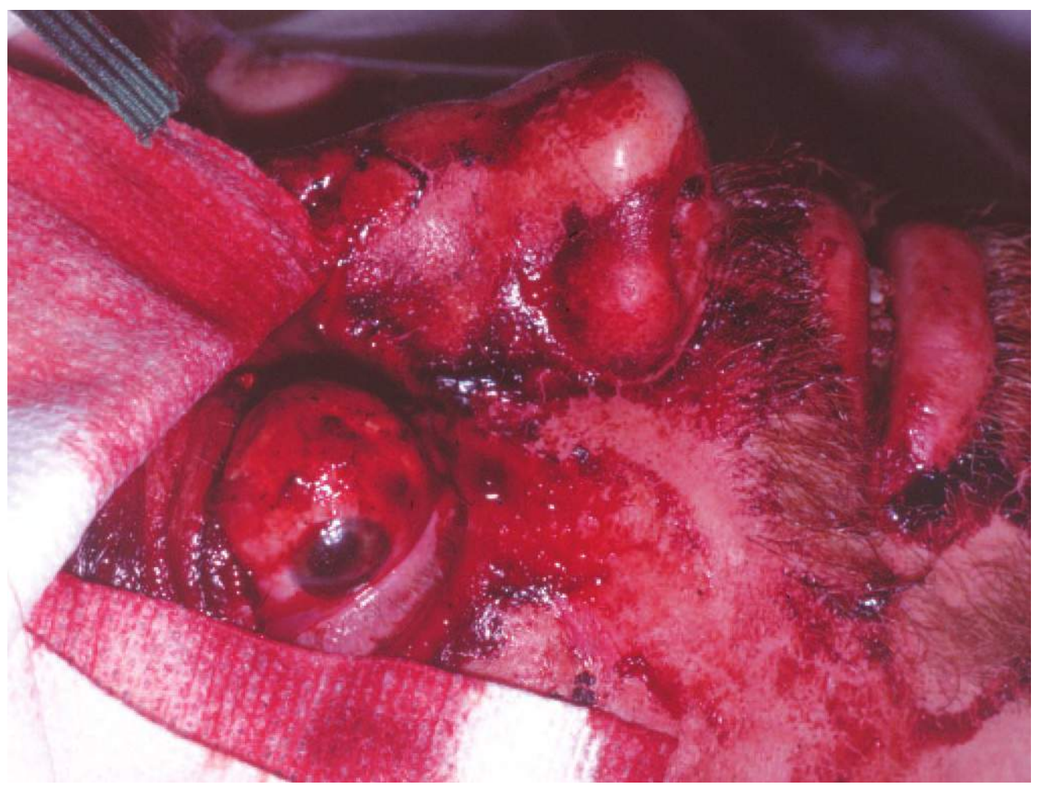
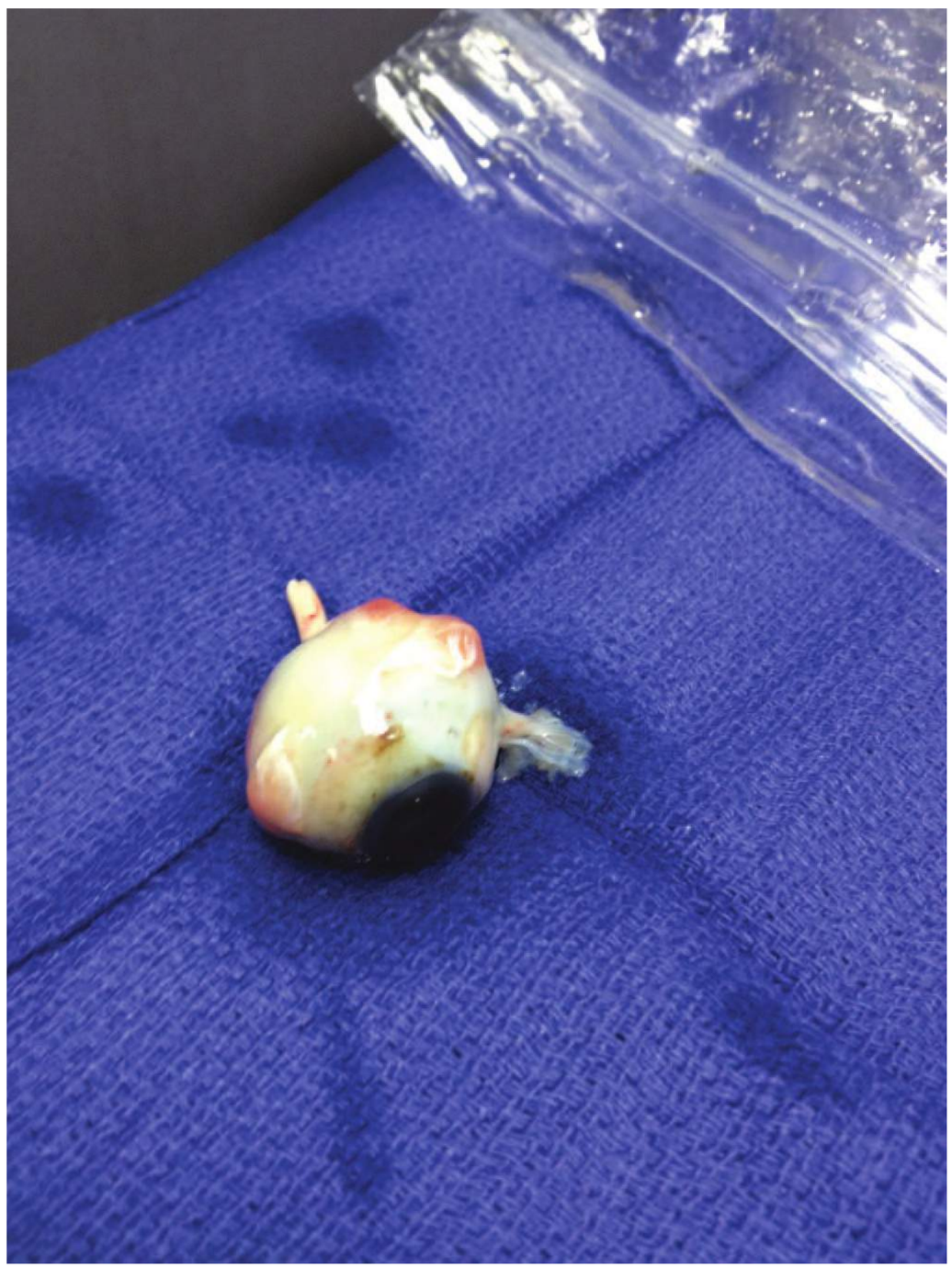


FIGURE 1.49 ■ **Traumatic Exophthalmos.** Anterior globe dislocation due to high energy facial trauma. There is no retrobulbar hematoma in this patient (Photo contributor: Lawrence B. Stack, MD.)



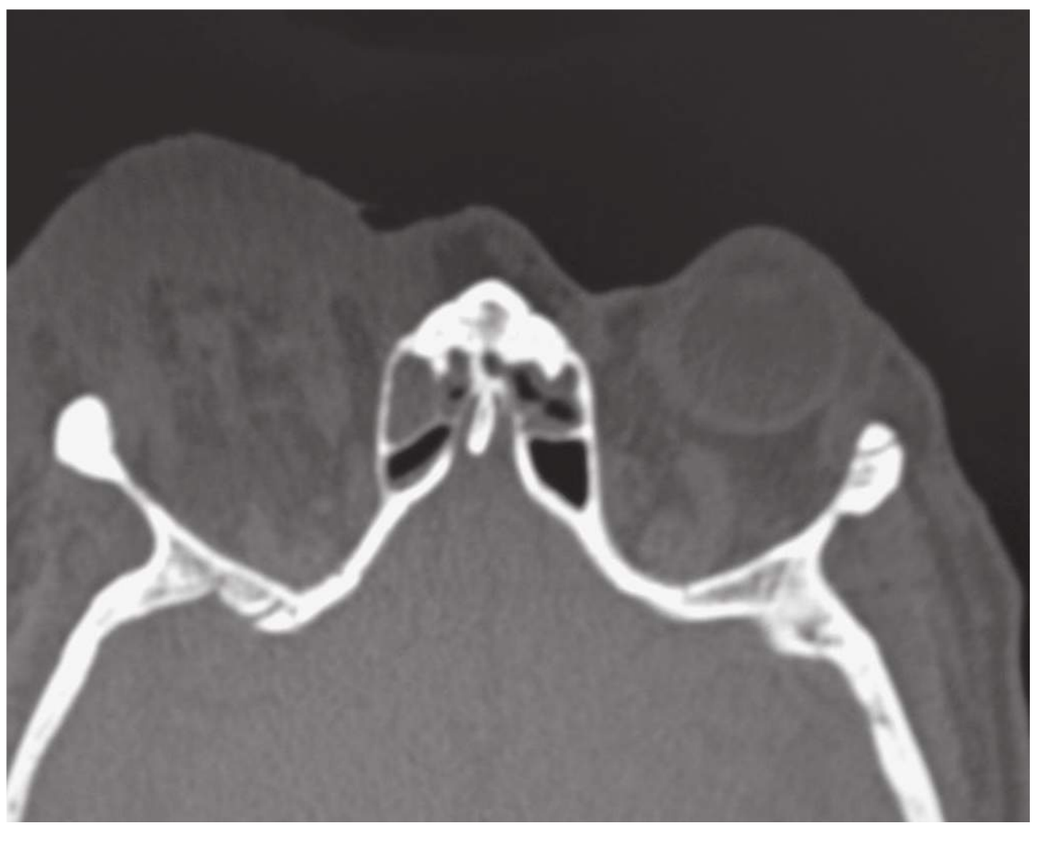


FIGURE 1.50 ■ **Traumatic Enucleation.** Complete enucleation of the right eye after a mechanical fall and hitting their face on the corner of a table. Family came with the patient with the eye in a plastic bag. No other injury is seen on orbit CT scan. (Photo contributor: Kevin S. Barlotta, MD)

Clinical Summary

Injury patterns of penetrating facial trauma can be predicted based on projectile type, entry location, and path. Midface injuries extend from the supraorbital rim superiorly to the oral commissure inferiorly and to the external auditory meatus posteriorly. Mandibular injuries extend from the oral commissure superiorly and to the lower border of the mandible inferiorly. Shotgun wounds typically involve both facial zones and will involve one or both eyes in 50% of patients. Fifty percent of patients with gunshot wounds (GSWs) to the mandible will require an emergency airway. Stab wounds are less likely to require emergency airway than GSWs. Additional structures that require consideration during the ED evaluation include brain, blood vessels, and esophagus.

Management and Disposition

After the primary survey, strongly consider intubation in patients with any gunshot injury to the mandible, blood or swelling in the oropharynx, or any close range (<7 m) shotgun injury. If evidence suggests a projectile remains in the face or cranium, obtain plain films in two planes to help guide subsequent evaluation such as CT angiogram or fine cuts of the orbits. CT will provide more detailed information in the injured structures to guide therapy. Removal of any projectile should only be performed after significant structure injury has been excluded and preparation for the consequences of removal is complete.

Pearls

1. Due to the likelihood of multisystem injury (vascular, ocular, cranial, face) penetrating facial trauma, the patient should be transferred to a facility with comprehensive subspecialty trauma care.
2. Intubation should be strongly considered in all patients with GSWs to the mandible or in GSWs to the midface if there is any blood or swelling in the oral cavity.
3. Removal of any projectile is best done in the operating room.



FIGURE 1.52 ■ **Midface Injury.** A jackhammer bit is lodged into the right maxillary sinus. (Photo contributor: R. Jason Thurman, MD.)

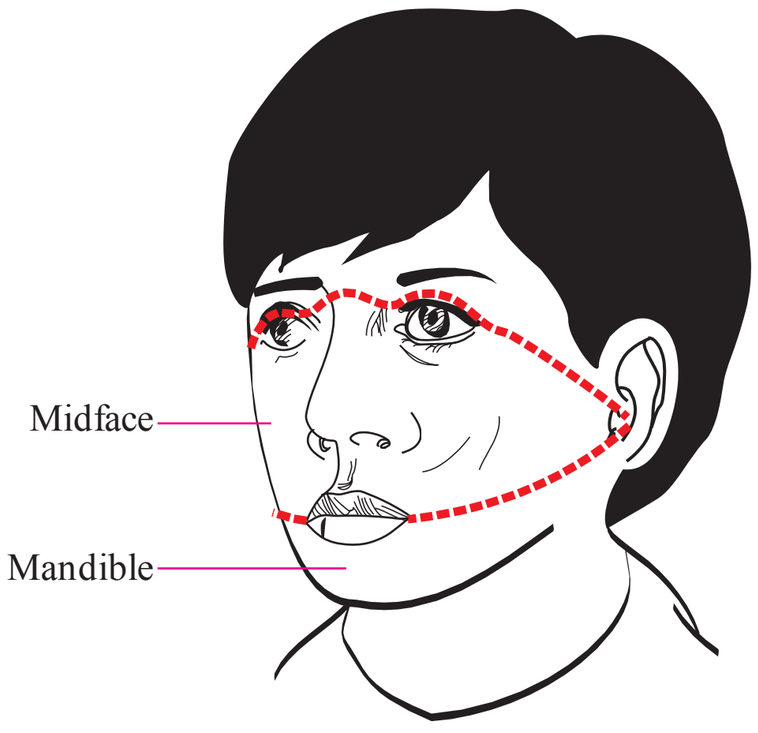


FIGURE 1.51 ■ **Facial Zones of Injury.** (Reproduced with permission from Chen AY, Stewart MG, Raup G. Penetrating injuries of the face. Otolaryngol Head Neck Surg. 1996;115:464-470.)



FIGURE 1.53 ■ **Midface Injury Radiograph.** Plain film of patient in Fig. 1.52. CT confirmed no other injury. Projectile was removed in the OR. (Photo contributor: R. Jason Thurman, MD.)

UNCAL HERNIATION SYNDROME

Clinical Summary

Severe head injury can result in extra-axial hematoma, cerebral contusion, or diffuse cerebral edema which, in turn, may cause one of five brain herniation syndromes: uncal, central transtentorial, cerebellotonsillar, subfalcine, and external. Uncal herniation occurs when the uncus of the temporal lobe is displaced inferiorly through the medial edge of the tentorium. Compression of cranial nerve III can cause an ipsilateral dilated pupil. Typically, patients with uncal herniation are unconscious and require intubation. A contusion to the eye may also result in a dilated, nonresponsive pupil and arouse suspicion for severe head injury and uncal herniation but typically these patients will be alert.



FIGURE 1.55 ■ **Ipsilateral Dilated Pupil due to Uncal Herniation.** CT revealed an epidural hematoma and unilateral effacement of the quadrigeminal cistern. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Intubate unconscious head trauma patients with a unilateral dilated pupil and transfer them immediately to a facility capable of caring for traumatic brain injury. A noncontrasted head CT scan can identify a subdural or epidural hematoma, diffuse edema, or temporal lobe contusion. These conditions often cause midline shift of cerebral structures and compression of the quadrigeminal cistern. Unilateral effacement of the quadrigeminal cistern confirms uncal herniation. Initial management focuses on maintaining cerebral perfusion pressure and normal tissue oxygenation as hypotension and hypoxia significantly contribute to secondary brain injury. Mannitol, hypertonic saline, burr holes, and hyperventilation should be considered in ED patients with uncal herniation. Definitive care requires neurosurgical consultation.

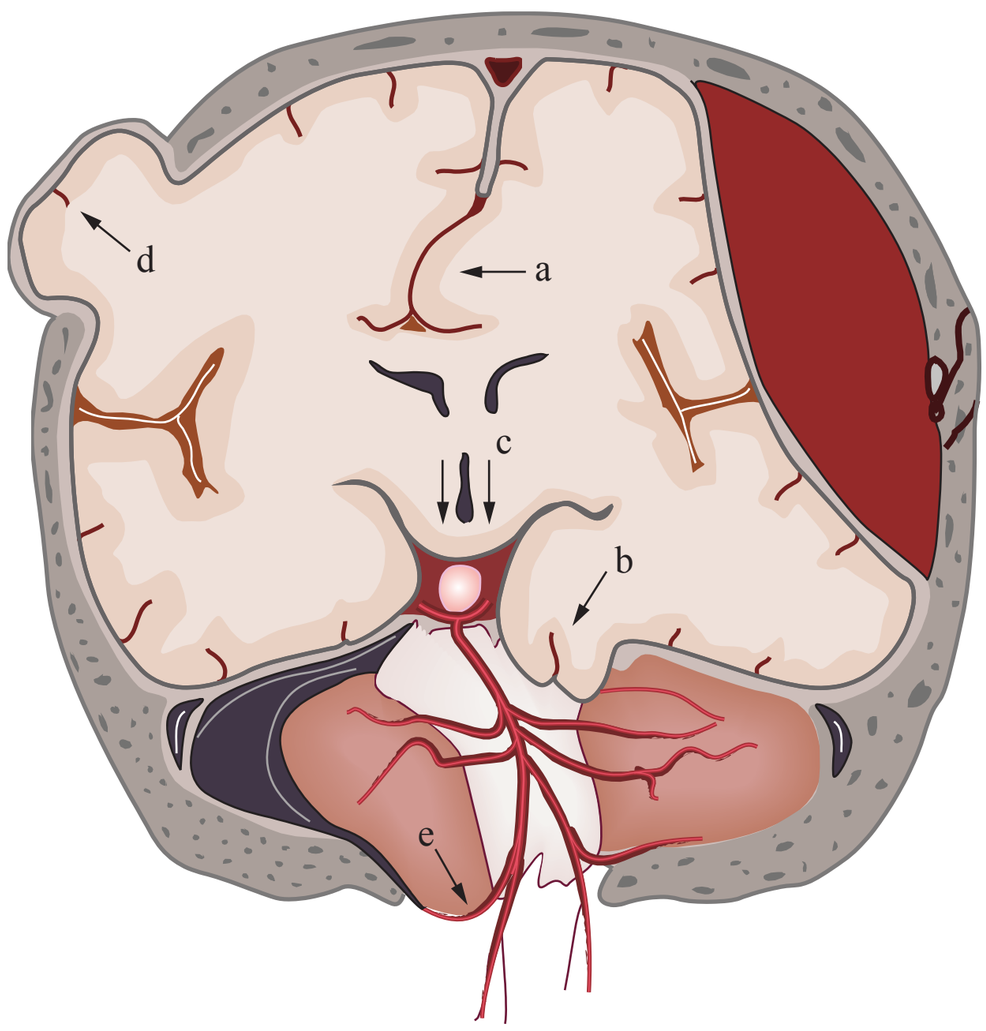


FIGURE 1.54 ■ **Herniation Syndromes. a.** Subfalcine; **b.** uncal; **c.** central transtentorial; **d.** external; **e.** cerebellotonsillar.

Pearls

1. Uncal herniation is the most common of the five herniation syndromes.
2. If a patient has a unilateral dilated pupil after head and face trauma but is awake and talking, be suspicious for isolated traumatic anisocoria.
3. Steroids have no role in the ED management of traumatic brain injury.
4. A temporal lobe contusion in an initially neurologically intact patient may continue to expand and cause uncal herniation.
5. Excessive hyperventilation ($PaCO_2$ < 25 mm Hg) in patients with severe traumatic brain injury is associated with cerebral ischemia.
6. Effacement of the quadrigeminal cistern is the hallmark CT finding of uncal herniation.

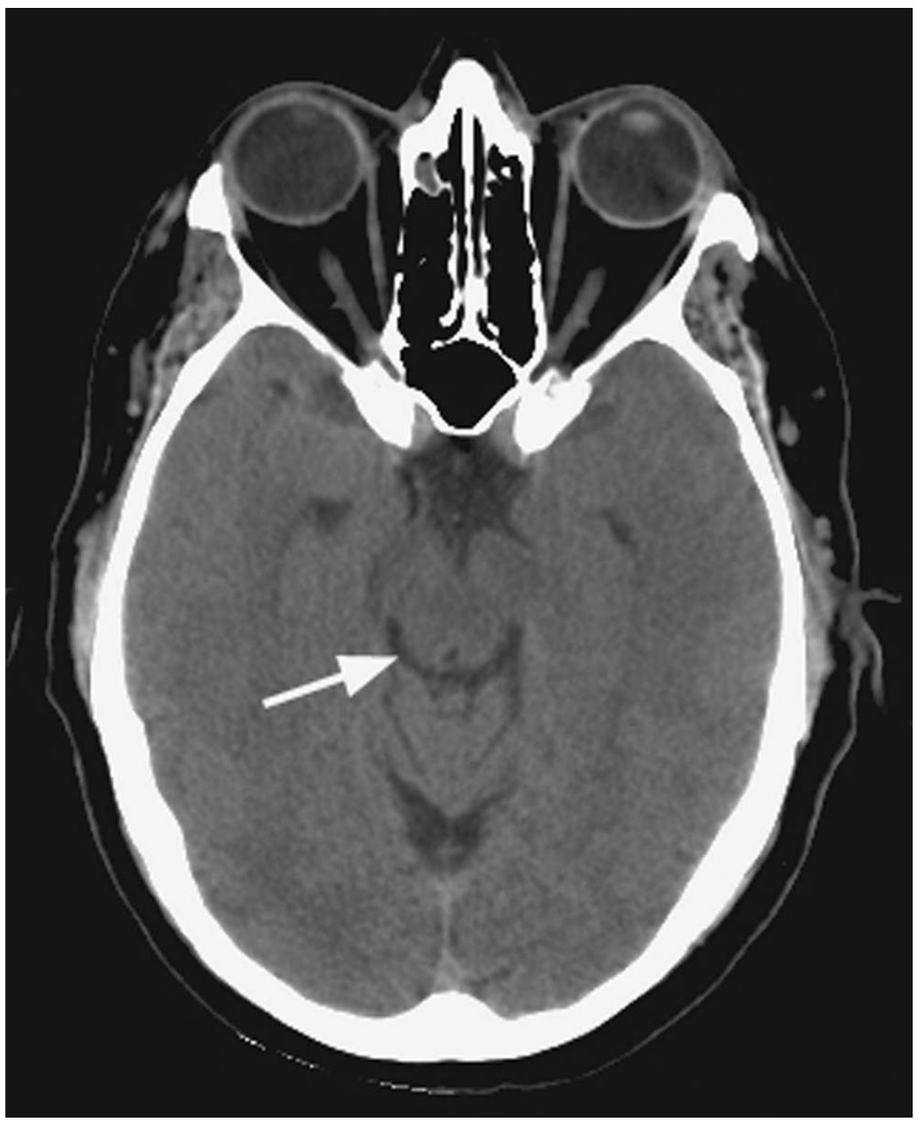


FIGURE 1.56 ■ **Normal Quadrigeminal Cistern.** The normal appearance of this CSF space is shaped like a baby's bottom (see arrow). It is located within two cuts superiorly of the dorsum sella. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 1.58 ■ **Subdural Hematoma**. A crescent-shaped subdural hematoma is seen on the left. The quadrigeminal cistern should be seen on this slice and is completely effaced, suggesting herniation. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 1.57 ■ **Epidural Hematoma.** A lens-shaped epidural hematoma is seen on the left. The quadrigeminal cistern should be seen on this slice and is completely effaced, suggesting herniation. (Photo contributor: Lawrence B. Stack, MD.)



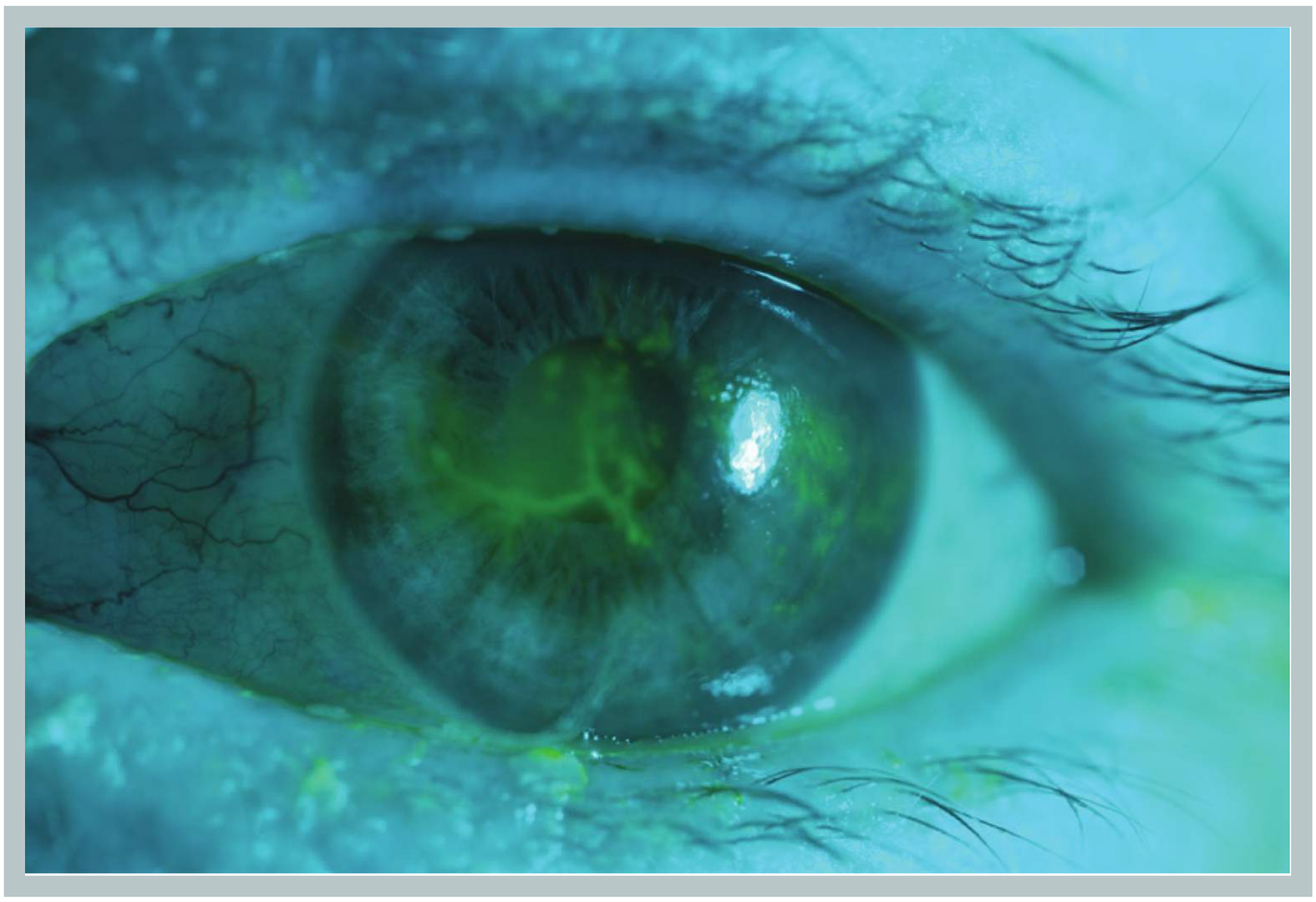
FIGURE 1.59 ■ **Temporal Lobe Contusion.** A temporal lobe contusion is seen on the right. The quadrigeminal cistern is partially effaced suggesting early herniation. (Photo contributor: Lawrence B. Stack, MD.)

Chapter 2

OPHTHALMIC CONDITIONS

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Herpes Simplex Keratitis. Branched dendritic lesion seen on the cornea in a patient with HSV. (Photo contributor: Lawrence B. Stack, MD.)

The authors acknowledge the special contribution of Marc E. Levsky, MD, and Paul DeFlorio, MD, for portions of this chapter written for the previous editions of this book.

NEONATAL CONJUNCTIVITIS (OPHTHALMIA NEONATORUM)

Clinical Summary

Neonatal conjunctivitis is acquired either during birth with passage through the mother's cervix and vagina, or from cross-infection in the neonatal period. Presenting symptoms for Neisseria gonorrhoeae infection include a hyperacute bilateral conjunctivitis with copious purulent discharge, lid swelling, chemosis, and preauricular adenopathy.

More common etiologies include Chlamydia trachomatis, viruses (herpes simplex), and bacteria (Staphylococcus aureus, Streptococcal pneumoniae, Haemophilus species). For chlamydial conjunctivitis, the clinical features range from mild swelling with a watery discharge to marked lid swelling with a red and thickened conjunctiva with a blood-stained discharge. Fluorescein staining of herpes simplex conjunctivitis demonstrates epithelial dendrites.

Management and Disposition

With any form of neonatal conjunctivitis, Gram stain and culture are indicated. Scrapings of the palpebral conjunctiva are more likely to be rewarding than examination of the discharge itself. Begin treatment in the emergency department (ED) and admit newborns with suspected gonococcal conjunctivitis. Evaluate concurrently for C trachomatis, since coinfection is common.

Treatment for chlamydial conjunctivitis is based upon a positive diagnostic test. While culture is the gold standard, nucleic acid amplification tests, despite lacking FDA approval, are reported to perform similarly. Untreated disease can result in corneal and conjunctival scarring. Bacterial neonatal conjunctivitis that is neither gonococcal nor chlamydial may be treated with erythromycin antibiotic ointment and should be reevaluated in 24 hours.

Herpes simplex conjunctivitis is treated with intravenous (IV) acyclovir and topical trifluridine. Despite the appearance of a localized herpes infection, there is high risk for central nervous system (CNS) or disseminated infection.

Evaluation of the newborn's parents should be undertaken in neonatal conjunctivitis due to Gonococcus, Chlamydia, or herpes simplex virus (HSV).

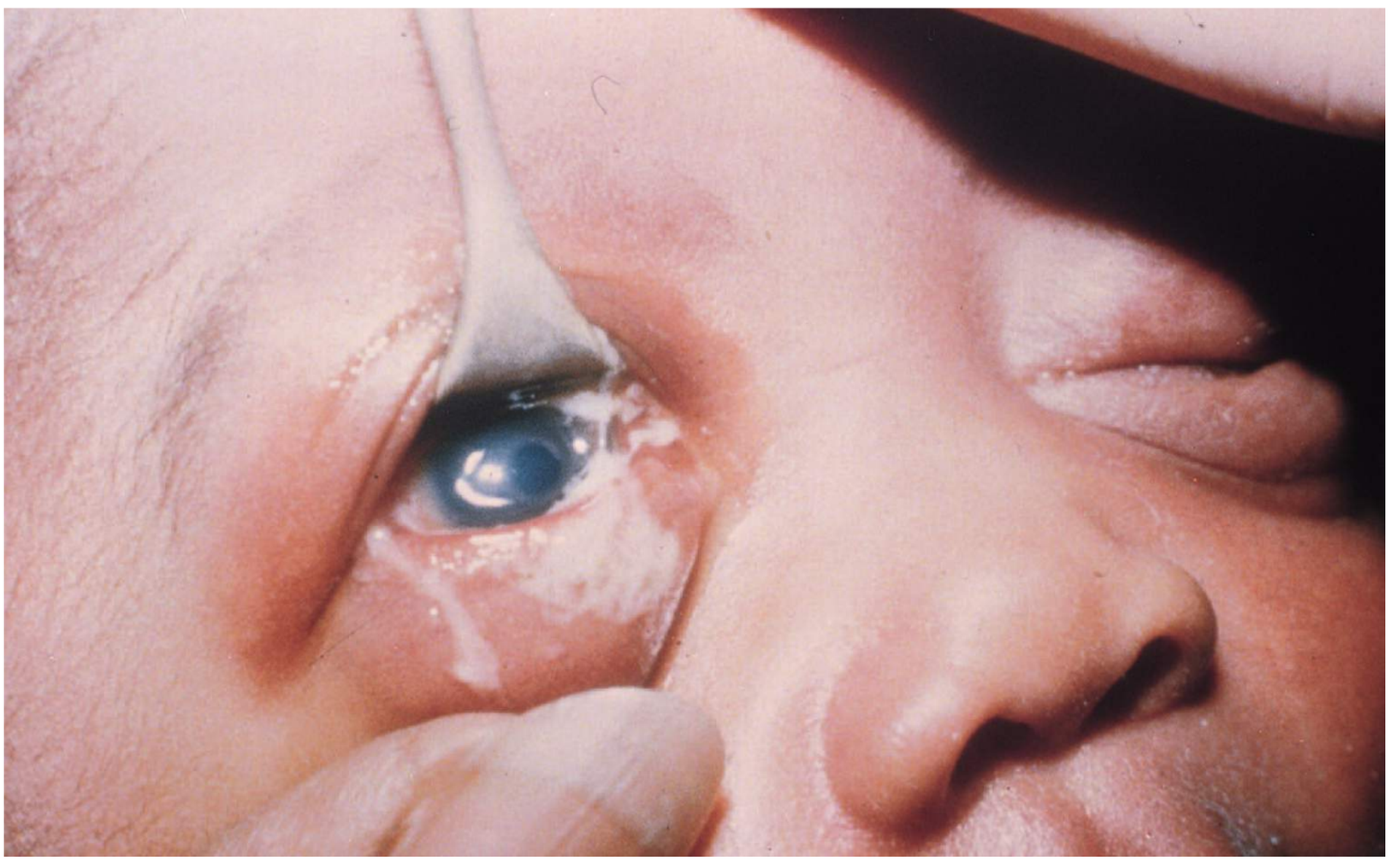


FIGURE 2.1 ■ **Neonatal Conjunctivitis (Ophthalmia Neonatorum).** Copious purulent drainage in a newborn with neonatal gonococcal conjunctivitis. (Reprinted with permission of the American Academy of Ophthalmology. Eye Trauma and Emergencies: A Slide-Script Program. San Francisco, 1985.)



FIGURE 2.2 ■ **Neonatal Conjunctivitis.** Thick purulent drainage in a newborn diagnosed with neonatal gonococcal conjunctivitis. (Photo contributor: David Effron, MD.)



FIGURE 2.3 ■ **Neonatal Conjunctivitis.** A purulent discharge is seen in this newborn. Management includes excluding Neisseria and Chlamydia. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 2.4 ■ **Neonatal Conjunctivitis.** A scant crusty discharge is seen in this newborn who was diagnosed in follow-up with nasolacrimal duct obstruction. (Photo contributor: Kevin J. Knoop, MD, MS.)

Pearls

1. The "rule of fives" may help predict the most likely bacterial cause.

0-5 days	Gonococcus
5 days-5 weeks	Chlamydia
5 weeks-5 years	Staphylococcus, Streptococcus, Haemophilus

2. Blindness can result from gonococcal eye infection in the neonate because the organism can invade the cornea. It is one of the few emergency conjunctival infections.
3. Nasolacrimal duct obstruction is common (up to 20%) in newborns and may present with findings suggestive of conjunctivitis. It is a diagnosis of exclusion in the neonate.
4. Advise parents that infants treated with macrolides are at risk for developing hypertrophic pyloric stenosis.

TABLE 2.1 ■ SUMMARY OF NEONATAL CONJUNCTIVITIS

Etiologic Agent	Time of onset	Clinical Features	Treatment
Chemical	Day 1	Common with silver nitrate; erythromycin more commonly used now for this reason	Self-limited
N gonorrhoeae	Day 2-7	May cause hyperacute disease with profuse discharge	Ceftriaxone IV. Caution in hyperbilirubinemia. Topicals alone inadequate
C trachomatis	Day 3-14	Clinical severity varies. Common cause of blindness worldwide	Erythromycin PO Azithromycin (alternate)
HSV 1,2	Day 2-16	Should be suspected if child has any vesicular lesions on body	Vidarabine or trifluridine topically; consider systemic acyclovir
Other bacterial organisms	Day 2 and up	Empiric treatment based on Gram stain	Erythromycin ointment for gram positive; gentamicin or tobramycin ointment for gram negative

BACTERIAL CONJUNCTIVITIS

Clinical Summary

Bacterial conjunctivitis is characterized by the acute onset of conjunctival injection and a thick yellow, white, or green mucopurulent drainage. Lid edema, erythema, and chemosis may also be seen. S aureus is the most common causative bacteria. Streptococcus pneumoniae and Haemophilus influenzae occur more frequently in children.

Hyperacute bacterial conjunctivitis, the most severe form of acute purulent conjunctivitis, is associated with N gonorrhoeae. Symptoms are hyperacute in onset, and findings include a purulent, thick, copious discharge, eyelid swelling and tenderness, marked conjunctival hyperemia, chemosis, and preauricular adenopathy. The condition is serious and threatens sight because Neisseria species are capable of invading an intact corneal epithelium. Corneal findings include epithelial defects, marginal infiltrates, and an ulcerative keratitis that can progress to perforation.

Management and Disposition

Encourage frequent hand washing and warm moist compresses. Empiric antibiotic choices include polymyxin/trimethoprim drops and erythromycin ophthalmic ointment. Other options include bacitracin, sulfacetamide, or polymyxin-bacitracin ointments or fluoroquinolone or azithromycin drops. Improvement should be noted in 1 to 2 days. Patients who do not improve should be referred to an ophthalmologist.

Fluoroquinolones should be prescribed for contact lens wearers (and keratitis ruled out) due to concern for Pseudomonas infection. Hyperacute bacterial conjunctivitis requires immediate ophthalmologic consultation and hospitalization.



FIGURE 2.5 ■ **Bacterial Conjunctivitis.** Mucopurulent discharge, conjunctival injection, and lid swelling in a 10-year-old with H influenzae conjunctivitis. (Photo contributor: Frank Birinyi, MD.)



FIGURE 2.6 ■ **Bacterial Conjunctivitis.** Mucopurulent discharge and conjunctival injection in an adult with conjunctivitis. (Photo contributor: Lawrence E. Heiskell, MD.)

Pearls

1. Conjunctivitis is a common presentation of the red eye, and should be considered after more serious causes, such as acute angle-closure glaucoma (ACG), iritis, and infectious keratitis, have been ruled out.
2. Red flags include a significantly decreased visual acuity, ciliary flush, and corneal opacity.
3. Worsening symptoms during topical treatment with Neosporin or a sulfonamide suggest a contact allergic reaction.
4. N gonorrhoeae conjunctivitis must be considered in the sexually active adult with a prominent, thick, copious discharge. Urethritis is usually present.
5. Fluoroquinolones should be prescribed for contact lens wearers because of concern for Pseudomonas infection in these patients.

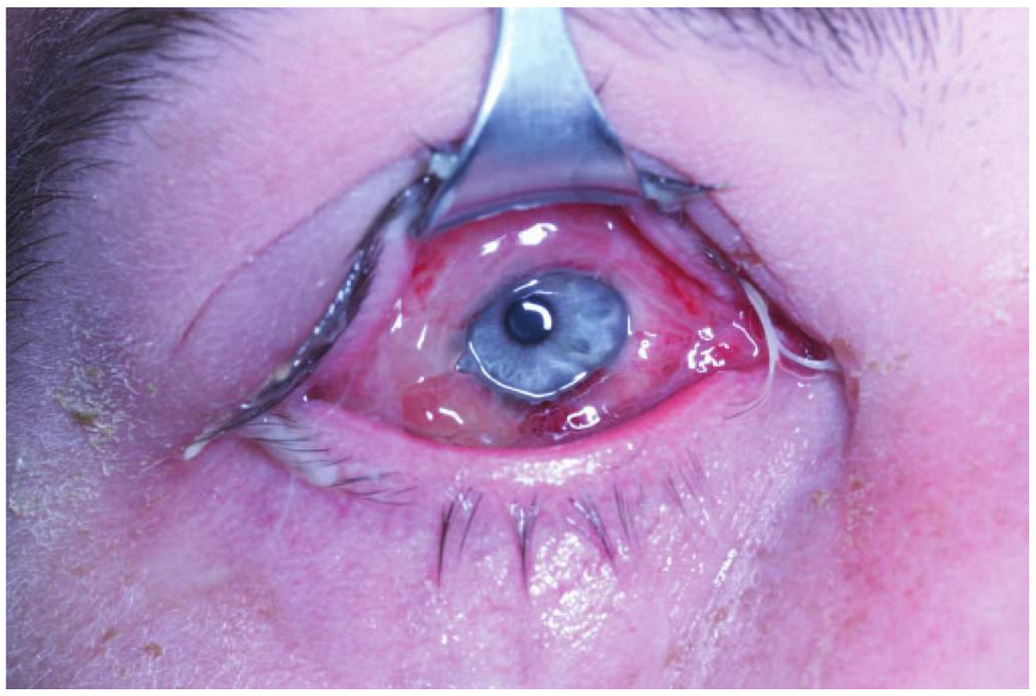


FIGURE 2.7 ■ **Gonococcal Conjunctivitis.** Right eye chemosis, purulent discharge, and pain in young adult male with urethritis. (Photo contributor: Catherine Burger, MD.)

Clinical Summary

Viral conjunctivitis is a common presentation of the red eye. Findings are mild and include a thin watery discharge, crusting in the morning, burning or irritation, conjunctival injection (typically diffuse), and lid edema. The tarsal conjunctiva may appear bumpy secondary to hyperplastic lymphoid tissue (follicles). Preauricular adenopathy may be present. The visual acuity is normal. The infection usually begins in one eye, but both eyes usually become involved due to autoinoculation. There are few to no systemic complaints. Adenovirus is the most common virus. A point of care test now available may aid clinicians to avoid empiric antibiotic therapy.

Epidemic keratoconjunctivitis (EKC) is a severe and highly contagious adenovirus infection that also involves the cornea. Additional features may include foreign body sensation, photophobia, and pseudomembranes overlying the palpebral conjunctiva. By the eighth day, a painful punctate keratitis that stains with fluorescein may develop; by the end of the second week, these are replaced by white macular subepithelial infiltrates located in the central cornea (that no longer stain). These may cause a decrease in visual acuity, but eventually resolve spontaneously.

Pharyngoconjunctival fever, usually caused by adenovirus type 3, is highly contagious and should be considered if there is associated upper respiratory tract infection and fever.

Management and Disposition

Viral conjunctivitis is self-limited and usually mild. Warm or cool compresses may be helpful. Careful hand washing by patient and staff is important. Over-the-counter topical antihistamines (Naphcon A) may provide symptomatic relief. The eye irritation and discharge should improve after 5 to 7 days, but may take 2 to 3 weeks for complete resolution of all symptoms. Antivirals and antibiotics are ineffective. Patients with keratitis should follow up with an ophthalmologist.

Pearls

1. A nonspecific adenovirus conjunctivitis will improve after 5 to 7 days and resolve in 10 to 14 days, but a virulent adenovirus causing EKC will peak in 5 to 7 days and may last 3 to 4 weeks.
2. Consider and rule out serious causes of red eye (acute ACG, iritis).
3. Frequent hand washing and the use of separate linens are advised for patients and family members.
4. Fastidious hand and equipment hygiene is necessary to prevent nosocomial transmission, as adenovirus can be recovered for extended periods of time from these surfaces.



FIGURE 2.8 ■ **Viral Conjunctivitis.** Note the characteristic asymmetric conjunctival injection. Symptoms first developed in the left eye, with symptoms spreading to the other eye a few days later. A thin watery discharge is also seen. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 2.9 ■ **Epidemic Keratoconjunctivitis (EKC).** Diffuse injection of the bulbar conjunctiva is seen in addition to a papillary reaction of the palpebral conjunctiva—a classic finding in EKC. (Photo contributor: Department of Ophthalmology, Naval Medical Center, Portsmouth, VA.)

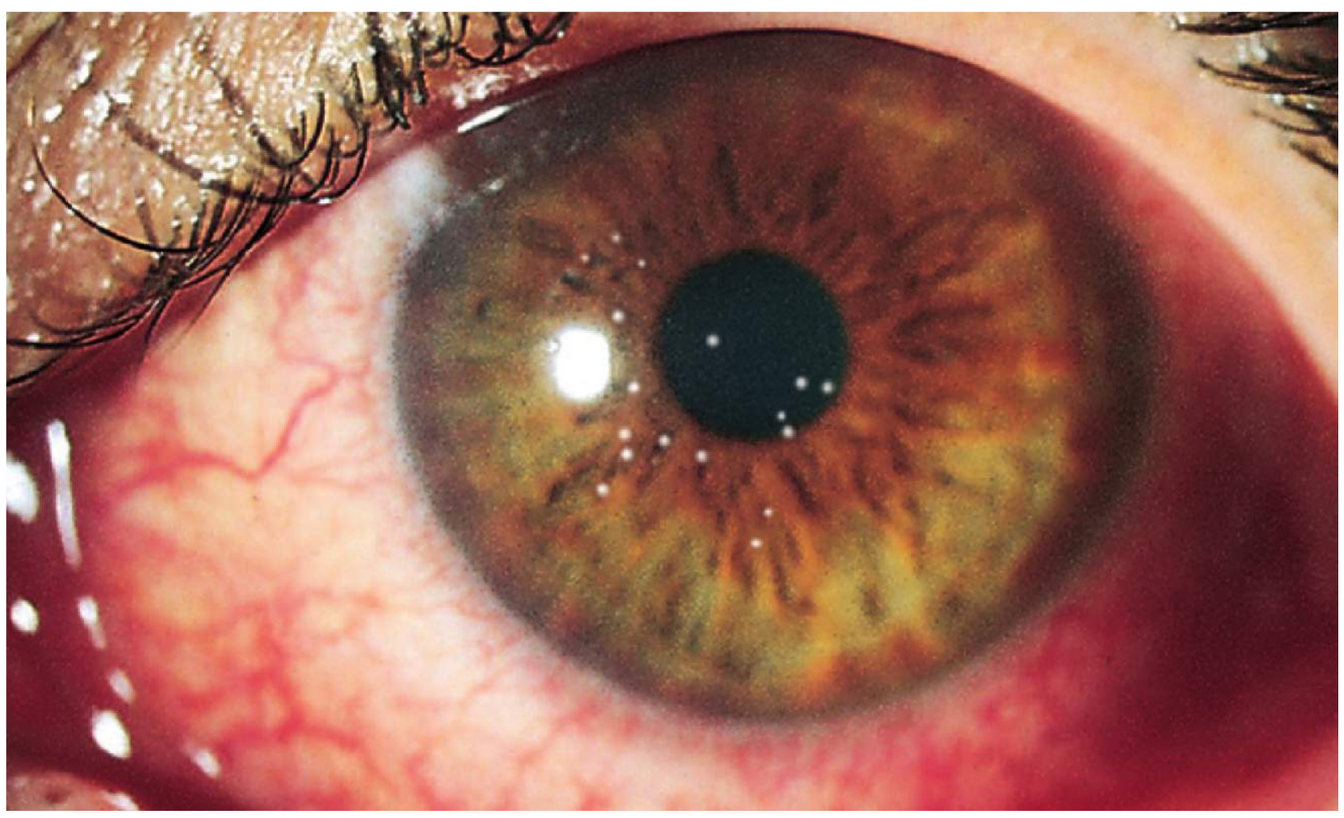


FIGURE 2.10 ■ **Subepithelial Infiltrates (EKC).** These usually develop in the second week. (Photo contributor: Katrina C. Santos, MD.)

Clinical Summary

Allergic conjunctivitis is a condition whereby airborne allergens precipitate type 1 IgE-mediated hypersensitivity reactions in the conjunctiva. Allergens include pollens, animal dander, mites, mold, and dust. Approximately 50% of patients have a personal or family history of allergic conditions such as atopy, eczema, asthma, and allergic rhinitis.

Itching is the hallmark symptom. Associated clinical features include conjunctival injection and edema, burning, discharge (clear, white, or mucopurulent), chemosis, and eyelid redness and swelling. Small papillae may be seen on the tarsal conjunctiva.

Vernal conjunctivitis is a rare but serious form of allergic conjunctivitis. The highest incidence is seen in the arid areas of the Middle East and North Africa secondary to wind and dust storms. Symptoms are similar to allergic conjunctivitis, but are more intense. Itching is severe and a vigorous knuckle rubbing is a typical observation. Giant, raised, pleomorphic papillae ("cobblestones") seen over the upper tarsal plate are pathognomonic.

Management and Disposition

The initial approach is to eliminate the allergen. Avoiding animal dander, using air conditioners with appropriate filters, and limiting time outdoors will improve the condition. Topical tear substitutes are effective to dilute or wash away the allergen. H1 antihistamine-vasoconstrictor combinations such as over-the-counter Naphcon A are recommended to relieve mild itching and redness. For more severe or frequent attacks, olopatadine, an antihistamine with mast cell stabilizing properties, is more effective. Mild topical steroids are an option only after consultation with an ophthalmologist.

Options for vernal conjunctivitis include cromolyn, aspirin, and cold compresses. Topical cyclosporin may be useful in resistant cases.

Pearls

1. Itching is the hallmark symptom of allergic conjunctivitis.
2. Itching is not common in nonallergic conjunctivitis.
3. Symptoms are usually bilateral.
4. Naphcon A and olopatadine are effective in treating allergic conjunctivitis.
5. Complications of topical corticosteroid use include glaucoma, cataract formation, secondary infection, and corneal perforation.

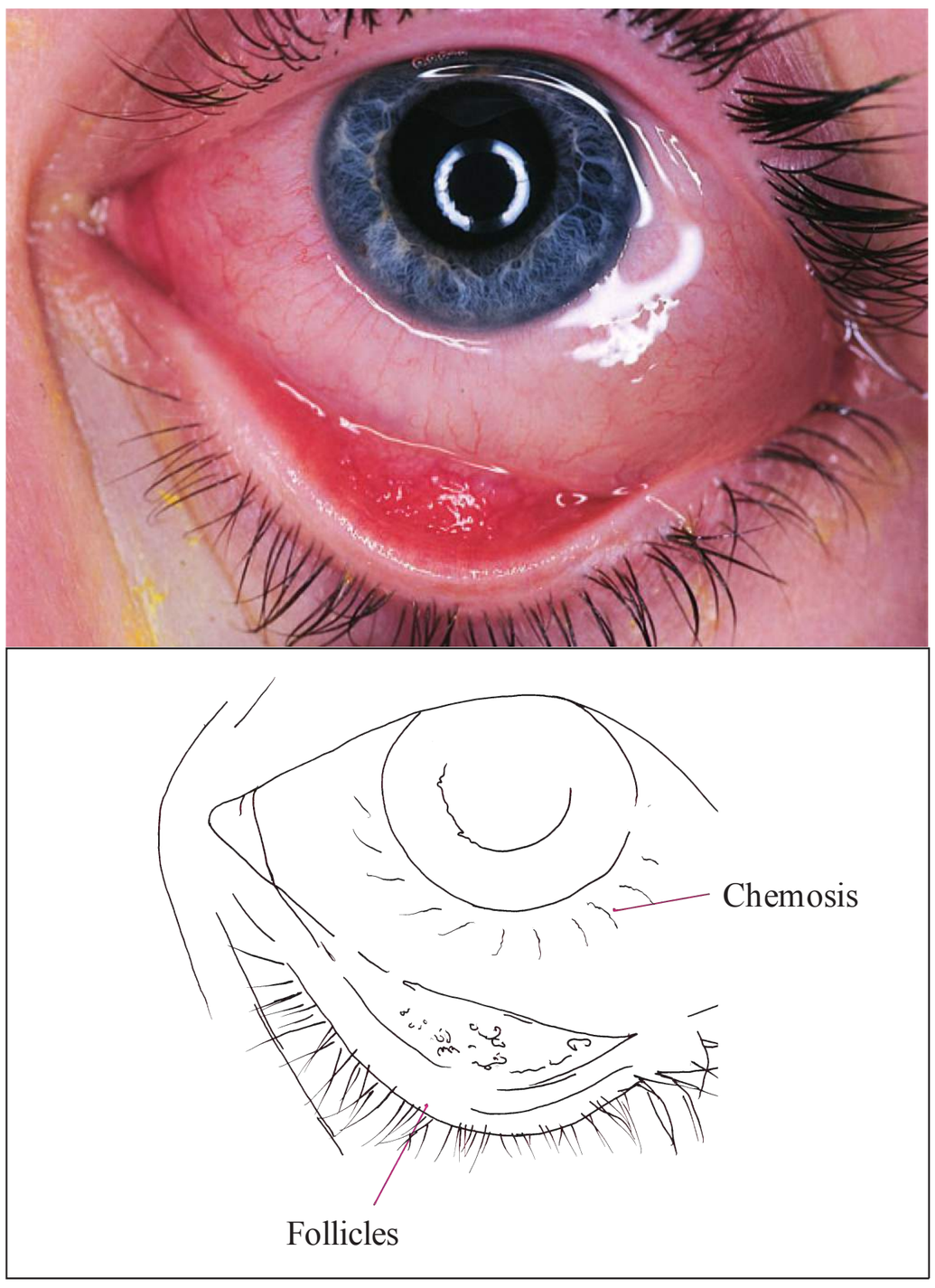


FIGURE 2.11 ■ **Allergic Conjunctivitis.** Conjunctival injection, chemosis, and a follicular response in the inferior palpebral conjunctiva are seen in this patient with allergic conjunctivitis secondary to cat fur. (Photo contributor: Timothy D. McGuirk, DO.)



FIGURE 2.12 ■ **Vernal Conjunctivitis.** The tarsal conjunctiva demonstrates giant papillae and a cobblestone appearance pathognomonic for vernal conjunctivitis. (Photo contributor: William Beck, CRA.)

DACRYOCYSTITIS

Clinical Summary

Dacryocystitis is an inflammation of the lacrimal sac, positioned immediately distal to the canaliculi and proximal to the nasolacrimal duct. Inflammation is usually secondary to obstruction of the nasolacrimal duct. Clinical findings include swelling over the lacrimal sac, redness, tearing, eyelash matting and crusting, and conjunctival redness. Tears and mucopurulence may be expressed from the punctum when pressure is applied over the lacrimal sac. Complications include conjunctivitis and orbital or preseptal cellulitis.

Up to 20% of normal newborns have a closed nasolacrimal passage, and 90% spontaneously open within the first 6 months.

Management and Disposition

Ophthalmologic consultation is recommended. The most common organisms isolated in children are S aureus, Staphylococcus epidermidis, and α-hemolytic Streptococci. Oral clindamycin for 7 to 10 days is recommended for outpatient management. Febrile and acutely ill patients require IV vancomycin in combination with a third-generation cephalosporin.

Treatment of nasolacrimal duct obstruction is managed initially with downward lacrimal sac massage ("Crigler" massage) 2 to 3 times a day. Unresolved nasolacrimal duct obstruction requires lacrimal duct probing by the ophthalmologist.

Pearls

1. Nasolacrimal duct obstruction is the most common cause of persistent tearing and ocular discharge in children.
2. Swelling is localized to the extreme nasal aspect of the lower lid and may be confused with a hordeolum. Occasionally, conjunctival redness may be present.
3. Dacryocystitis may be confirmed by pressure on the lacrimal sac and the reflux of tears and purulent material from the punctum. The lacrimal sac and lacrimal fossa lie in the inferior medial aspect of the orbit, not on the side of the nose.
4. Urgent referral should be made for any signs of orbital cellulitis. These include proptosis, limitation of extraocular movements, and loss of vision.



FIGURE 2.13 ■ **Dacryocystitis.** Swelling and erythema over the medial lower lid and lacrimal sac developed in this 10-year-old patient with streptococcal pharyngitis. (Photo contributor: Kevin J. Knoop, MD, MS.)

Clinical Summary

Acute dacryoadenitis typically involves children and young adults with associated systemic infections such as gonorrhea, mumps, Epstein-Barr virus, and Staphlyococcus species. Findings are localized to the outer one-third of the upper eyelid and include fullness or swelling, erythema, and tenderness. A characteristic "S"-shaped deformity with ptosis of the lid may be seen. In more advanced cases proptosis, inferonasal globe displacement, ophthalmoparesis, and diplopia may be present.

Chronic dacryoadenitis is more common, is seen in older patients, and is usually due to tumor or associated inflammatory disorders such as sarcoidosis, Sjögren syndrome, or IgG4-related diseases.

Management and Disposition

For acute dacryoadenitis, amoxicillin-clavulanate or IV ampicillin-sulbactam are used, depending on the severity and patient's toxicity. In cases of dacryoadenitis due to mumps or Epstein-Barr virus, warm compresses are recommended. Resolution occurs spontaneously. Patients should return to the ED for symptoms suggestive of orbital cellulitis such as decreased ocular motility or proptosis.

Treatment of chronic dacryoadenitis involves treatment of the underlying disorder.

Nonemergent ophthalmology follow-up is appropriate.

Pearls

1. Swelling is localized over the lateral one-third of the upper lid and imparts an "S"-shaped curve to the lid margin.
2. Acute dacryoadenitis is usually seen as a complication of mumps, with (bilateral) parotid swelling.
3. Chronic dacryoadenitis is more common, and is seen in older patients. Malignancy should be considered.
4. IgG4-related disease should be considered in patients (particularly middle-aged and older men) with bilateral disease and either salivary gland enlargement or pancreatitis of unknown origin.
5. Urgent referral is recommended for patients with diplopia, limitation of the extraocular muscles, or reduction of vision.



FIGURE 2.14 ■ **Dacryoadenitis.** Unilateral localized swelling and chemosis are present laterally secondary to inflammation of the lacrimal gland. (Used with permission from the American Academy of Ophthalmology. External Disease and Cornea: A Multimedia Collection. San Francisco, 1994.)

PTERYGIUM/PINGUECULA

Clinical Summary

Pterygium (Greek, pterygion meaning wing-like) is a benign proliferation of fibrovascular tissue. It usually originates in the nasal conjunctiva on the horizontal meridian of the limbus. It progressively encroaches onto the cornea and visual axis. Its appearance ranges from flat and white to thick, pink or red, and fibrovascular. Typically, it assumes a triangular appearance, the apex directed toward the pupil. Predisposing factors include exposure to ultraviolet light, wind, and dust. Older individuals living in warmer areas with high levels of sunlight are more likely to develop pterygia.

Pterygia may be asymptomatic or become inflamed, causing mild symptoms of irritation and foreign body sensation. Decreased visual acuity may result as the pterygium encroaches on the visual axis or if the lesion induces astigmatism.

Pinguecula (Latin, pingueculus meaning fatty) is a common degenerative lesion of the bulbar conjunctiva, also arising in the horizontal meridian. It appears as a light brown or yellow-white amorphous, slightly raised conjunctival tissue adjacent to the limbus. It too may be asymptomatic or become episodically inflamed; however, it may enlarge and become a pterygium.

Management and Disposition

Mild disease can be treated with artificial tears or a topical vasoconstrictor (Naphcon A). In more severe cases, consultation with an ophthalmologist is appropriate regarding topical steroids and topical nonsteroidal anti-inflammatory drugs (NSAIDs). Surgical excision by the ophthalmologist is indicated if the pterygium interferes with contact lens wear, encroaches significantly on the visual axis resulting in induced astigmatism or opacity, or restricts eye movement.

Pearls

1. Pterygia are more likely than pinguecula to be found on the nasal conjunctiva and bilateral.
2. Pterygia and pingueculae are found on the horizontal meridian. Conjunctival neoplasms often occur in axes other than the horizontal axis.
3. Following surgical excision, recurrence of pterygia is common.
4. Pterygium-mimics include localized conjunctival neoplasia, conjunctivitis, and episcleritis.

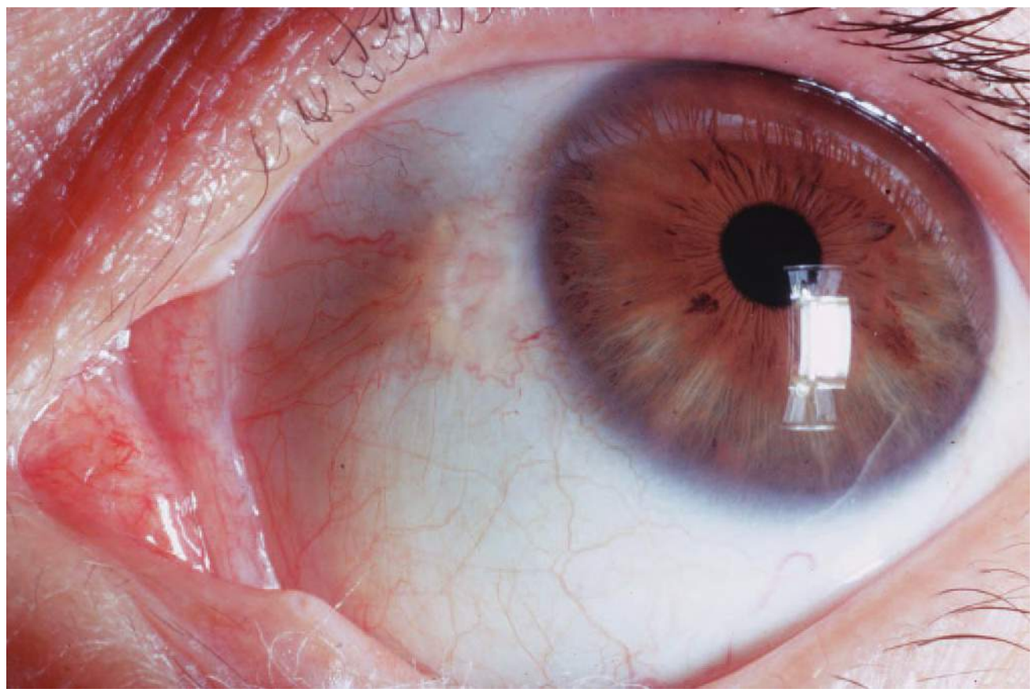


FIGURE 2.15 ■ **Pinguecula.** A small area of yellowish "heaped up" conjunctival tissue is seen adjacent to the nasal limbus. (Photo contributor: Department of Ophthalmology, Naval Medical Center, Portsmouth, VA.)

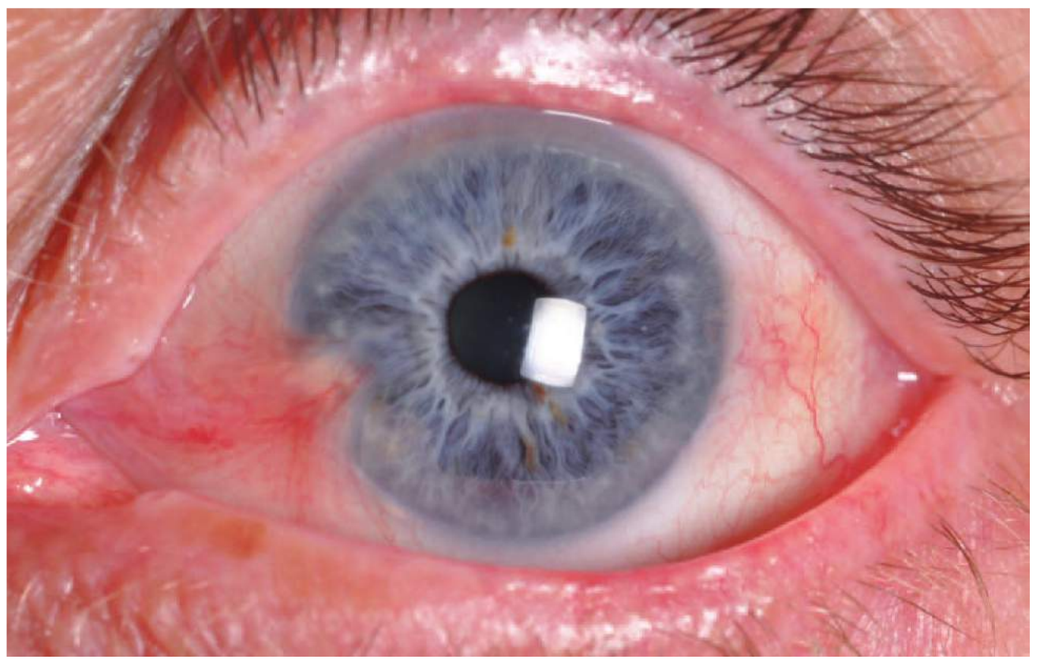


FIGURE 2.16 ■ **Pterygium.** Pterygium appears as a raised vascular triangular area of bulbar conjunctiva that encroaches onto the cornea. (Photo contributors: Top: Rebecca Kasl, MD; Bottom: Andrew J. Hendershot, MD.)

Clinical Summary

A hordeolum is a localized abscess involving the glands of the eyelid. An external hordeolum (stye) involves an eyelash (cilia) follicle or the tear glands of the lid margin (glands of Zeis, Moll). An internal hordeolum involves the meibomian glands. S aureus is the most frequent isolate. Clinical findings include focal swelling, edema, erythema, and tenderness.

A chalazion is a chronic localized inflammation that results from the obstruction of the meibomian glands. It may arise from a hordeolum. The lesion may point anteriorly (toward the skin of the eyelid) or posteriorly (toward the tarsal conjunctiva). It may become sufficiently large as to press on the globe and cause astigmatism. Pain and redness may be seen early in the course, but these may dissipate over time.

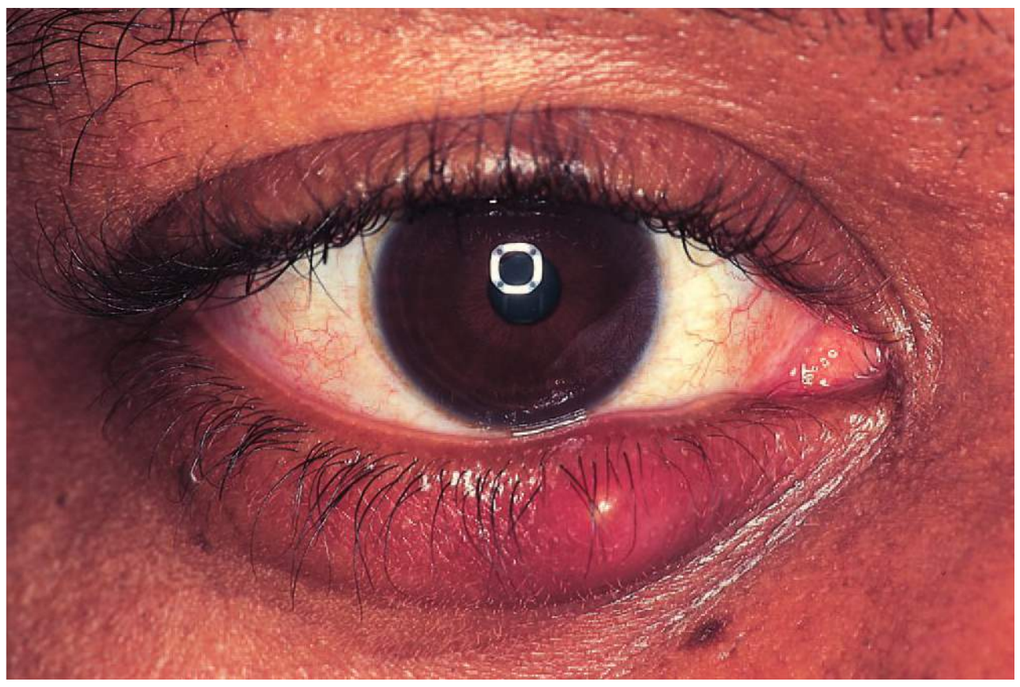


FIGURE 2.18 ■ **Hordeolum.** Focal swelling and erythema at the lid margin are seen in this hordeolum. (Photo contributor: Frank Birinyi, MD.)

Management and Disposition

Hordeola can be treated with warm compresses. The clinical course of a hordeolum is usually self-limited, with spontaneous drainage and resolution in 5 to 7 days. Topical antibiotics are not helpful. Systemic antibiotics are unnecessary unless there is concurrent preseptal cellulitis. If the mass persists or is large (distorting vision), ophthalmology referral is recommended for incision and curettage or intralesional corticosteroid injection.

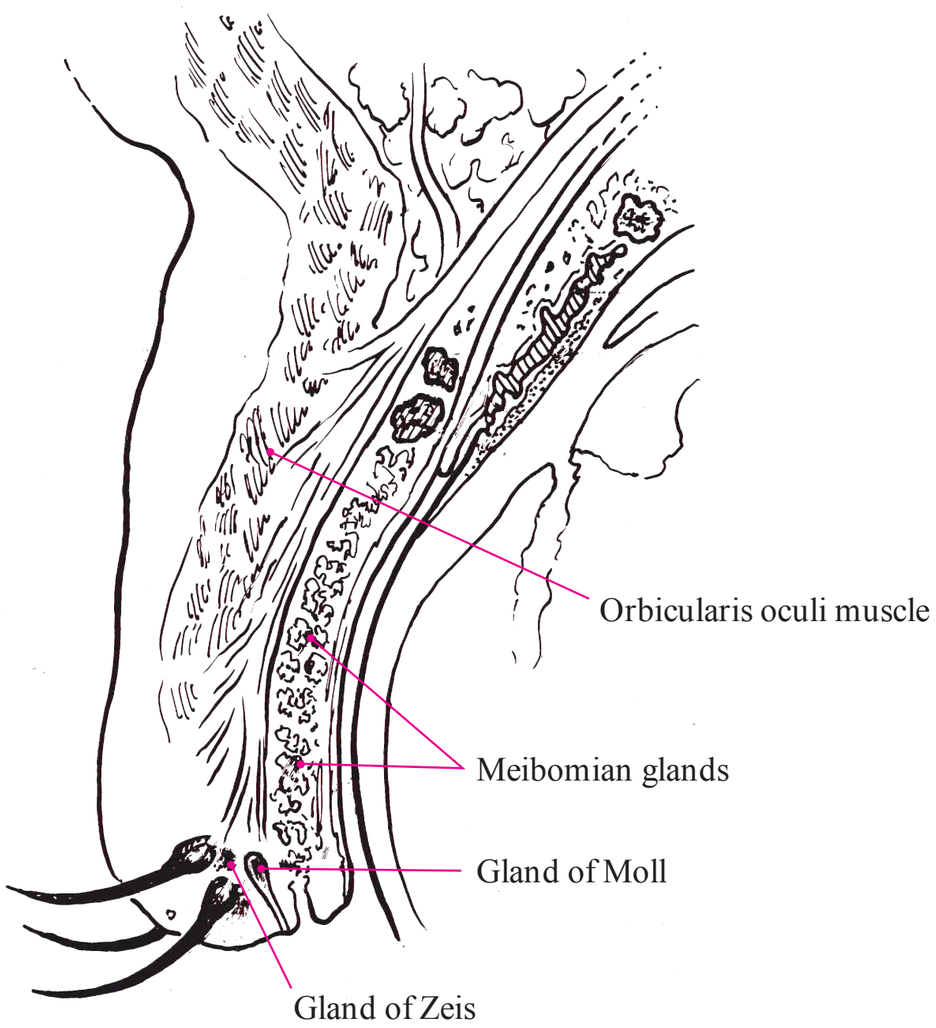


FIGURE 2.17 ■ **Eyelid Anatomy.** Anatomic structures related to eyelid pathology.

Frequent warm compresses applied to chalazia expedite spontaneous drainage. Most chalazia spontaneously clear after several weeks. Antibiotics are not indicated. Recalcitrant lesions should be referred to the ophthalmologist for incision and curettage or glucocorticoid injection.

Pearls

1. Uncomplicated hordeola and chalazia can be treated with warm compresses, and generally resolve spontaneously.
2. Patients with rosacea or marginal blepharitis (a chronic low-grade inflammation of the lid margins with crusts around the lashes) have frequent hordeola.
3. Older patients with recurrent chalazia should be referred to the ophthalmologist to rule out malignancy.



FIGURE 2.19 ■ **Chalazion.** This chalazion shows painful nodular focal swelling and erythema from meibomian cyst formation. (Photo contributor: Frank Birinyi, MD.)



FIGURE 2.20 ■ **Chalazion.** Two months of focal pain and swelling of the lateral upper lid. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 2.22 ■ **Ocular Herpes Simplex.** This 10-year-old has recurrent ocular herpes simplex since age 6. Vesicles should not be mistaken for hordeola. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 2.21 ■ **Chalazion.** A nontender bipartite chalazion is shown. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 2.23 ■ **Blepharitis.** The eyelid margins are inflamed and erythematous. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 2.24 ■ **Chalazion.** This lower lid chalazion was seen only with lid eversion. (Photo contributor: James Dahle, MD.)

Clinical Summary

Scleritis is painful, destructive, and potentially blinding. The pain is constant and boring and may radiate to the face and periorbital region. Associated features include tearing, photophobia, globe tenderness to palpation, and painful ocular movement.

The conjunctival vessels are injected. The eye itself may be intensely red with a violaceous or purple hue secondary to engorgement of the deep vessels of the episclera and scleral thinning. These deep vessels do not move when the overlying conjunctiva is moved with a cotton-tipped applicator, nor do they blanch with topical phenylephrine. On slit-lamp microscopy, the episcleral vessels are displaced outward by scleral edema. Corneal involvement, iritis (with cells and flare in the anterior chamber), and decreased visual acuity may accompany scleritis. An associated scleritis may occur with severe infectious keratitis. Primary infectious scleritis is rare. In either instance, topical and systemic antibiotics are indicated after appropriate cultures are obtained.

In up to 50% of cases, scleritis is associated with underlying autoimmune or infectious systemic disease, rheumatoid arthritis the most common. It occurs more frequently in women and in the fourth to sixth decades of life.

Management and Disposition

Ophthalmology consultation is required. Treatment varies according to underlying disease (if present), and can involve NSAID therapy, glucocorticoids, and immunosuppressive medications. Rheumatology consultation by the ophthalmologist is often required for optimal management in patients with underlying autoimmune disorders.

Pearls

1. Scleritis is associated with a systemic disease in approximately 50% of cases, most commonly rheumatoid arthritis.
2. Most cases of scleritis involve the anterior portion (anterior to the insertion of the medial and lateral rectus muscles).
3. Pain is exacerbated with ocular movements because the extraocular muscles insert into the sclera itself.
4. Anterior uveitis can occur in up to 40% of patients because the uvea is immediately adjacent to the sclera.
5. Check intraocular pressure (IOP) to rule out acute ACG as another cause of painful red eye.

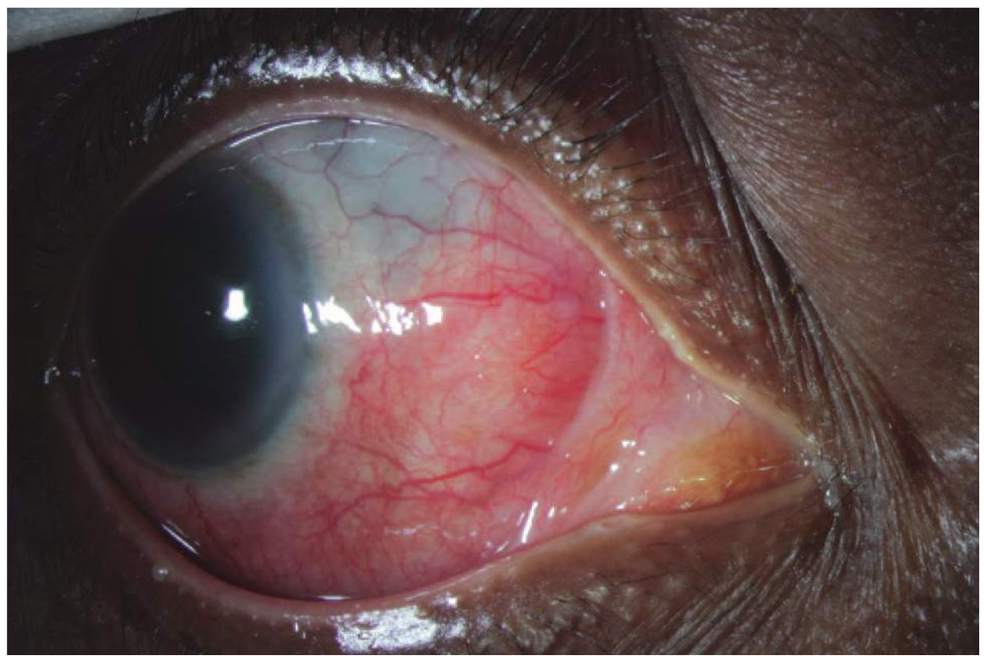


FIGURE 2.25 ■ **Scleritis.** A prominent generalized vascular injection is present. A bluish hue is also seen superiorly due to scleral thinning. These vessels do not move when the overlying conjunctiva is moved with a cotton-tipped applicator. (Photo contributor: Jeffrey Goshe, MD.)

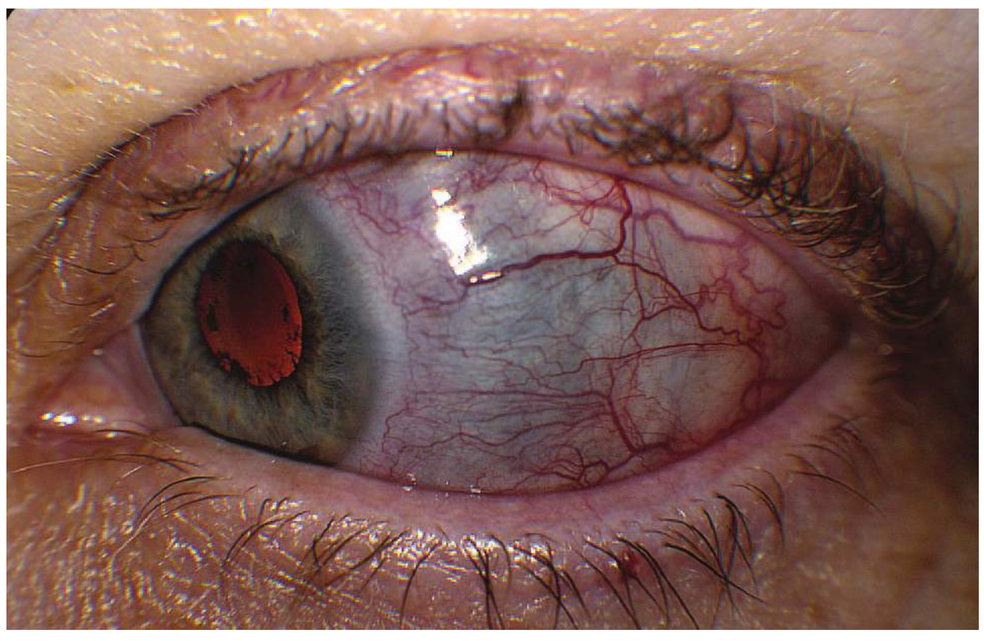


FIGURE 2.26 ■ **Scleritis.** A 55-year-old female with scleritis of the left eye associated with rheumatoid arthritis. Note dilation of the deep conjunctival and episcleral vessels and blue hue suggesting thinning of the sclera temporally. (Used with permission from Brice Critser, CRA, The University of Iowa and EyeRounds.org.)



FIGURE 2.27 ■ **Sectorial Scleritis.** Deep-boring pain experienced by this patient distinguishes this segmental area of erythema from episcleritis. (Photo contributor: Kevin J. Knoop, MD, MS.)

EPISCLERITIS

Clinical Summary

Episcleritis is a common and benign inflammation of the episclera, typically affecting young and middle-aged adults. Seventy percent of cases occur in females. The episclera lies just beneath the bulbar conjunctiva. Its vessels are large, run in a radial direction, and can be seen beneath the overlying conjunctiva. Episcleral and conjunctival vessels blanch with the use of topical 5% phenylephrine drops, unlike deep episcleral vessels.

Patients may complain of foreign body sensation, mild tenderness, irritation, mild photophobia, and excessive lacrimation. Pain is unusual but can occur, particularly in chronic cases. One-half of cases are bilateral. Eye findings are notable for a localized pink or bright red conjunctival injection, with involvement of the vessels in the superficial episcleral vascular plexus. Visual acuity is normal.

Episcleritis is usually an isolated condition, though it may be associated with a number of systemic diseases, including rheumatoid arthritis, inflammatory bowel disease, lupus, and vasculitis.

Management and Disposition

For many patients, the condition is self-limited and will resolve within 3 weeks, with or without treatment. For mild cases, use over-the-counter artificial tears. For those with recurrent or recalcitrant lesions, referral to the ophthalmologist is indicated.

Pearls

1. It is important to differentiate episcleritis from sight-threatening scleritis. Episcleritis presents with only mild pain and bright red episcleral vessels, which will blanch with topical phenylephrine.
2. Conjunctivitis is a more common cause of red eye, and symptoms usually include morning crusting. Injection that is localized rather than diffuse is more likely to be episcleritis.



FIGURE 2.28 ■ **Episcleritis.** A localized area of hyperemia is seen involving the temporal sclera. (Photo contributor: Robert Trieff, MD.)

Clinical Summary

Acute angle-closure glaucoma (ACG) is secondary to narrowing or closure of the anterior chamber angle, resulting in increased IOP, with subsequent damage to the optic nerve. Normally, aqueous humor drains out of the anterior chamber via Schlemm canal in the anterior chamber angle. In ACG, this flow is impeded, and the IOP rises from a normal range of 10 to 21 mm Hg to 50 mm Hg or higher.

ACG presents as an acutely inflamed eye. Nausea and vomiting are common and may be the presenting complaints. Eye pain and headache vary in severity. As the IOP reaches the 50 to 60 mm Hg range, fluid is forced into the cornea, resulting in corneal edema. Patients report blurred vision and rainbow-colored halos around lights. Clinical findings include tearing, perilimbal injection ("ciliary flush"), a cloudy ("steamy") cornea, a nonreactive mid-dilated pupil, anterior chamber inflammation, and an increased IOP. Using a penlight or slit-lamp microscopy, the anterior chamber may appear shallow.

Management and Disposition

ACG is an emergency that requires ophthalmology consultation, and treatment is directed at reducing the IOP. Aqueous outflow is increased by topical myotics (pilocarpine) that pull the peripheral iris taut away from the anterior chamber angle. Decreased production of aqueous is accomplished with topical β-blockers (timolol), an α-adrenergic agonist (apraclonidine), or a carbonic anhydrase inhibitor (acetazolamide or methazolamide systemically, dorzolamide topically). Osmotic agents reduce the aqueous volume within the eye and include glycerol, oral isosorbide, and IV mannitol. Latanoprost, a prostaglandin derivative, increases aqueous outflow through nontrabecular meshwork pathways.

The definitive treatment of ACG is laser peripheral iridectomy, done usually after the IOP normalizes.

Pearls

1. Vision loss and blindness can occur during the course of the attack (hours to days). ACG is a true ophthalmologic emergency.
2. Medicines with anticholinergic effects are capable of inducing pupillary dilation which might provoke an angle-closure attack. These include over-the-counter decongestants, motion sickness medications, antipsychotics, and antidepressants.
3. ACG often occurs in the evening, when lower light levels cause mydriasis.
4. ACG patients may easily be misdiagnosed initially as having a migraine headache or a CNS catastrophe because they may present with severe headache and vomiting.
5. The elevated IOP in acute ACG can be reduced pharmacologically by three mechanisms: first, by opening the closed angle with myotics; second, by reducing aqueous formation with β-blockers, α-agonists, or carbonic anhydrase inhibitors; and third, by reducing the aqueous volume within the eye by osmotic agents.

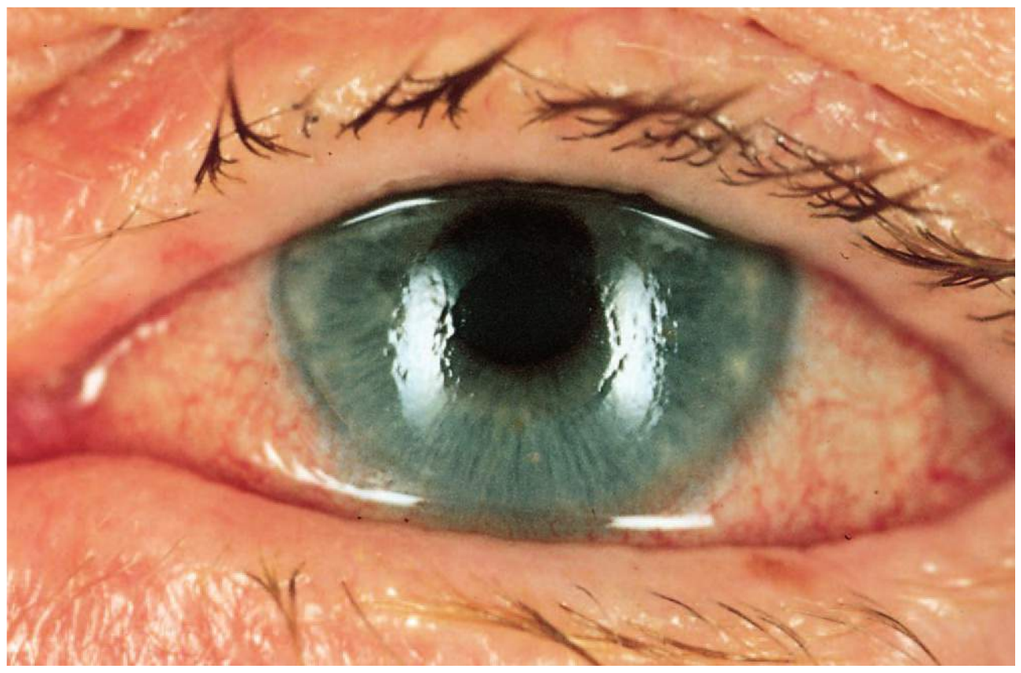


FIGURE 2.29 ■ **Acute Angle-Closure Glaucoma.** The cornea is edematous, manifest by the indistinctness of the iris markings and the irregular corneal light reflex. Conjunctival hyperemia is also present. (Photo contributor: Kevin J. Knoop, MD, MS.)

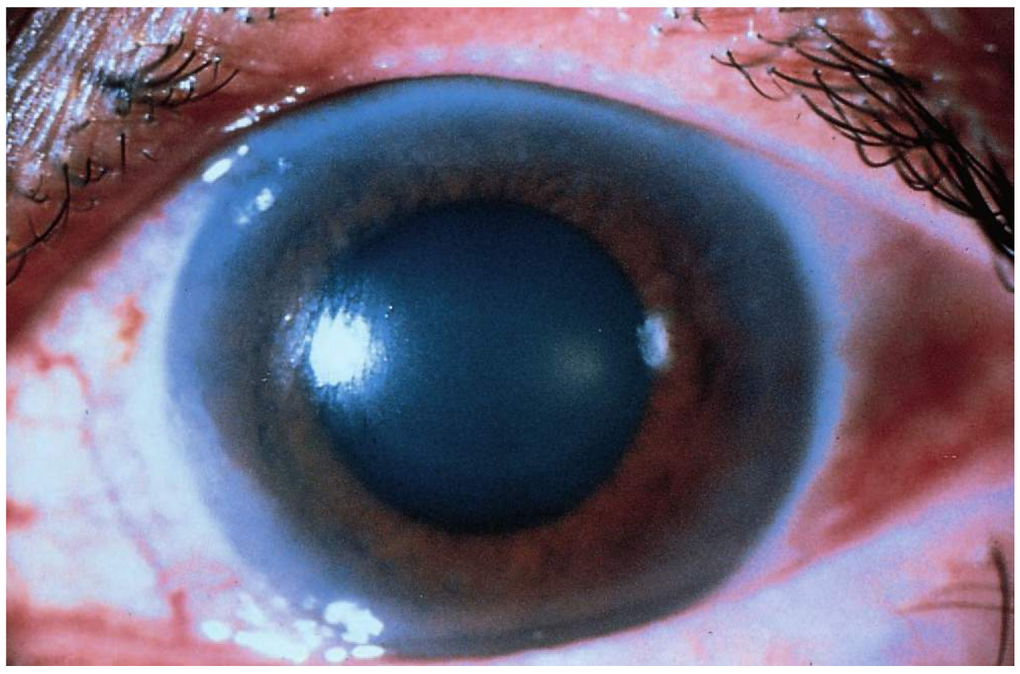


FIGURE 2.30 ■ **Acute Angle-Closure Glaucoma.** Note the cloudy or "steamy" appearance of the cornea and the midposition pupil. Conjunctival hyperemia is not as evident. (Photo contributor: Gary Tanner, MD.)

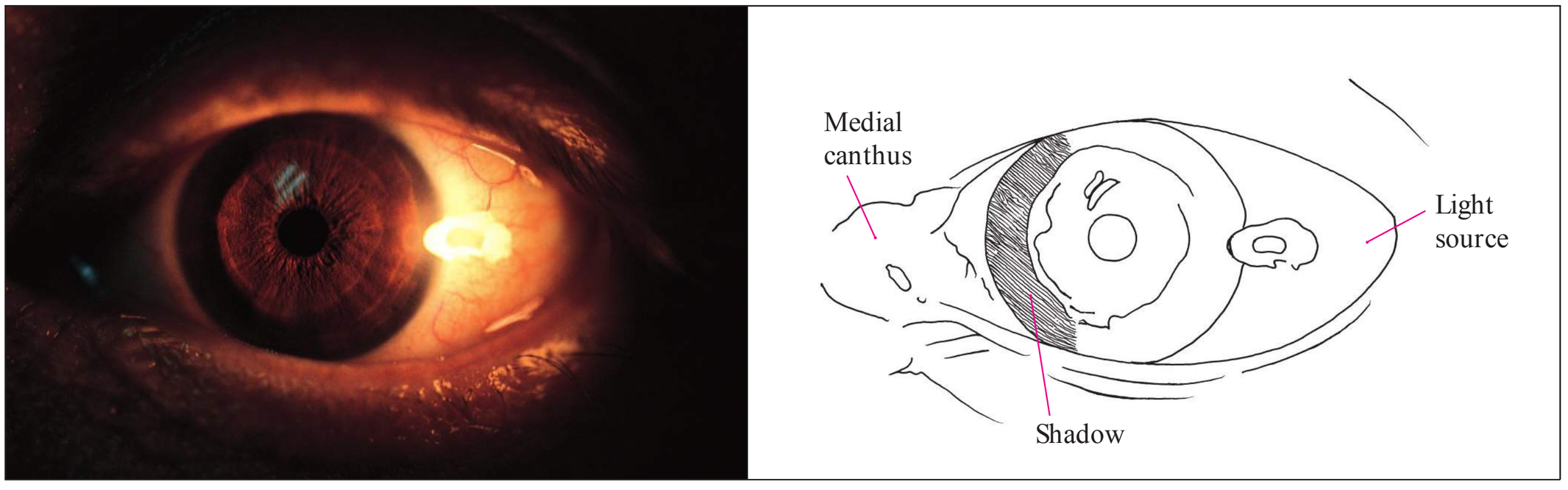


FIGURE 2.31 ■ **Penlight Test.** A penlight, held laterally and directed nasally, projects a shadow on the nasal side of an iris with a shallow anterior chamber. This patient presented with acute angle-closure glaucoma. (Photo contributor: Alan B. Storrow, MD.)

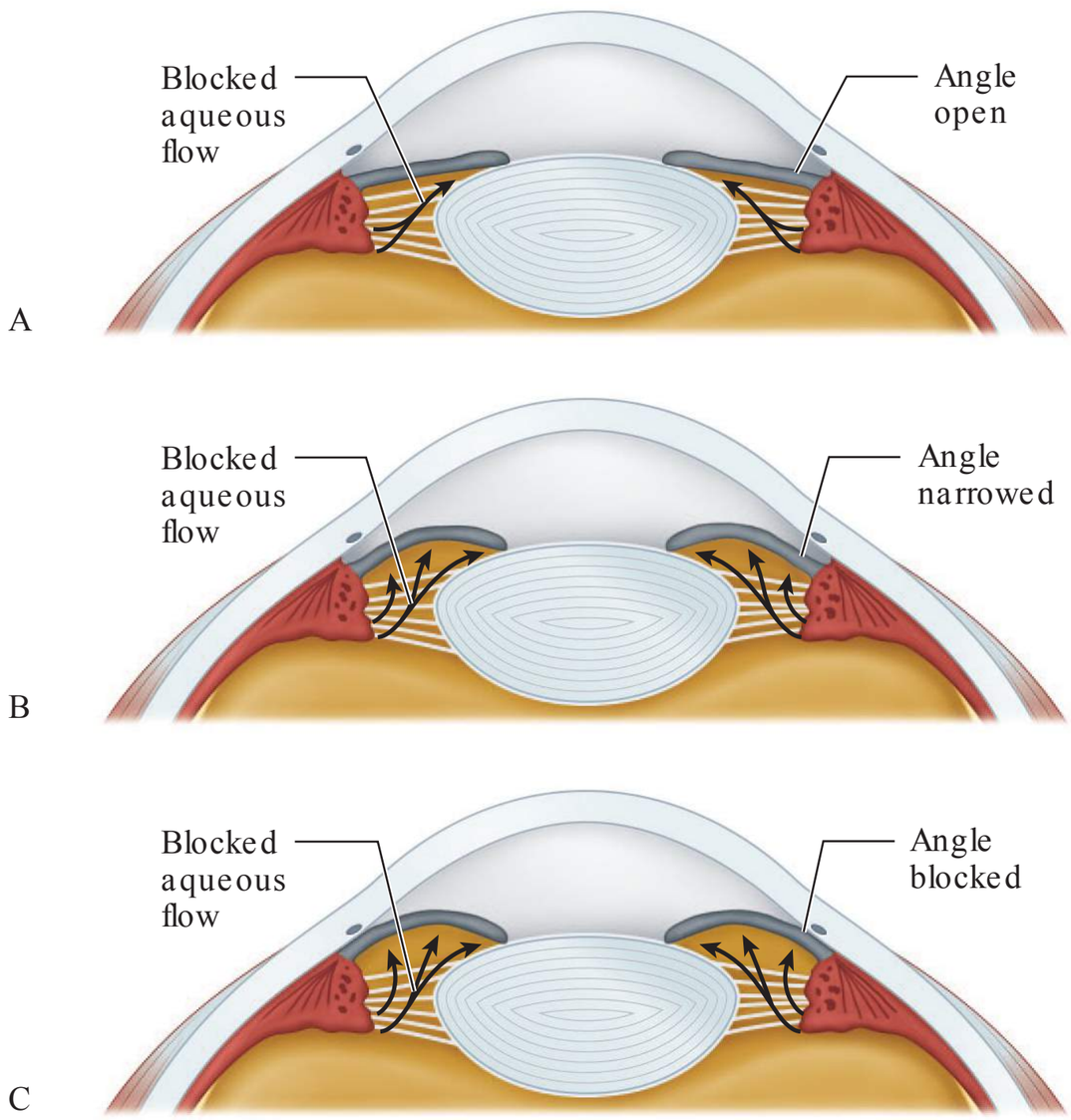


FIGURE 2.32 ■ **Iris Bombé.** (**A**) A physiologic block normally occurs where the posterior surface of the iris touches the lens. (**B**) When this block is increased, the iris as a result bows forward (iris bombé) and the angle is narrowed and pressure increases. (**C**) As pressure increases, the angle becomes blocked.

Clinical Summary

The uvea is the middle layer of the eye. Its anterior portion includes the iris and ciliary body; the posterior portion includes the choroid. Inflammation of the anterior portion is called anterior uveitis or iritis. It is often idiopathic, but approximately half of cases are associated with systemic disease. These include inflammatory disorders (rheumatoid arthritis, Behçet disease, sarcoid), HLA-B27-associated conditions (ankylosing spondylitis, inflammatory bowel disease, Reiter syndrome), and infectious causes (zoster, tuberculosis, toxoplasmosis, AIDS).

Clinical features include conjunctival hyperemia, hyperemic perilimbal vessels ("ciliary flush"), miosis, decreased visual acuity, photophobia, tearing, and pain. The slit-lamp may demonstrate a hypopyon, cells, flare, and keratic precipitates. Keratic precipitates are agglutinated inflammatory cells adherent to the posterior corneal endothelium. These precipitates appear either as fine gray-white deposits or as a large, flat, greasy-looking area ("mutton fat"). The IOP may be decreased due to decreased aqueous production by the inflamed ciliary body, or increased secondary to inflammatory debris within the trabeculae of the anterior chamber angle obstructing outflow.

Management and Disposition

The patient's history forms the basis for the evaluation and laboratory testing. The history should focus on rheumatic illness, dermatologic problems, bowel disease, infectious exposures, and sexual history. Treatment of the uveitis is nonspecific. Topical cycloplegics and corticosteroids may be prescribed in conjunction with the ophthalmologist. Antibiotics are not usually prescribed.

Pearls

1. Iritis is usually associated with a miotic pupil, pain, and redness primarily at the limbus ("ciliary flush").
2. When uveitis is associated with a systemic disorder, the associated condition is usually evident. Common exceptions include sarcoidosis and syphilis.
3. In patients with uveitis of unknown etiology, a chest x-ray looking for sarcoidosis and serologic testing for syphilis are reasonable.
4. Visual loss may occur with uveitis.
5. Topical analgesics do not significantly ameliorate the pain of anterior uveitis.
6. Consider sympathetic ophthalmia with unexplained uveitis and a history of eye trauma.



FIGURE 2.33 ■ **Anterior Uveitis**. Atraumatic left eye pain, photophobia, and limbal injection, in a middle-aged male with inflammatory bowel disease. Miosis is also present, a hallmark of uveitis. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 2.34 ■ **Anterior Uveitis**. Marked conjunctival injection and perilimbal hyperemia ("ciliary flush") are seen in this patient with recurrent iritis. (Photo contributor: Frank Birinyi, MD.)

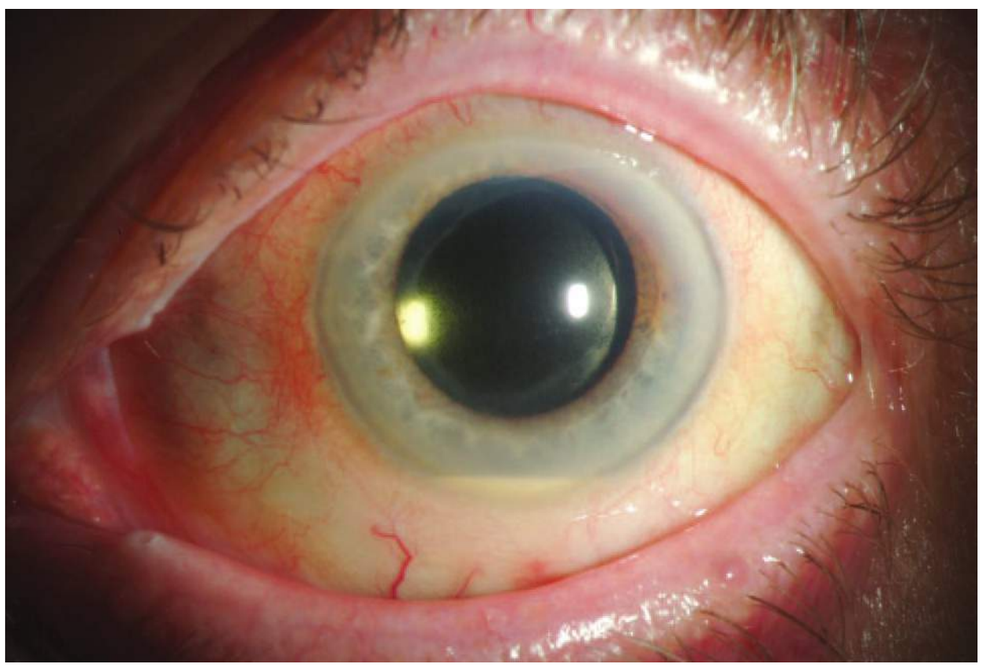


FIGURE 2.35 ■ **Hypopyon.** A thin layering of white blood cells is present in the inferior anterior chamber. (Used with permission from Brice Critser, CRA, The University of Iowa and EyeRounds.org.)

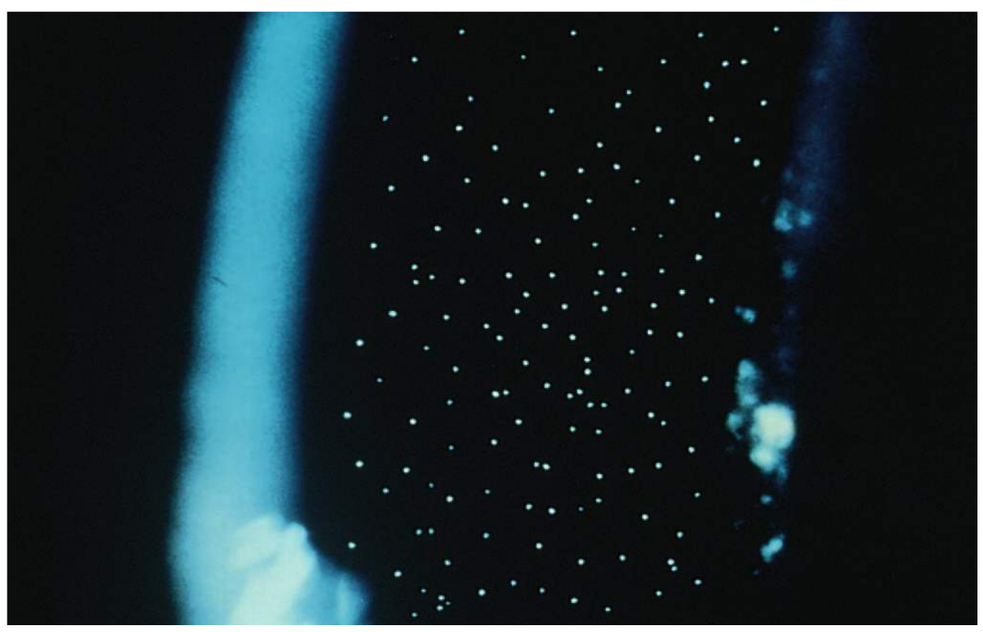


FIGURE 2.36 ■ **Anterior Chamber Cells**. Cells in the anterior chamber are a sign of inflammation or bleeding and appear similar to particles of dust in a sunbeam. They are best seen with a narrow slit-lamp beam directed obliquely across the anterior chamber. (Used with permission from Spalton DJ, Hitchings RA, Hunter PA (eds). Atlas of Clinical Ophthalmology. 2nd ed. London, UK: Mosby-Wolfe Limited; 1994.)

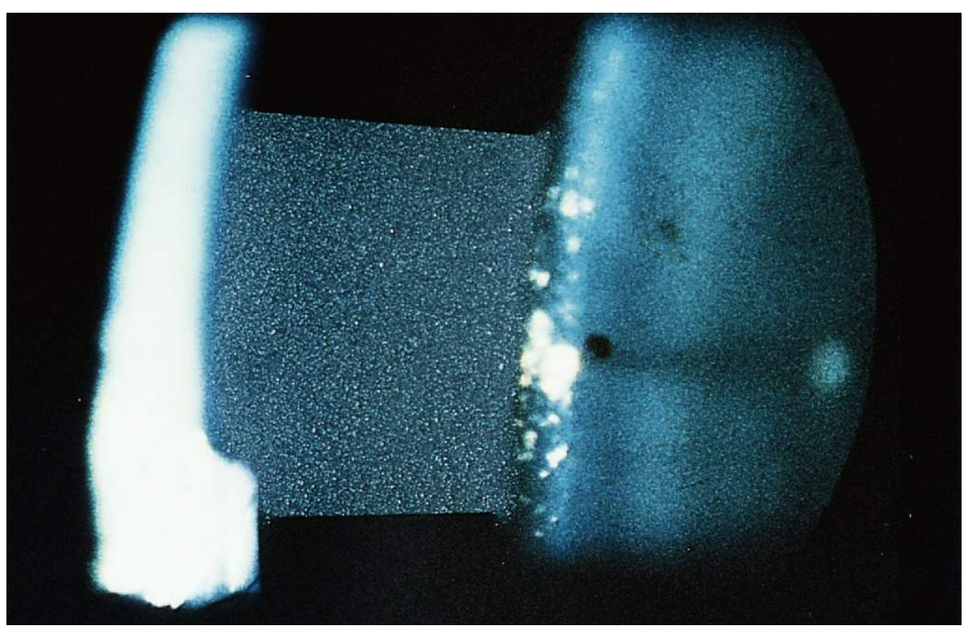


FIGURE 2.37 ■ **Anterior Chamber Flare.** Flare in the anterior chamber represents an elevated concentration of plasma proteins from inflamed, leaking intraocular blood vessels. Flare seen in a slit-lamp beam appears similar to a car headlight cutting through the fog. (Used with permission from Spalton DJ, Hitchings RA, Hunter PA (eds). Atlas of Clinical Ophthalmology. London, UK: Mosby-Wolfe Limited; 1984.)

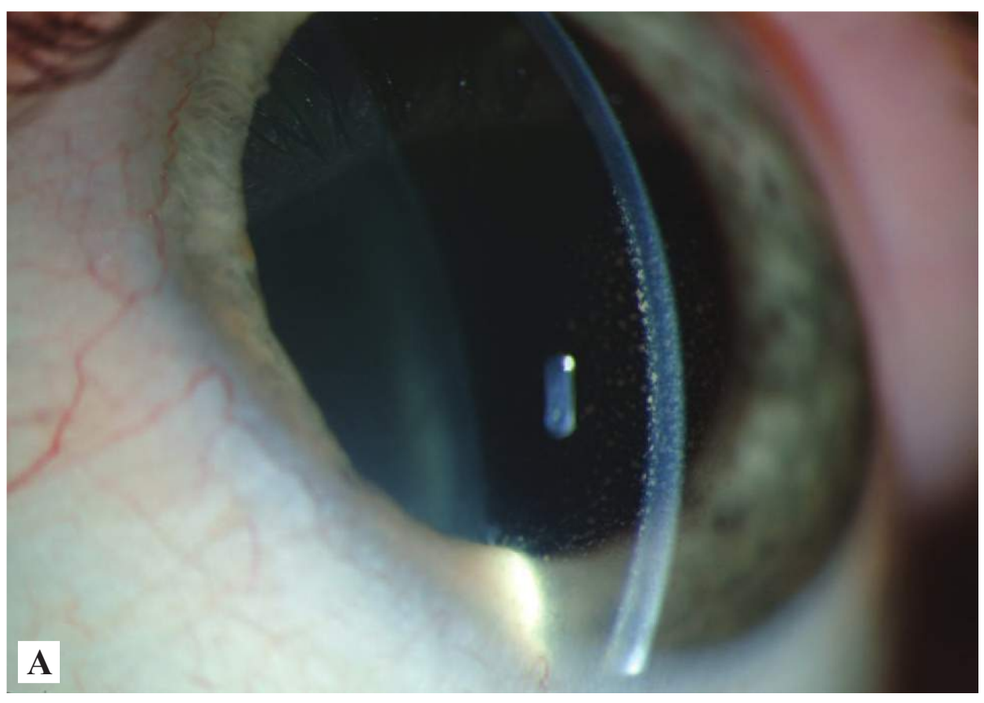


FIGURE 2.38 ■ **Keratic Precipitates.** Deposits of cells on the endothelial layer of the cornea are seen in these photographs with a slit-lamp beam (**A**), and under diffuse light (**B**). (Used with permission from Brice Critser, CRA, The University of Iowa and EyeRounds.org.)

SYMPATHETIC OPHTHALMIA

Clinical Summary

This rare disorder presents as an insidious or acute anterior uveitis of both eyes (exciting and sympathizing eye) after recent or remote trauma to the exciting eye. The initial injury is usually from prior penetrating eye trauma with delayed wound closure or, less commonly, from prior eye surgery.

The disease is thought to be an autoimmune inflammatory response against choroidal melanocytes released due to trauma. It may develop rapidly or insidiously, presenting from days to years after the inciting traumatic event. Most cases develop within 1 year.

Earliest symptoms include eye floaters and loss of accommodation. The disease may progress to severe uveitis with redness, pain, and photophobia in the sympathizing, noninjured eye.

Classically, there is a granulomatous anterior chamber reaction with mutton-fat keratic precipitates seen on the corneal endothelium.

Management and Disposition

Diagnosis is suspected clinically. Consider other causes of granulomatous uveitis such as sarcoidosis, tuberculosis, and syphilis. Consult ophthalmology for management, which may include topical steroids, evisceration, or enucleation of the eye with prior trauma.

Pearls

1. Consider sympathetic ophthalmia with unexplained uveitis and a history of penetrating eye trauma.
2. Solicit a history of eye trauma or prior eye surgery when evaluating eye pain, blurred vision, and redness.
3. Early intervention can be sight saving.



FIGURE 2.39 ■ **Sympathetic Ophthalmia.** This patient with prior ocular trauma (untreated globe rupture) OS presented with pain and redness OD, thought to be due to sympathetic ophthalmia. OS enucleation and treatment with steroids resolved condition. (Photo contributor: R. Jason Thurman, MD.)

ANISOCORIA

Clinical Summary

Anisocoria is a disparity of pupil size. To determine the abnormal pupil, compare pupil sizes in light and dark. It is accentuated in the paretic muscle. If the iris sphincter (or its innervation) is involved, the anisocoria will be increased in bright light. If the iris dilator muscle (or its innervation) is affected, it will be more pronounced in darkness. Up to 20% of normal individuals have physiologic anisocoria of 1 to 2 mm.

Other causes of anisocoria with an abnormally large pupil include mydriatic drops, contamination from a scopolamine patch, an Adie pupil, and ocular trauma with iris sphincter damage. A dilated pupil due to anticholinergic agents (eg, atropine) does not react to light, although a dilated pupil due to sympathomimetics still has some response.

An abnormally small pupil may be secondary to Horner syndrome, chronic Adie pupil (8 weeks or more after the event), iritis, and eye drops (pilocarpine). Miosis secondary to pupillary sphincter muscle spasm may be transiently observed after ocular trauma, followed by mydriasis.

Management and Disposition

Evaluation is dependent on clinical presentation. A patient with the acute onset of third-nerve palsy with associated headache or trauma should be evaluated as a neurosurgical emergency.

Pilocarpine may be helpful to differentiate the etiology of pupil dilation. With low concentrations (0.125%), an Adie pupil will constrict more than the unaffected eye secondary to denervation supersensitivity. With higher concentrations (1%), a pupil that fails to constrict is most likely secondary to topical anticholinergic mydriatics (scopolamine, atropine, or cyclopentolate). Unlike the mydriasis seen with intracranial pathology, pharmacologic mydriasis is not associated with pain, ptosis, or diplopia. Other pathology within the iris sphincter muscle that prevents constriction to 1% pilocarpine includes iris muscle trauma and synechiae.

Pearls

1. A patient with a dilated pupil, ptosis, and abnormal extraocular movements should be evaluated emergently for an aneurysm or expanding supratentorial mass with tentorial herniation.
2. With physiologic anisocoria, the pupil size disparity is the same in light and dark, and there are no other ocular findings.
3. A driver's license or ID badge may be helpful to document a preexisting anisocoria.
4. Some brands of eye make-up contain belladonna alkaloids which can cause mydriasis.
5. An Adie pupil initially is dilated, but may become miotic over time. It is a benign condition that affects young adults, women more often than men, and is associated with decreased reflexes.



FIGURE 2.40 ■ **Posttraumatic Anisocoria.** Marked chronic anisocoria secondary to prior trauma as a child. (Photo contributor: R. Jason Thurman, MD.)

HERPES ZOSTER OPHTHALMICUS

Clinical Summary

Reactivation of endogenous latent varicella-zoster virus within the trigeminal ganglion with neuronal spread through the ophthalmic branch results in crops of grouped vesicles on the forehead and periocularly.

Patients typically present with periocular rash and an injected eye, along with a watery discharge. The most common corneal lesion is punctate epithelial keratitis, in which the cornea has a ground-glass appearance because of stromal edema. Pseudodendrites, also very common, form from mucous deposition, are usually peripheral, and stain moderate to poorly with fluorescein. These may be differentiated from the dendrites of herpes simplex in that the pseudodendrites lack the rounded terminal bulbs at the end of the branches, and are broader and more plaquelike. Anterior stromal infiltrates may be seen in the second or third week after the acute infection. Follicles (hyperplastic lymphoid tissue that appears as gray or white lobular elevations, particularly in the inferior cul-de-sac) and regional adenopathy may or may not be present. Iritis is seen in approximately 40% of patients.

Management and Disposition

Treat patients with epithelial defects with topical broad-spectrum antibiotics to prevent secondary infection. Initiate oral antivirals within 72 hours of onset, and treat for 7 to 10 days. Use cycloplegics if an iritis is present. Artificial tears or ointment may be helpful and narcotic analgesics may be required. Ophthalmologic consultation is indicated.

Pearls

1. Ocular complications may follow the rash by many months to years. These complications have a highly variable presentation that can mimic almost any ophthalmic condition.
2. Recurrence is more common in the immunocompromised host.
3. Perform a careful eye exam with corneal staining. Nearly two-thirds of patients will develop ocular lesions.
4. Corneal hypesthesia and the appearance of dendrites with fluorescein staining are seen in both herpes zoster ophthalmicus and herpes simplex keratitis.
5. Patients with skin lesions on the tip of the nose (Hutchinson sign) are at high risk for ocular involvement. However, the eye may be involved without a nasal lesion.



FIGURE 2.41 ■ **Herpes Zoster Ophthalmicus.** A vesicular rash in the distribution of the ophthalmic division (V1) of the trigeminal nerve is seen. The presence of the lesion near the tip of the nose (Hutchinson sign) increases the risk of ocular involvement. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 2.42 ■ **Herpes Zoster Ophthalmicus.** A large circular dendrite is seen in this patient with ocular involvement from HZV. (Photo contributor: Alexandra Bingnear, RN.)

OCULAR HERPES SIMPLEX

Clinical Summary

Ocular herpetic disease may be neonatal, primary, or recurrent. Neonatal disease occurs secondary to passage through an infected birth canal, and is usually HSV type 2. Primary ocular herpes may present as a blepharitis (grouped eyelid vesicles on an erythematous base), conjunctivitis, or keratoconjunctivitis. Patients with keratoconjunctivitis commonly note pain, irritation, foreign body sensation, redness, photophobia, tearing, and occasionally decreased visual acuity. Follicles and preauricular adenopathy may be present. Initially, the keratitis is diffuse and punctate, but after 24 hours, fluorescein demonstrates either serpiginous ulcers or multiple diffuse epithelial defects. True dendritic ulcers are rarely seen in primary disease.

Most ocular herpetic infections are manifestations of recurrent disease rather than a primary ocular infection. These may be triggered by ultraviolet laser treatment, topical ocular medications (β-blockers, prostaglandins), and immunosuppression (especially ophthalmic topical glucocorticoids). Recurrent disease most commonly presents as keratoconjunctivitis with a watery discharge, conjunctival injection, irritation, blurred vision, and preauricular lymph node involvement. Corneal involvement initially is punctate, but evolves into a dendritic keratitis. The linear branches classically end in bead-like extensions called terminal bulbs. Fluorescein dye demonstrates primarily the corneal defect; the terminal bulbs are best seen with rose stain. In addition to the dendritic pattern, fluorescein stain may instead take on a geographic or ameboid shape, secondary to widening of the dendrite. Most patients (80%) with herpes simplex keratitis have decreased or absent corneal sensation in the area of the dendrite or geographic ulceration. Deeper corneal stromal inflammation may also occur (disciform keratitis). Recurrent disease can also present with iritis or with blepharitis, with vesicles grouped in focal clusters.



FIGURE 2.43 ■ **Ocular Herpes Simplex.** This 18-year-old has a history of ocular herpes simplex since childhood. Grouped vesicles on an erythematous base with periorbital erythema are seen. Honey-colored crusts suggest secondary impetigo. (Photo contributor: Lawrence B. Stack, MD.)

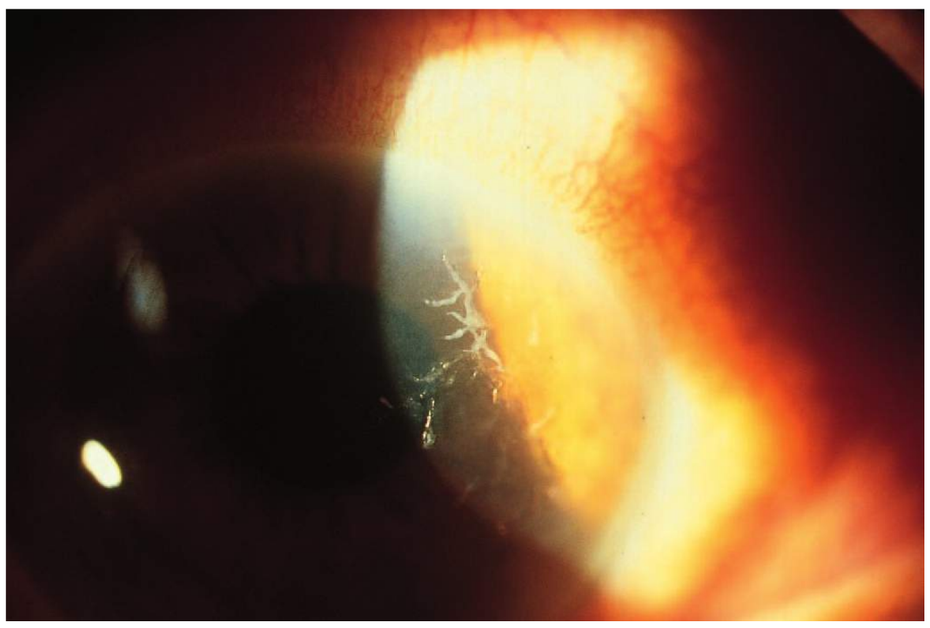


FIGURE 2.44 ■ **Herpes Simplex Keratitis.** A slit-lamp view of unstained dendritic lesions. (Photo contributor: Lawrence B. Stack, MD.)

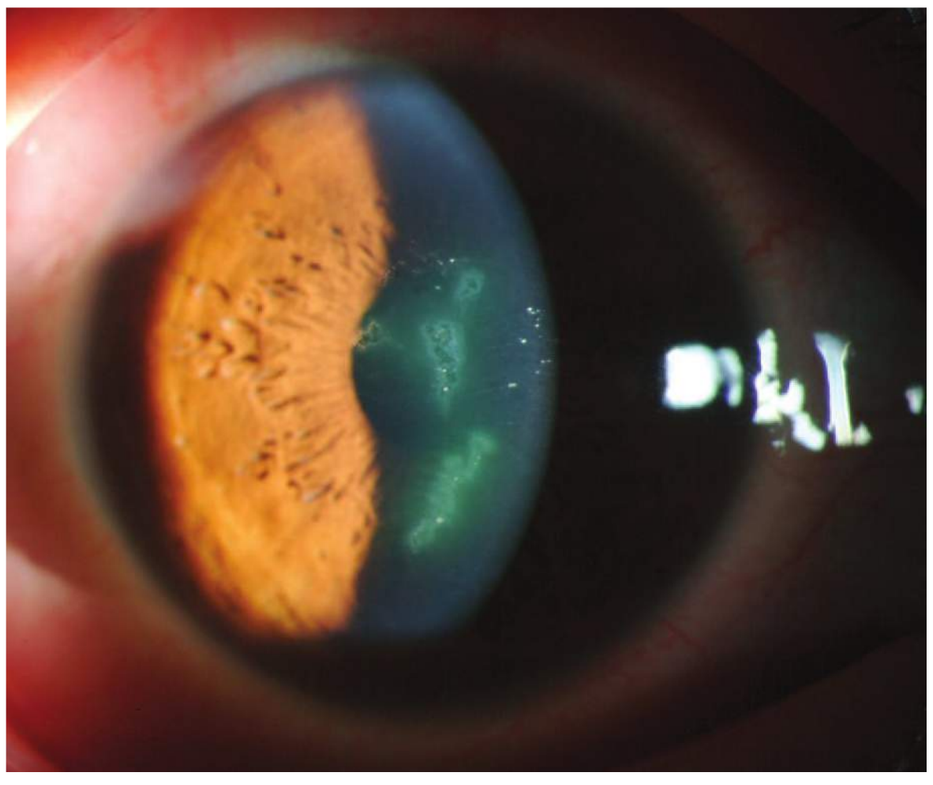


FIGURE 2.45 ■ **Herpes Simplex Keratitis.** A slit-lamp view of dendritic lesions under white light. (Used with permission from The University of Iowa and EyeRounds.org.)

Management and Disposition

There is a high association in neonatal ocular herpes infections between ocular HSV disease and serious systemic or neurologic infection, and an emergency pediatric or infectious disease consult is necessary. IV acyclovir is indicated, and the ocular disease itself may be treated with topical antivirals (idoxuridine, vidarabine, trifluorothymidine). Other sexually transmitted diseases such as chlamydia or gonorrhea should be explored.

Treatment of those with primary ocular herpes (beyond the neonatal period) presenting as blepharitis or periocular dermatitis consists of good local hygiene and a prophylactic topical antiviral such as trifluorothymidine or idoxuridine ointment. Patients with corneal involvement should additionally receive topical antibiotics to prevent secondary bacterial infection.

In those with recurrent disease, topical antivirals (trifluridine, ganciclovir, acyclovir, vidarabine) are effective, as is oral acyclovir. If the cornea is involved, trifluorothymidine and topical antibiotics are added. Ophthalmology consultation is required in patients with ocular HSV disease. Episodes of recurrent stromal disease may be limited by the long-term use of low-dose oral antivirals.

Pearls

1. Most ocular HSV infections are manifestations of recurrent disease.
2. Corneal disease is the most prevalent form of ocular HSV disease.
3. Oral antivirals are now frequently used for keratitis because of their convenience, although topical optical antivirals are equally effective.
4. HSV dendrites, when stained with fluorescein, appear as branching lesions with terminal bulbs.
5. Corneal hypesthesia may be easily overlooked in the initial evaluation of a red eye. If a topical anesthetic has been given, a reexamination 1 hour later is helpful for evaluating hypesthesia.

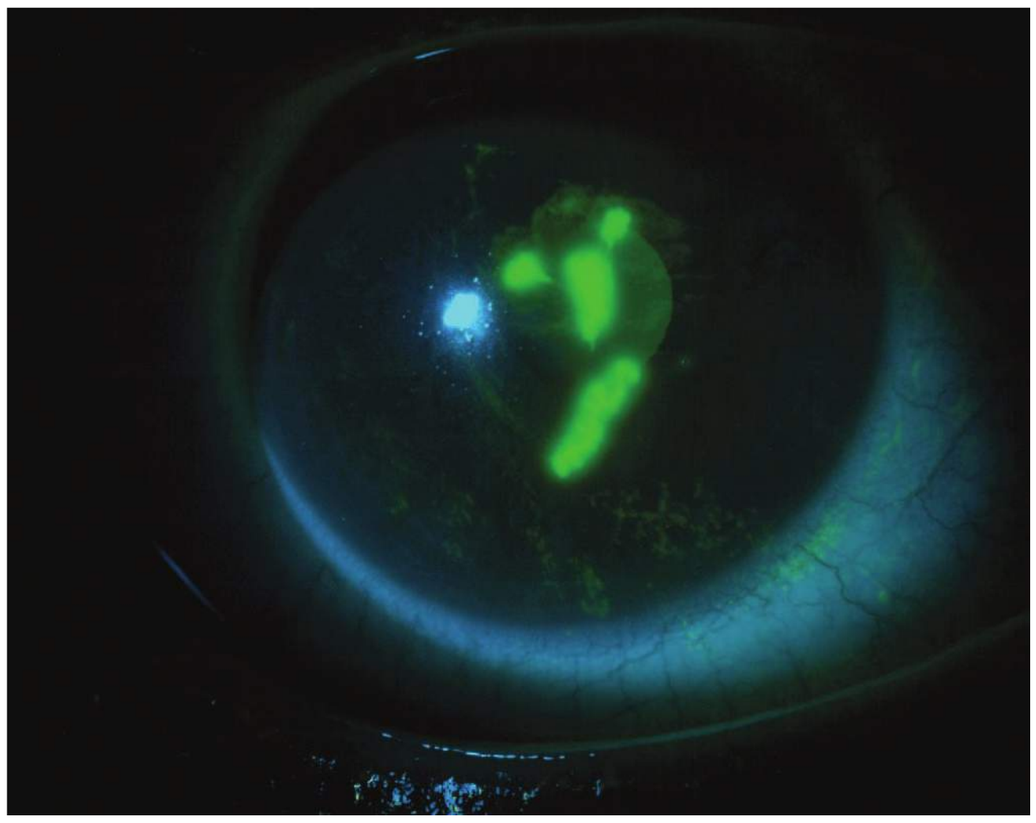


FIGURE 2.46 ■ **Herpes Simplex Keratitis.** Epithelial dendrites seen in Fig. 2.45 after fluorescein staining. (Used with permission from The University of Iowa and EyeRounds.org.)

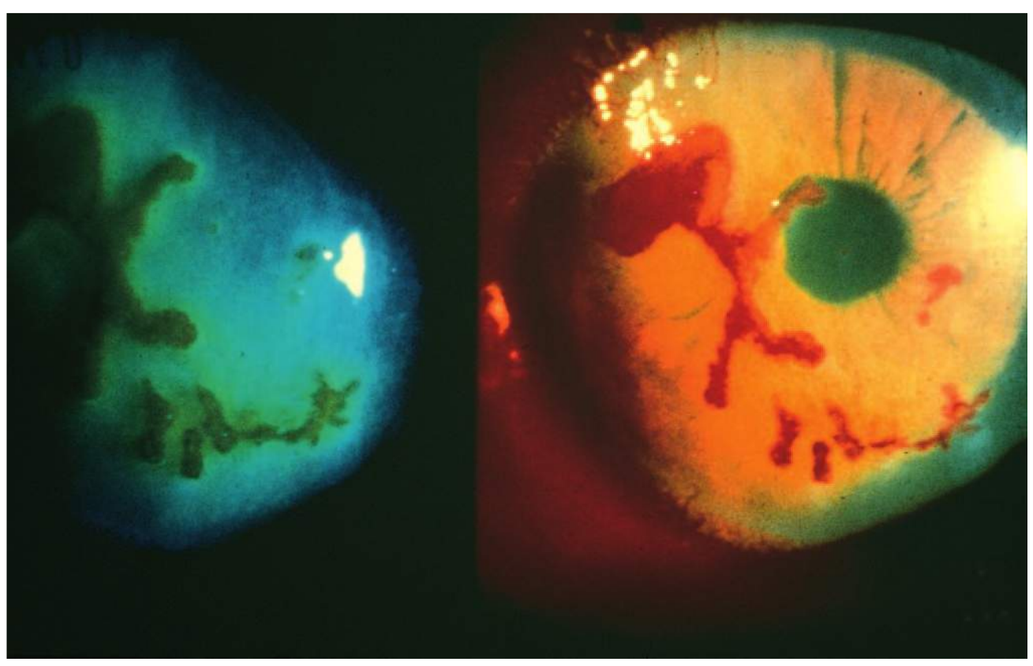


FIGURE 2.47 ■ **Herpes Simplex Keratitis**. Fluorescein (left) and rose bengal (right) stains demonstrate characteristic dendritic patterns. Whereas fluorescein staining is used to detect epithelial defects, rose bengal staining additionally demonstrates degenerating or dead epithelial cells and is particularly good for demonstrating the club-shaped terminal bulbs at the end of each branch. (Used with permission from the American Academy of Ophthalmology. External Disease and Cornea: A Multimedia Collection. San Francisco; 1994.)

Clinical Summary

A corneal ulcer is an inflammatory and ulcerative keratitis. Common infectious etiologies include bacteria (Staphylococcus, Streptococcus, Pseudomonas) and viruses (herpes simplex, adenovirus). Bacterial corneal ulcers are commonly associated with extended-wear contact lenses. Rare causes of corneal ulcers include fungal infections and Acanthamoeba, a ubiquitous protozoan associated with contaminated contact lens solutions. Fungal infections may also arise from trauma involving vegetable matter such as a tree branch. Acanthamoeba infections may also occur from swimming in lakes, especially while wearing contact lenses.

Patients present with pain, photophobia, decreased vision, discharge, and a foreign body sensation. Ocular findings include a corneal infiltrate, typically a round white spot, with conjunctival hyperemia, meiosis, and chemosis. Slit-lamp biomicroscopy may demonstrate an epithelial defect with fluorescein uptake. Anterior chamber findings can include cells and flare, keratic precipitates, and a hypopyon.

Management and Disposition

Corneal ulcers are an ophthalmologic emergency requiring emergent ophthalmology consultation. Stains and cultures should be obtained as expeditiously as possible. Intensive topical treatment using fortified antibiotics is the most effective treatment route, initially given every 30 to 60 minutes. For mild cases, a single fluoroquinolone agent may suffice. For more severe cases, dual therapy using a cephalosporin or vancomycin combined with an aminoglycoside is recommended. Clinical improvement is usually noted after 2 to 3 days. Systemic antibiotics are used in cases where the sclera is involved (Pseudomonas) or if there is a high risk of concurrent systemic disease (Neisseria, Haemophilus). Cycloplegics are recommended if there is an accompanying iritis. Steroids and eye patching are contraindicated. A contact lens wearer must discontinue contact lens wear.

Pearls

1. A corneal ulcer is an ophthalmologic emergency.
2. Extended-wear contact lens use is a risk factor for corneal ulcer.
3. Pseudomonas aeruginosa, associated with thick yellow-green or blue-green mucopurulent tenacious exudate, is capable of destroying the cornea within 6 to 12 hours.
4. Acanthamoeba should be suspected in contact lens wearers with contaminated lens solutions or who swim wearing their contact lens. Classically, these patients have pain out of proportion to their clinical findings.
5. Infectious ulcers tend to develop centrally, away from the vascular supply and immune system of the limbus.

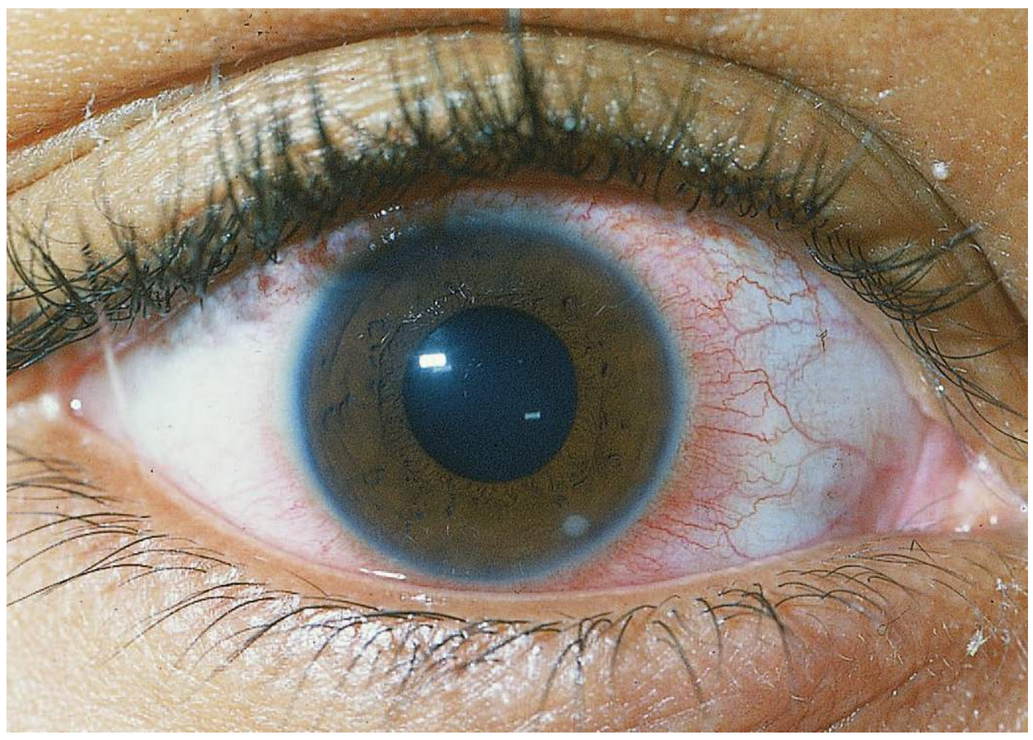


FIGURE 2.48 ■ **Corneal Ulcer.** An elliptical ulcer at 5-o'clock position near the limbus is seen. This location is atypical for a bacterial ulcer. The patient presented with painful red eyes and normal uncorrected vision, but wore cosmetic soft contact lenses. Bilateral corneal ulcers were diagnosed, which cleared after treatment with topical ciprofloxacin. Note that the ciliary flush seen in the nasal portion of the limbus is not to be mistaken for conjunctivitis. (Photo contributor: Kevin J. Knoop, MD, MS.)

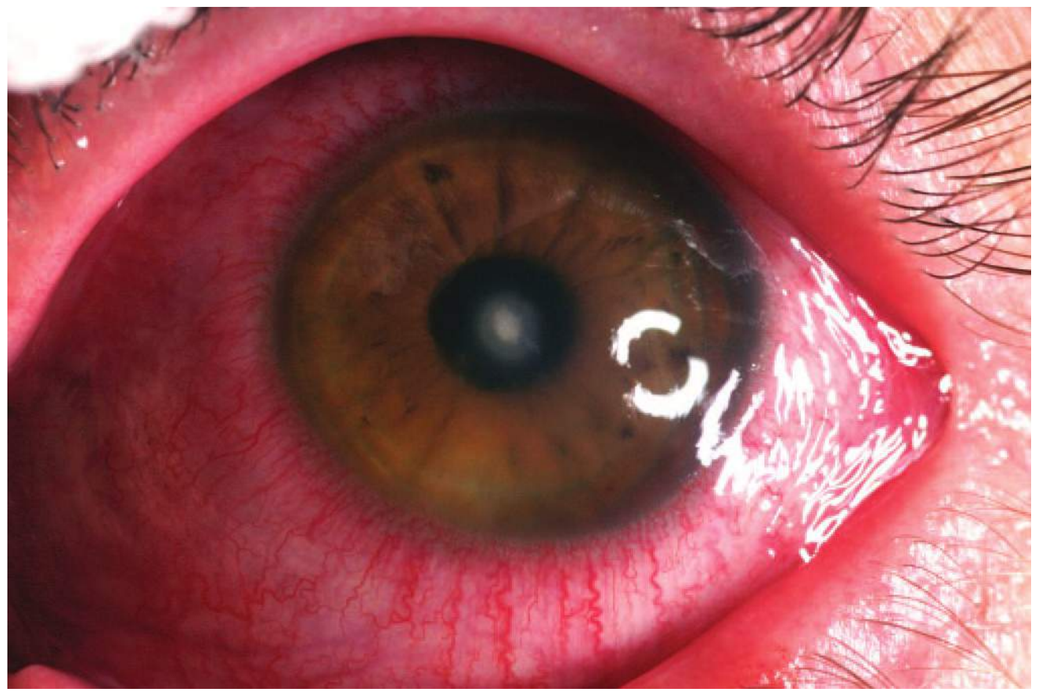


FIGURE 2.49 ■ **Corneal Ulcer.** A faint white circular corneal infiltrate is seen in the central visual axis. (Photo contributor: Kevin J. Knoop, MD, MS.)

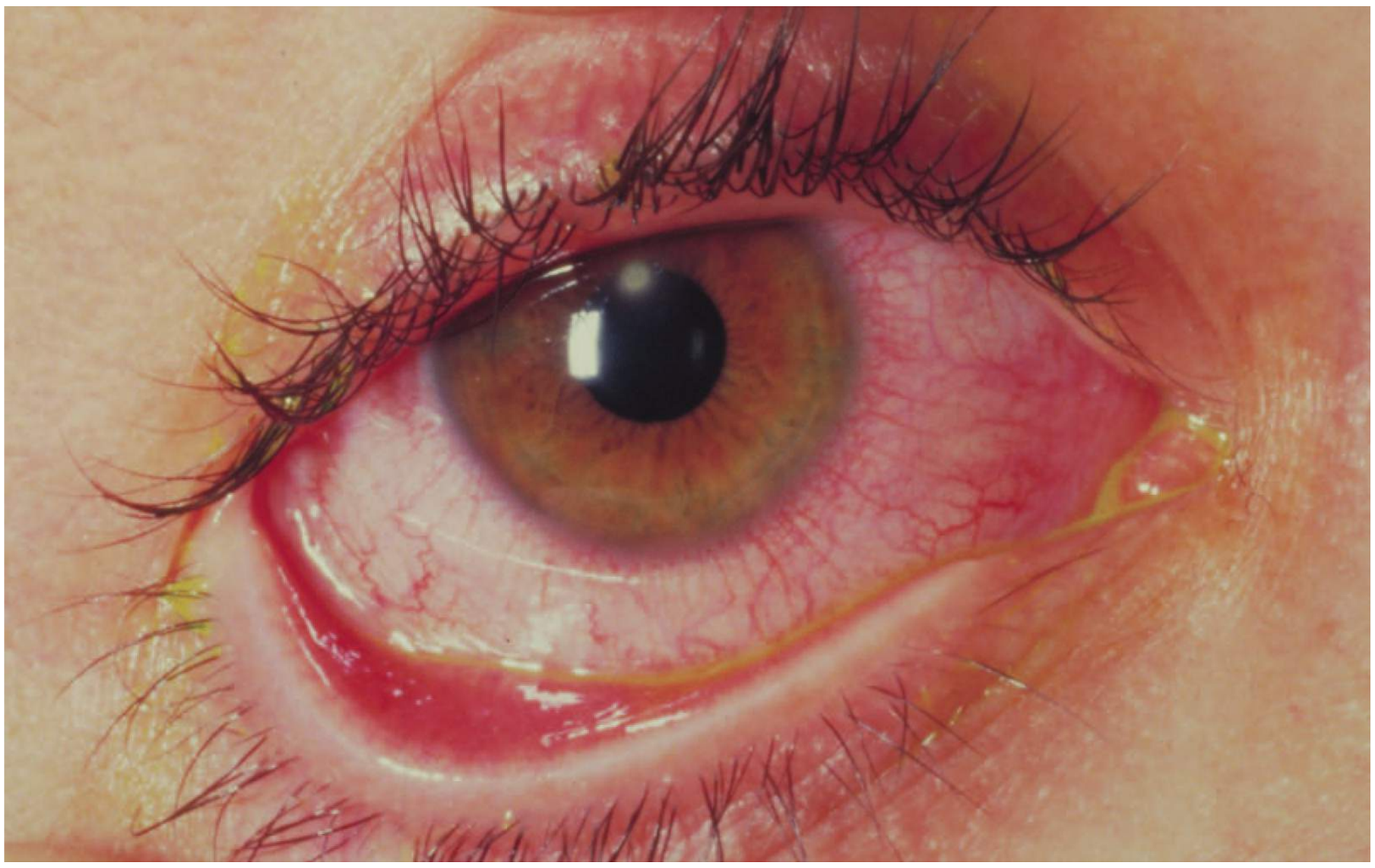


FIGURE 2.50 ■ **Corneal Ulcer**. A small circular corneal infiltrate is seen adjacent to the white flash photography reflection. Diffuse conjunctival hyperemia with a nasal ciliary flush is seen. (Photo contributor: Lawrence B. Stack, MD.)

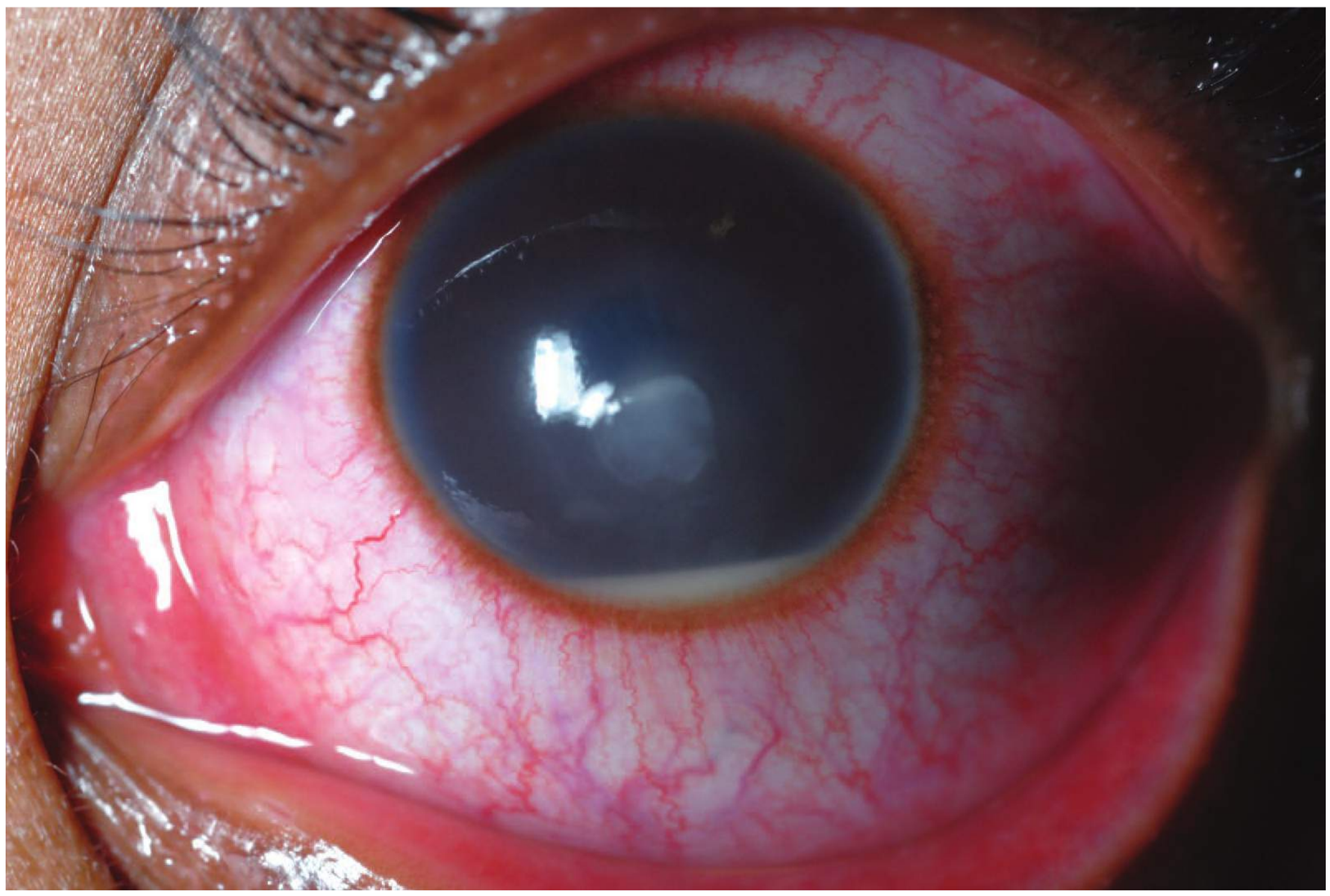


FIGURE 2.51 ■ **Corneal Ulcer with Hypopyon.** A hypopyon has developed from a corneal ulcer seen near the visual axis. The conjunctiva is inflamed and a ciliary flush is present. (Photo contributor: R. Jason Thurman, MD.)

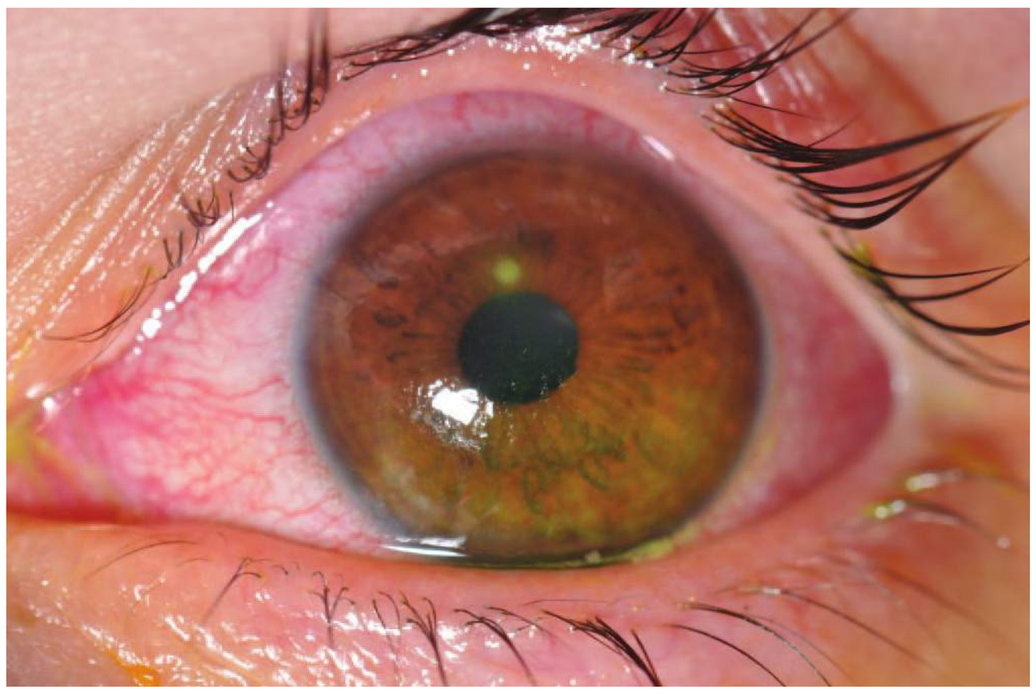


FIGURE 2.52 ■ **Corneal Ulcer.** Diminished visual acuity and pain due to the corneal ulcer just superior to the visual axis contributed to his MVC and airbag deployment, causing a large corneal abrasion of the inferior third of the cornea. (Photo contributor: Lawrence B. Stack, MD.)

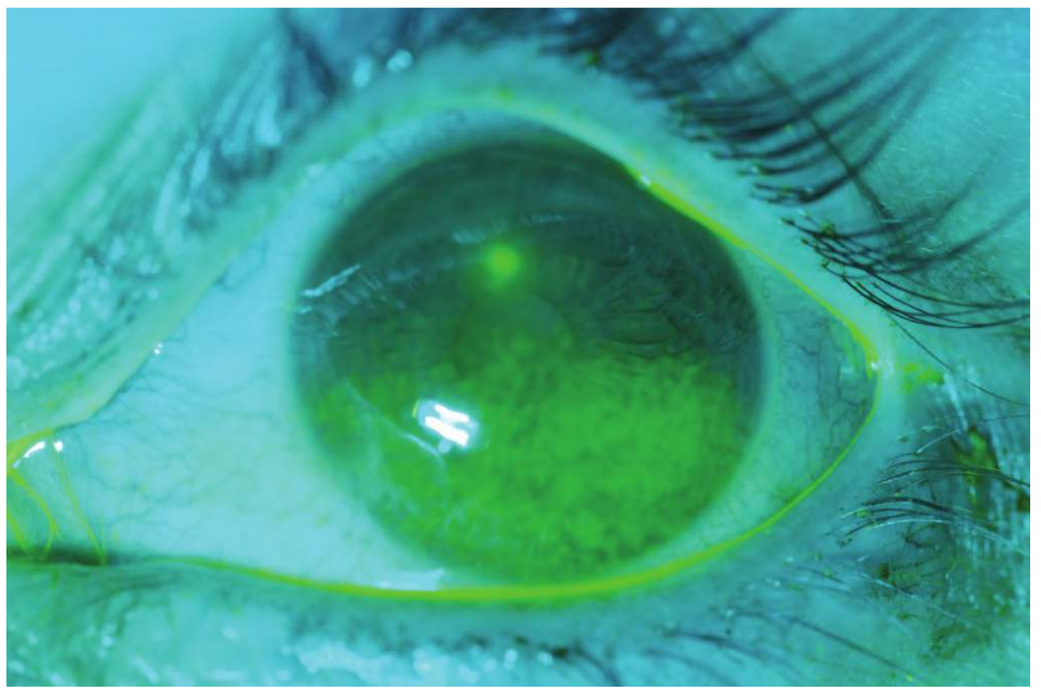


FIGURE 2.53 ■ **Corneal Ulcer.** Fluorescein stain uptake of the corneal ulcer just above the visual axis and the corneal abrasion of the inferior third of the corneal surface due to airbag deployment. (Photo contributor: Lawrence B. Stack, MD.)

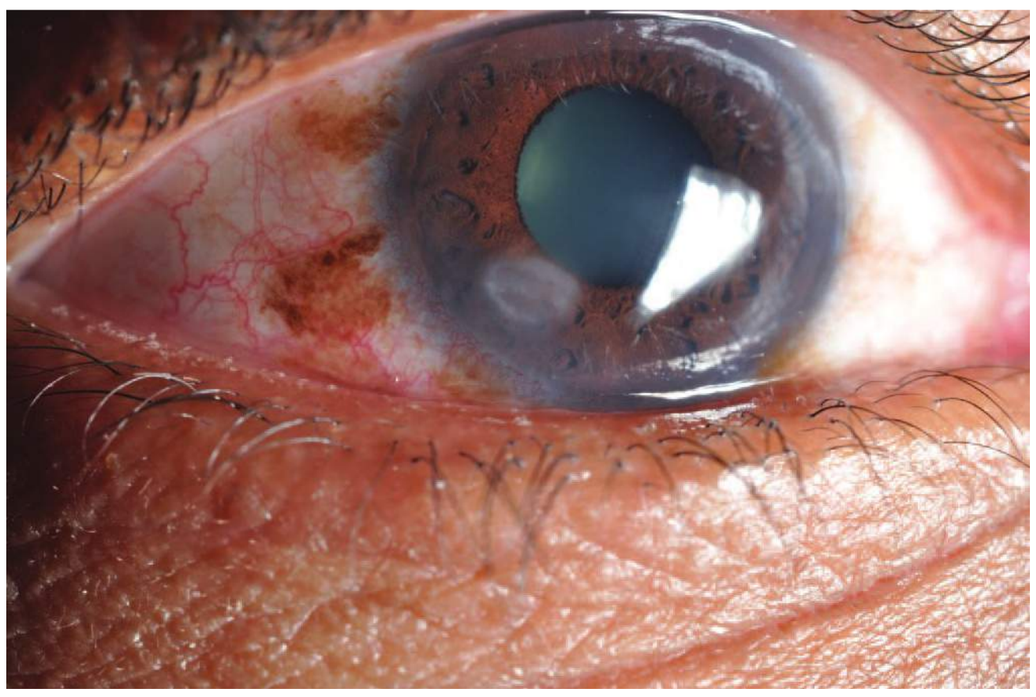


FIGURE 2.54 ■ **Corneal Ulcer**. A large oval opaque corneal ulcer is seen at 8-o'clock position toward the limbus in a contact-lens wearing patient. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 2.55 ■ **Corneal Ulcer with Hypopyon**. A middle-aged female accidentally splashed dishwashing detergent in her eye one week prior to ED visit. A large inferior corneal ulcer, with corneal edema and hypopyon, is seen. (Photo contributor: Eugene Eiland, MD.)

AFFERENT PUPILLARY DEFECT (MARCUS GUNN PUPIL)

Clinical Summary

Pupil size is controlled in the midbrain, from which efferent nerves travel to both pupils resulting in symmetric pupils, even with a unilateral light stimulus. The perception of the light stimulus, however, may be decreased by disease within anterior visual structures such as the retina, optic nerve, chiasm, optic tract, and midbrain pathways. With diminished afferent stimulation, less light is "perceived," and pupil contraction diminishes (ie, dilates) as a result. In this situation, the affected side is said to demonstrate an afferent pupillary defect (APD), and the pupil is called a Marcus Gunn pupil.

A Marcus Gunn pupil is best appreciated by the swinging flashlight test, which discloses differences in afferent stimuli between the two eyes. A flashlight is directed onto one pupil and then the other. The normal response is a prompt constriction. (Generally, this is followed by a slight "release" dilation.) During the brief interval required to move the light from one to the other eye, both pupils begin to dilate. Thus, under normal circumstances, when the light reaches the second eye, reflex constriction again occurs. With a diseased eye or pathway, the diseased side perceives less light and the midbrain therefore sets the pupil size to be larger, and as a result the pupils dilate (or continue to dilate following the "release" dilation).

Although sensitive, the swinging flashlight test is not specific, since the pathology may be anywhere in the visual pathway from the retina to the midbrain. The test cannot distinguish between unilateral afferent disease and asymmetric bilateral disease. Typically, a positive finding indicates optic nerve disease such as ischemic optic neuropathy, optic neuritis (including that seen with multiple sclerosis), retrobulbar optic neuritis, and glaucoma.

Management and Disposition

The finding of a Marcus Gunn pupil is nonspecific. A fundoscopic exam may show occlusion of the central retinal vein or central retinal artery, or a dense hemorrhage in the vitreous. Otherwise, a neurologic evaluation (history, physical examination, and CT scan) is important to assess for treatable conditions. In the absence of gross ocular or neurologic disease, a clinically stable patient may be discharged from the ED with ophthalmology follow-up.

Pearls

1. An APD does not cause anisocoria because any change in light input results in a bilaterally symmetric output from the midbrain.
2. In the swinging flashlight test, both pupils will dilate when the flashlight moves from the normal pupil to the Marcus Gunn pupil, since pupil size is centrally mediated in the midbrain.
3. Bilaterally symmetric disease produces no APD, since the basis for the swinging flashlight test is asymmetry of disease.
4. A dense vitreous hemorrhage is capable of producing a mild defect. Lesser ocular pathology such as branch retinal vein or artery occlusion or a cataract is unlikely to produce a clinically detectable APD.
5. Dim room illumination may be helpful when performing the swinging flashlight test. The patient should focus on an object 15 feet away to avoid the pupil constriction normally seen with accommodation.

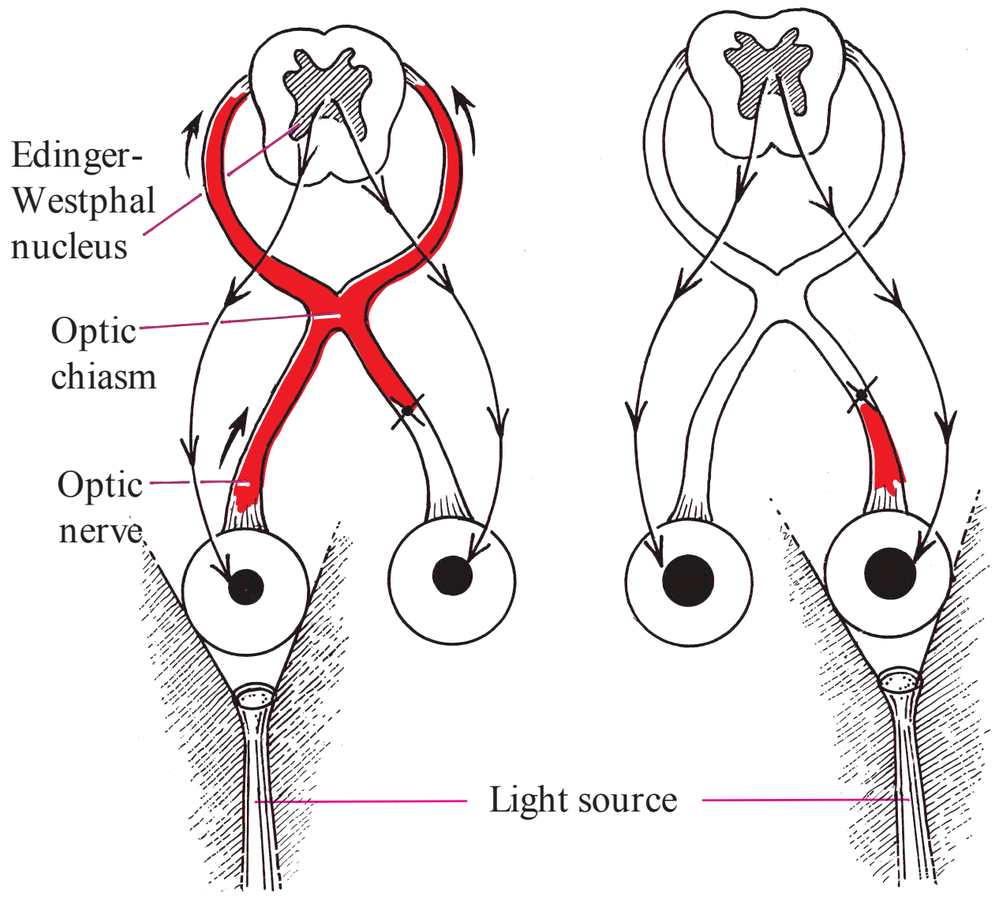


FIGURE 2.56 ■ **Afferent Pupillary Defect.** Schematic representation of an afferent pupillary defect (APD) due to neurologic lesion in the anterior visual pathway.



FIGURE 2.57 ■ **Marcus Gunn Pupil**. These photographs demonstrate an afferent pupillary defect of the left pupil. When the light shines in the affected left eye, the pupils are less constricted than when the light shines in the right eye. Thus, when the light is swung from the right pupil to the left, the pupils appear to dilate. The anisocoria here is subtle. (Photo contributor: Frank Birinyi, MD.)

Clinical Summary

Horner syndrome (miosis, ptosis, and anhidrosis) is secondary to loss of ocular sympathetic innervation. Ptosis is less than 2 mm, the result of paralysis of Müller muscle, innervated by the sympathetic pathway. Anhidrosis is often not apparent to patients or clinicians. A pupillary finding specific in Horner syndrome is dilation lag. Because the dilator muscle is weak, the pupil dilates more slowly than the normal pupil.

This loss of ocular sympathetic innervation can be produced by a lesion anywhere along a three-neuron sympathetic pathway, from the hypothalamus down through the brain stem to the cervical cord, in the apex of the chest, along the carotid sheath, and in the cavernous sinus or orbit. Isolated Horner syndrome presenting with head or neck pain suggests an internal carotid artery dissection.

Management and Disposition

Associated signs and symptoms help direct the ED workup. A patient with cranial nerve abnormalities requires CT or MRI imaging and admission. In the setting of cervical spine trauma, neck immobilization and appropriate imaging studies are instituted. Consider carotid artery dissection in neck pain without trauma.

Pearls

1. Classic signs of a Horner syndrome include meiosis and mild ptosis. Anhidrosis is less readily appreciated, and occurs with first- or second-order lesions only.
2. Miosis and dilation lag are accentuated in a darkened room.
3. A Pancoast tumor usually is caused by bronchogenic carcinoma and may present, as its presenting sign, as a Horner syndrome.
4. Cluster headaches, with autonomic sympathetic system dysfunction, are capable of producing an ipsilateral Horner syndrome.
5. Ipsilateral extraocular paresis, especially a sixth-nerve palsy, without other brainstem signs localizes pathology to within the cavernous sinus.



FIGURE 2.58 ■ **Horner Syndrome.** Unilateral meiosis and ptosis are seen in this patient with Horner syndrome and sarcoma metastatic to the spine. (Photo contributor: Frank Birinyi, MD.)

THIRD-NERVE PALSY

Clinical Summary

The third cranial nerve innervates all the extraocular muscles except the lateral rectus and superior oblique (LR6, SO4). It also controls the levator palpebrae muscle and supplies parasympathetics to the pupillary constrictor and ciliary muscles. Therefore, the clinical examination is notable for a dilated and unreactive pupil, limited extraocular movements, and ptosis. The eye rests in a position of abduction because of unopposed action of the lateral rectus.

Third-nerve dysfunction can result from lesions anywhere along its path from the oculomotor nucleus in the midbrain, within the subarachnoid space, traversing the cavernous sinus, and terminating in the extraocular muscles within the orbit. Contralateral hemiparesis suggests brainstem involvement (Weber syndrome). Pathology within the subarachnoid space causing a third-nerve palsy includes compression of the nerve by a posterior communicating artery aneurysm, uncal herniation, or compressive neoplasm.

Pathology within the cavernous sinus causing a third-nerve palsy includes carotid artery aneurysm, cavernous sinus thrombosis, and carotid-cavernous fistula. Third-nerve lesions here are often accompanied by lesions involving the fourth, fifth (ophthalmic branch), and sixth cranial nerves. Orbital pathology such as inflammation, trauma, or neoplasm should be suspected when orbital findings such as chemosis, conjunctival injection, or proptosis are seen.

Isolated third-nerve palsies ("pupil-sparing") are usually caused by microvascular ischemia. Diabetes, hypertension, and advanced age are risk factors. These typically present with intact pupillary function probably because of the superficial location of the pupillomotor fibers.

Management and Disposition

In patients with brainstem involvement, CT or MRI is indicated. Associated fever, headache, and altered consciousness should prompt CT scanning and subsequent spinal tap. MRI with gadolinium is preferred for evaluation of the cavernous sinus, and CT scanning is recommended for suspected orbital pathology.

Of particular concern is the sudden onset of third-nerve palsy accompanied by a "thunderclap" headache, stiff neck, and depressed level of consciousness. Even those with "pupil-sparing" should be evaluated as a neurosurgical emergency with emergent neuroimaging to evaluate for aneurysm and uncal herniation. If subarachnoid hemorrhage is not found and suspicion of aneurysmal leak remains high, a lumbar puncture should be considered.

In the setting of head trauma and oculomotor palsy, the workup should proceed expeditiously, with measures to reduce intracranial pressure.

Pearls

1. Patients with the abrupt onset of a "thunderclap" headache and third-nerve palsy require immediate evaluation for an aneurysm. The posterior communicating artery is a common site.
2. In patients over 50 with third-nerve palsies whose pupil is unaffected ("pupil-sparing"), the etiology is usually hypertensive or diabetic vascular disease.
3. Mild or moderate pain is common in ischemic lesions.
4. Third-nerve palsy is unlikely to cause isolated mydriasis. Other etiologies such as tonic pupil, iris sphincter damage, and pharmacologic mydriasis are more likely.
5. Eighty percent of carotid-cavernous fistulas result from trauma, and may present weeks after minor trauma. Findings include an ocular bruit, pain, pulsatile exophthalmos, and chemosis.

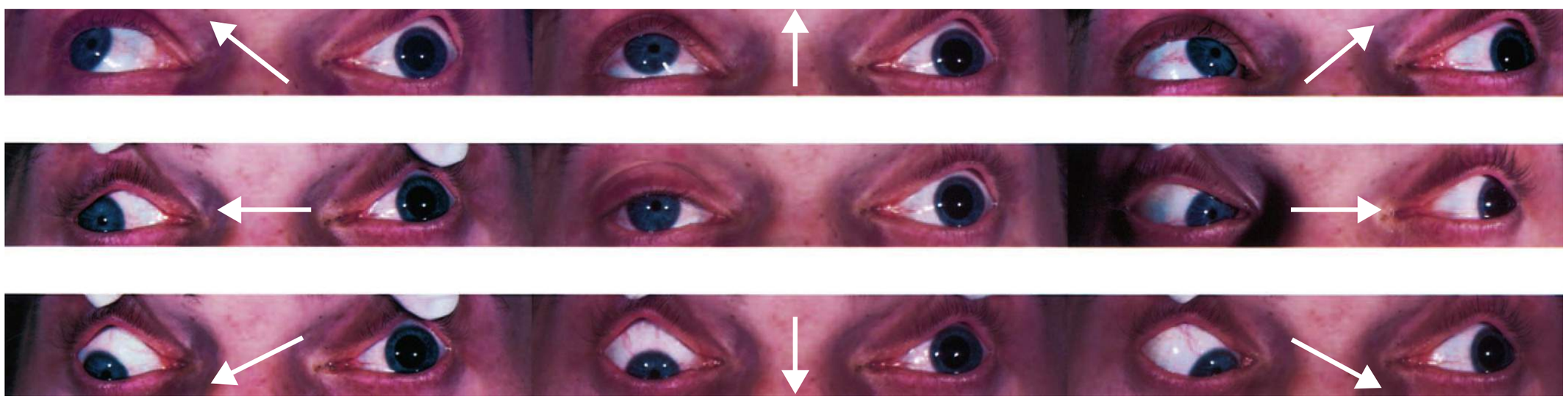


FIGURE 2.59 ■ **Third-Nerve Palsy**. This composite shows the defects of a left third cranial nerve palsy. Conjugate eye movement is present only when the affected eye gazes laterally to the affected side (intact lateral rectus). When gaze is directly ahead, exotropia is seen secondary to the unopposed lateral rectus muscle of the affected side. (Photo contributor: Frank Birinyi, MD.)



FIGURE 2.60 ■ **Isolated Third-Nerve Palsy**. This 60-year-old woman with diabetes mellitus presents with an isolated third-nerve palsy on the right. She is attempting to look leftward. Her symptoms began as an isolated pain above her right orbit 10 days prior at which time her double vision with leftward gaze began. (Photo contributor: Lawrence B. Stack, MD.)

SIXTH-NERVE PALSY

Clinical Summary

The abducens nerve innervates the lateral rectus muscle and is the most common single muscle palsy, causing loss of abduction and resultant horizontal diplopia, worse in ipsilateral gaze. Associated findings are dependent on the location of the lesion. Within the pons, involvement of the corticospinal tract results in contralateral hemiparesis. The abducens has the longest intracranial course of any nerve, and therefore is vulnerable to stretching or compression secondary to elevated intracranial pressure, trauma, neurosurgical manipulation, and cervical traction. Also, any meningeal process (infectious, inflammatory, or neoplastic) can affect this portion of the sixth nerve. Aneurysmal compression is uncommon.

Prior to entering the cavernous sinus, the nerve crosses the petrous portion of the temporal bone. Trauma with temporal bone fracture can result in a combination of sixth- and seventh-nerve palsies. Cavernous sinus pathology is suggested by the involvement of the internal carotid artery, venous drainage of the eye and orbit, trochlear and oculomotor nerves, the first division of the trigeminal nerve, and the ocular sympathetics. Microvascular changes secondary to diabetes, hypertension, and giant cell arteritis can compromise function.

Management and Disposition

Associated signs and symptoms guide the ED workup. CT or MRI is indicated if brain stem or cavernous sinus involvement is suspected. Pathology localizing to the subarachnoid space should prompt consideration for CT scanning and subsequent spinal tap. In the elderly, an isolated sixth-nerve palsy is likely ischemic, transient, and not indicative of underlying neurologic disease. In these cases, a glucose and erythrocyte sedimentation rate is appropriate; these patients can be followed as outpatients provided close follow-up is arranged.

There is no treatment for the palsy itself except for patching the affected eye if diplopia is bothersome.

Pearls

1. An isolated sixth-nerve palsy is commonly due to microvascular disease, not an aneurysm.
2. Basilar skull fractures of the temporal bone are capable of producing a sixth-nerve palsy.
3. A sixth-nerve palsy associated with a Horner is usually localized to the cavernous sinus, since sympathetic fibers, as they traverse from the internal carotid artery to the oculomotor nerve, may briefly accompany the abducens nerve.

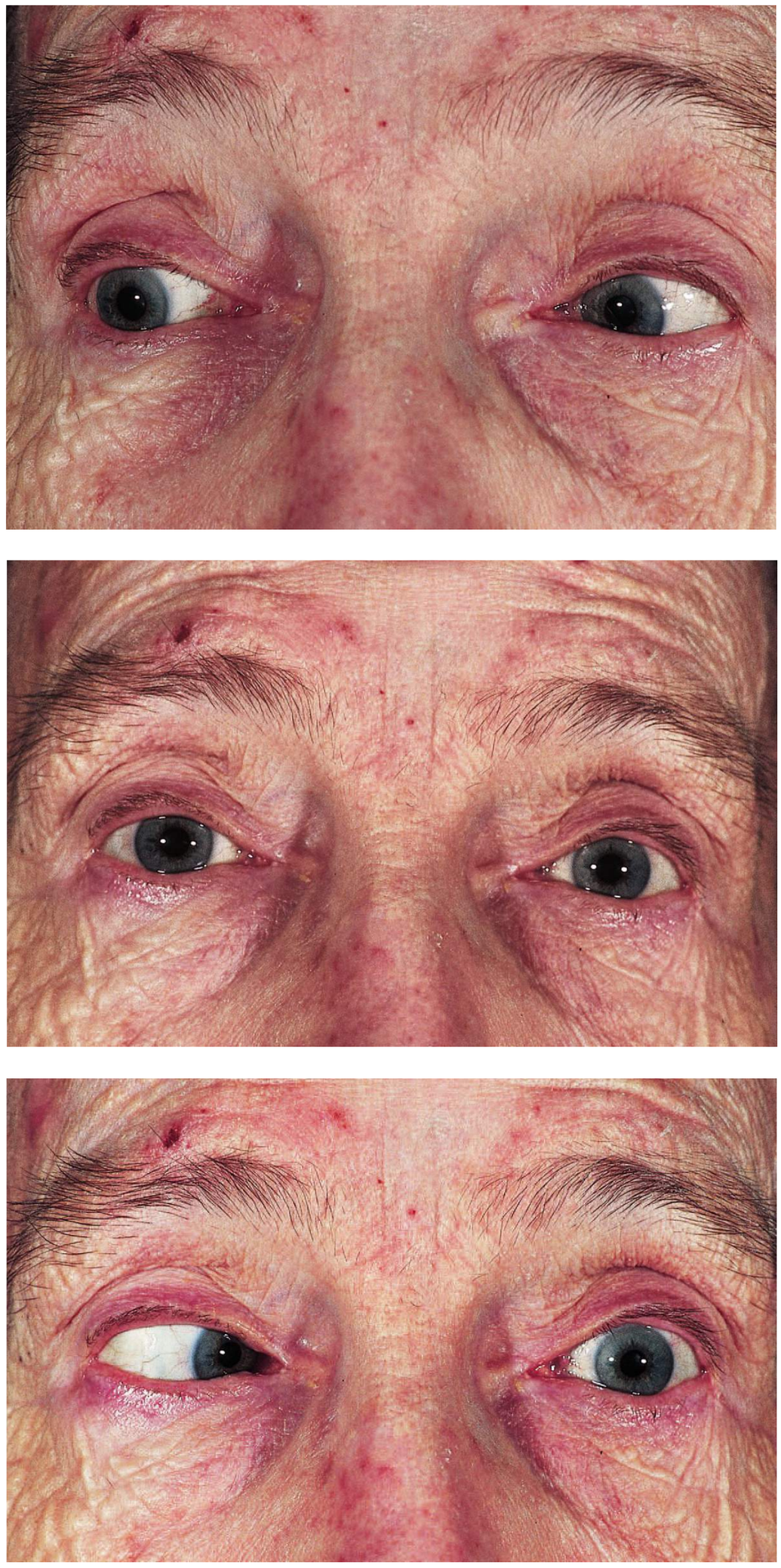


FIGURE 2.61 ■ **Sixth-Nerve Palsy.** Loss of abduction of the left eye is seen in lateral gaze. (Photo contributor: Frank Birinyi, MD.)

Chapter 3

FUNDUSCOPIC FINDINGS

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Beverly C. Forcier
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Papilledema. Blurred disc margins indicate papilledema. A small subhyaloid hemorrhage is also seen inferior to the disc at 5-o'clock position. (Photo contributor: Aaron Sobol, MD.)

NORMAL FUNDUS

Clinical Summary

Disk

The disk is pale pink, approximately 1.5 mm in diameter, with sharp, flat margins. The physiologic cup is located within the disk and usually measures less than six-tenths the disk diameter. The cups should be approximately equal in both eyes.

Vessels

The central retinal artery and central retinal vein travel within the optic nerve, branching near the surface into the inferior and superior branches of arterioles and venules, respectively. Normally the walls of the vessels are not visible; the column of blood within the walls is visualized. The venules are seen as branching, dark red lines. The arterioles are seen as bright red branching lines, approximately two-thirds or three-fourths the diameter of the venules.

Macula

This is an area of the retina located temporal to the disk; it is void of visible vessels. The fovea is an area of depression approximately 1.5 mm in diameter (similar to the optic disk) in the center of the macula. The foveola is a tiny pit located in the center of the fovea. These areas correspond to central vision.

Background

The background fundus is red; there is some variation in the color, depending on the amount of individual pigmentation and the visibility of the choroidal vessels beneath the retina.

Pearls

1. Fundal examination should be an integral part of any eye examination.
2. The cup/disk ratio is slightly larger in the African American population.
3. The normal fundus should be void of any hemorrhages, exudates, or tortuous vasculature.

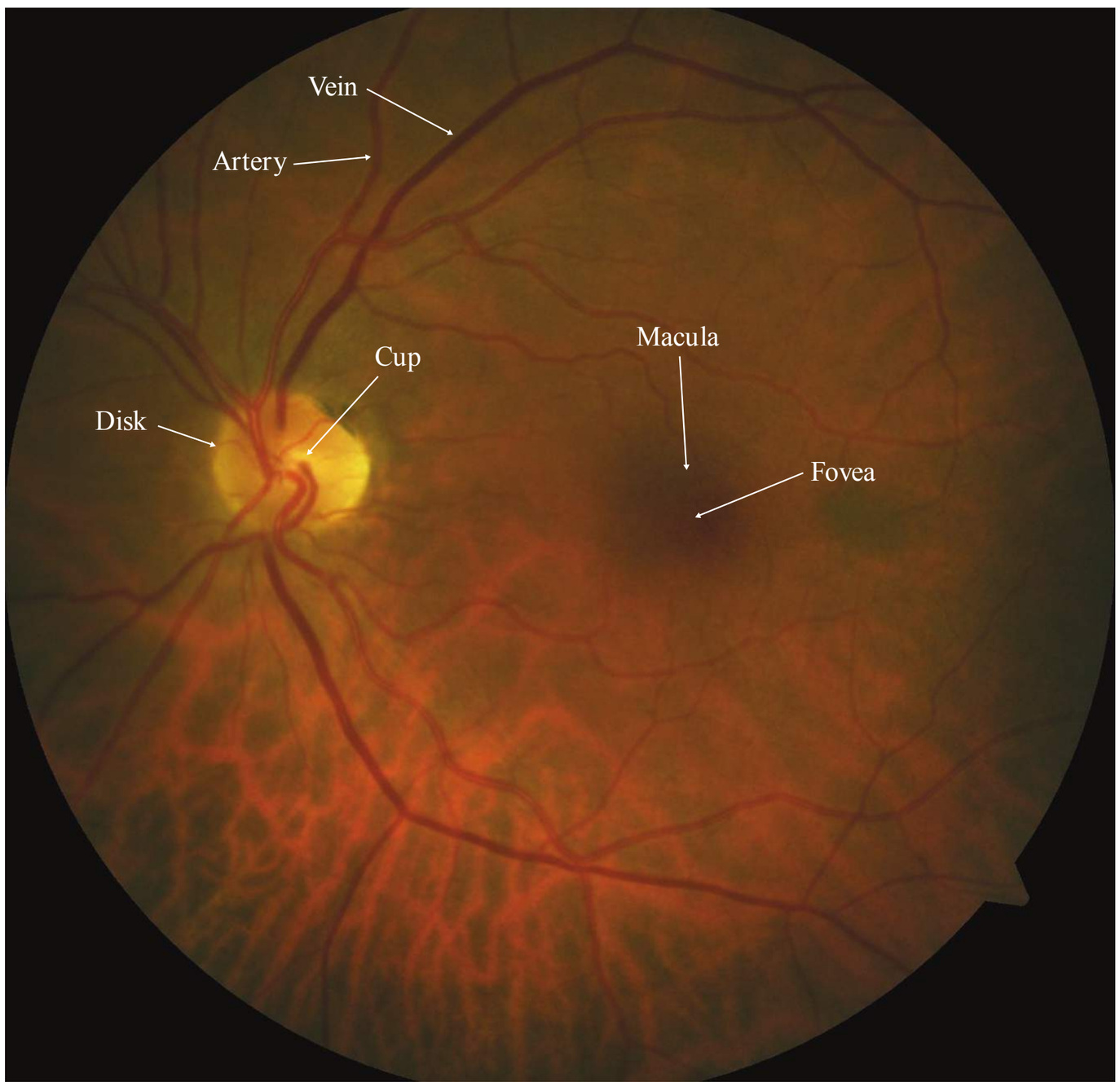


FIGURE 3.1 ■ **Normal Fundus.** The disk has sharp margins and is normal in color, with a small central cup. Arterioles and venules have normal color, sheen, and course. Background is in normal color. The macula is enclosed by arching temporal vessels. The fovea is located by a central pit. (Photo contributor: Jeffrey Goshe, MD.)

AGE-RELATED MACULAR DEGENERATION

Clinical Summary

Age-related macular degeneration increases in incidence with each decade over 50 and is evidenced by accumulation of either hard drusen (small, discrete, round, punctate nodules) or soft drusen (larger, pale yellow or gray, without discrete margins that may be confluent). Most patients with drusen have good vision, although there may be decreased visual acuity and distortion of vision. There may be associated pigmentary changes and atrophy of the retina. Vision may slowly deteriorate if atrophy occurs.

Patients with early or late degenerative changes of the macula are at risk of developing choroidal neovascularization (CNV), which is associated with distortion of vision, blind spots, and decreased visual acuity. Macular appearance may show dirty gray lesions, hemorrhage, retinal elevation, and exudation.

Management and Disposition

Patients with drusen need ophthalmologic evaluation every 6 to 12 months or sooner if visual distortion or decreasing visual acuity develops. If a patient complains of deterioration of visual acuity or image distortion, prompt ophthalmic evaluation is warranted.

Pearls

1. Age-related macular degeneration is the leading cause of blindness in the United States in patients above 65 years of age.
2. Patient may have normal peripheral vision.
3. Untreated CNV can lead to visual loss within a few days.
4. Patients frequently complain of distortion with CNV.



FIGURE 3.3 ■ **Age-Related Macular Degeneration, Drusen.** Drusen are clustered in the center of the macula. (Photo contributor: Richard E. Wyszynski, MD.)



FIGURE 3.2 ■ **Age-Related Macular Degeneration, Drusen**. Typical macular drusen and retinal pigment epithelial (RPE) atrophy (scalloped pigment loss) in age-related macular degeneration.



FIGURE 3.4 ■ **Age-Related Macular Degeneration.** Hemorrhage seen beneath the retina in association with subretinal neovascularization. Note the retinal vessels are superficial to the hemorrhage, which lies just beneath the retina. (Photo contributor: Richard E. Wyszynski, MD.)

Clinical Summary

Hard exudates are refractile, yellowish deposits with sharp margins composed of fat-laden macrophages and serum lipids. Occasionally the lipid deposits form a partial or complete ring (called a circinate ring) around the leaking area of pathology. If the lipid leakage is located near the fovea, a spoke or star-type distribution of the hard exudates may be seen.

Cotton wool spots, or soft "exudates," are actually microinfarctions of the retinal nerve fiber layer, and appear white with soft or fuzzy edges.

Inflammatory exudates are secondary to retinal or chorioretinal inflammation.

Hard exudation and cotton wool spots are associated with vascular diseases such as diabetes mellitus, hypertension, and collagen vascular diseases but can be seen with papilledema and other intrinsic ocular conditions. Inflammatory exudates are seen in patients with such diseases as sarcoidosis and toxoplasmosis.

Management and Disposition

Routine referral for ophthalmologic and medical workup is appropriate.

Pearl

1. Hard exudates that are intraretinal may easily be confused with drusen occurring near Bruch membrane, which separates the retina from the choroid.

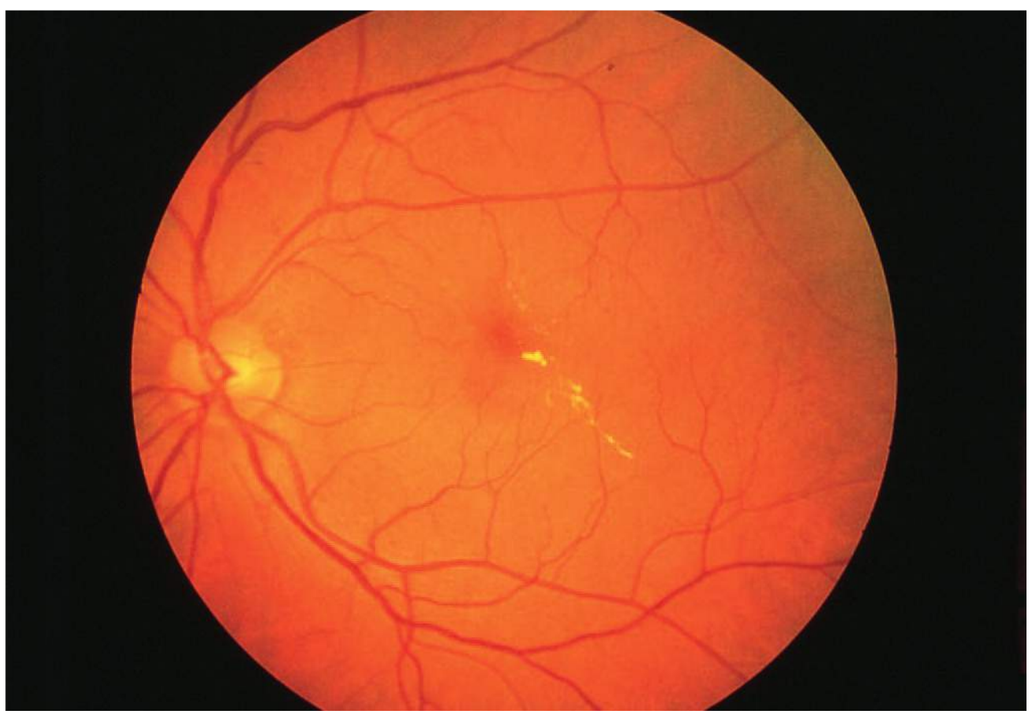


FIGURE 3.5 ■ **Hard Exudates.** Linear collection of yellow lipid deposits with sharp margins in macula. (Photo contributor: Beverly C. Forcier, MD.)

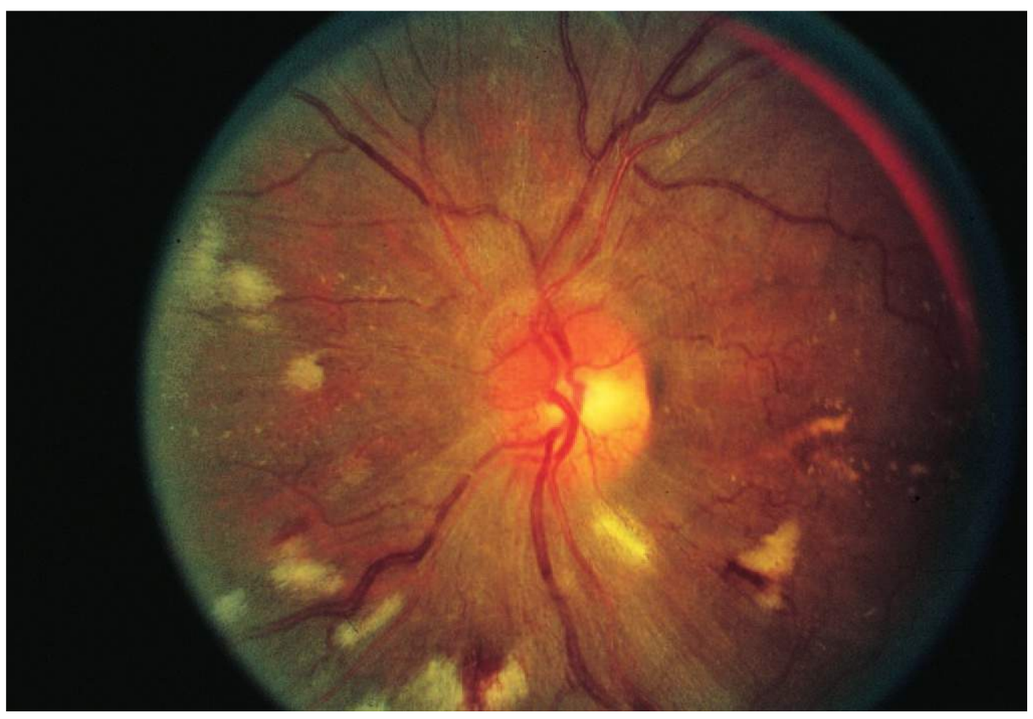


FIGURE 3.6 ■ **Cotton Wool Spots.** White lesions with fuzzy margins, seen here approximately one-fifth to one-fourth disk diameter in size. Orientation of cotton wool spots generally follows the curvilinear arrangement of the nerve fiber layer. Intraretinal hemorrhages and intraretinal vascular abnormalities are also present. (Photo contributor: Richard E. Wyszynski, MD.)

Clinical Summary

Roth spots are retinal hemorrhages with a white or yellow center. They may be seen in patients with a host of diseases such as anemia, leukemia, multiple myeloma, diabetes mellitus, collagen vascular disease, other vascular diseases, intracranial hemorrhage in infants, septic retinitis, and carcinoma. Flame-shaped or splinter hemorrhages or dot-blot hemorrhages may resemble Roth spots.

Management and Disposition

Routine referral for general medical evaluation is appropriate.

Pearls

1. Roth spots are not pathognomonic for any particular disease process and can represent a variety of clinical conditions.
2. These lesions represent red blood cells surrounding inflammatory cells.

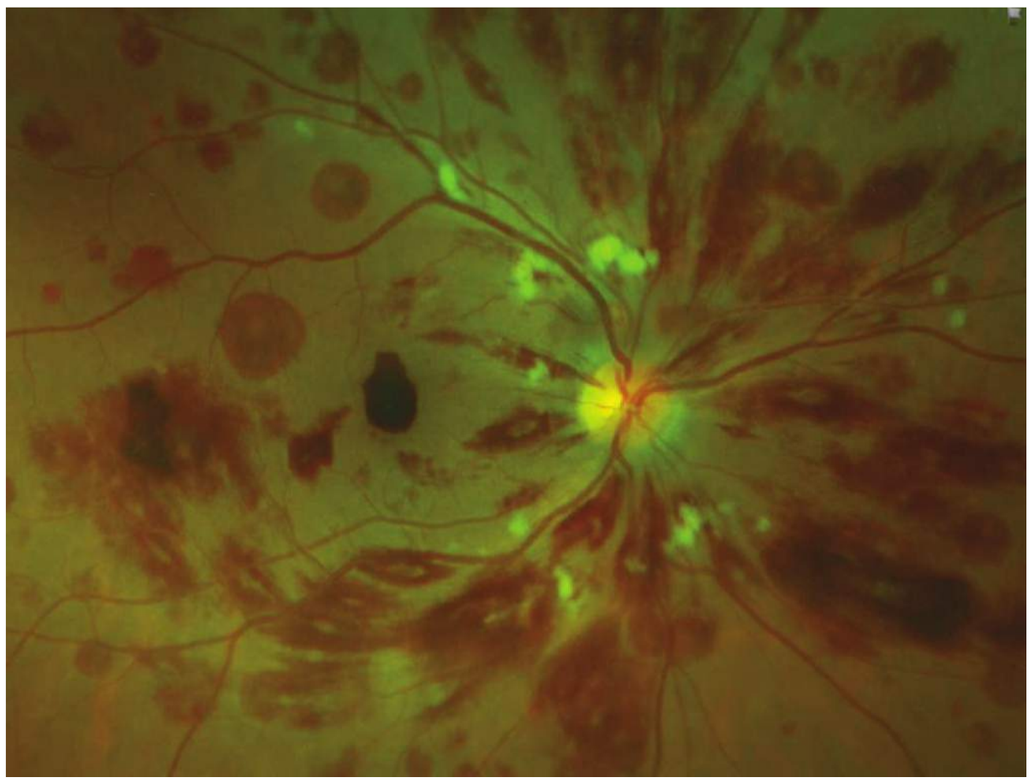


FIGURE 3.7 ■ **Roth Spots.** Multiple retinal hemorrhages with pale centers are seen. (Photo contributor: Tal Rubinstein, MD.)

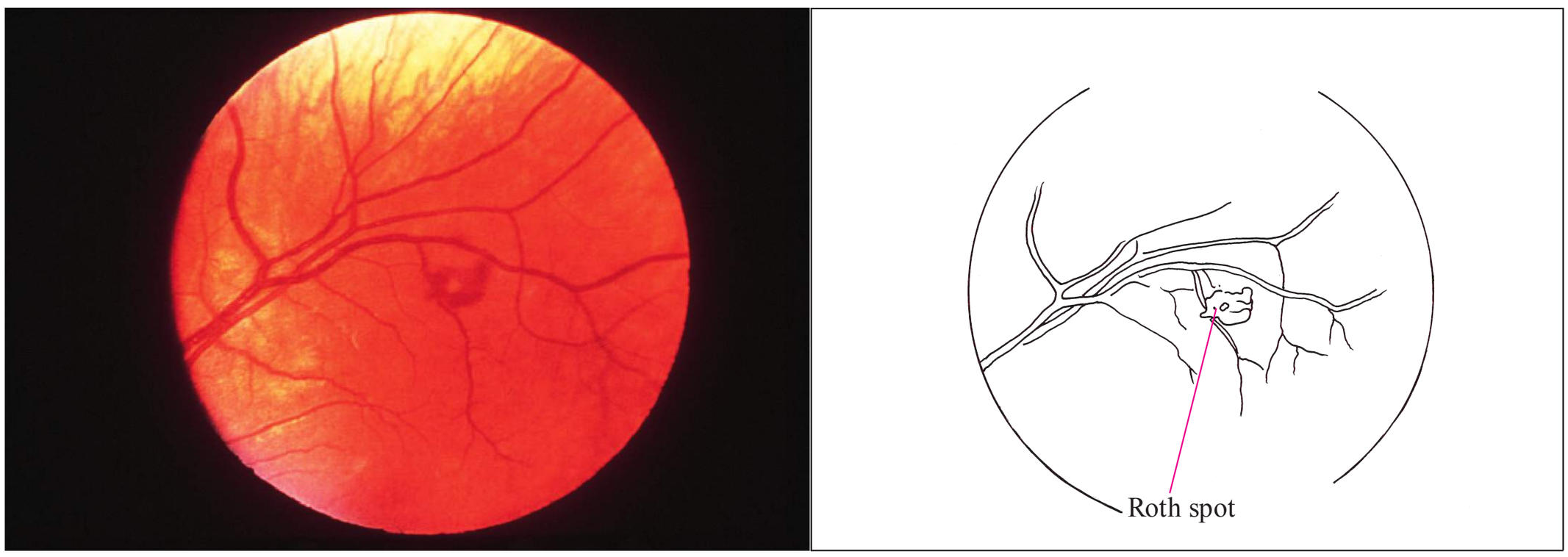


FIGURE 3.8 ■ **Roth Spot.** Retinal hemorrhage with pale center. (Photo contributor: William E. Cappaert, MD.)

Clinical Summary

Plaques, if present, are often found at arteriolar bifurcations. Patients may have signs and symptoms of vascular disease such as carotid bruits or stenosis, aortic stenosis, aneurysms, or atrial fibrillation. Amaurosis fugax, a transient loss of vision often described as a curtain of darkness obscuring vision with sight restoration within a few minutes, may be present in the history.

Cholesterol emboli (Hollenhorst plaques), associated with generalized atherosclerosis, often from carotid atheroma, are bright, highly refractile plaques. Platelet emboli (carotid artery or cardiac thrombus) are white and very difficult to visualize. Calcific emboli (cardiac valvular disease) are irregular and white or dull gray and much less refractile.

Management and Disposition

Referral for routine general medical evaluation is appropriate unless the patient presents with signs or symptoms consistent with showering of emboli, transient ischemic attack, or cerebrovascular accident, in which case admission should be considered.

Pearls

1. Retinal emboli may produce a loss of vision, either transient or permanent in nature.
2. Arteriolar occlusion may occur in either a central or a peripheral branch location.
3. Occurrence of retinal emboli should prompt the clinician to search for an embolic source as the event may be a precursor to impending ischemic stroke.

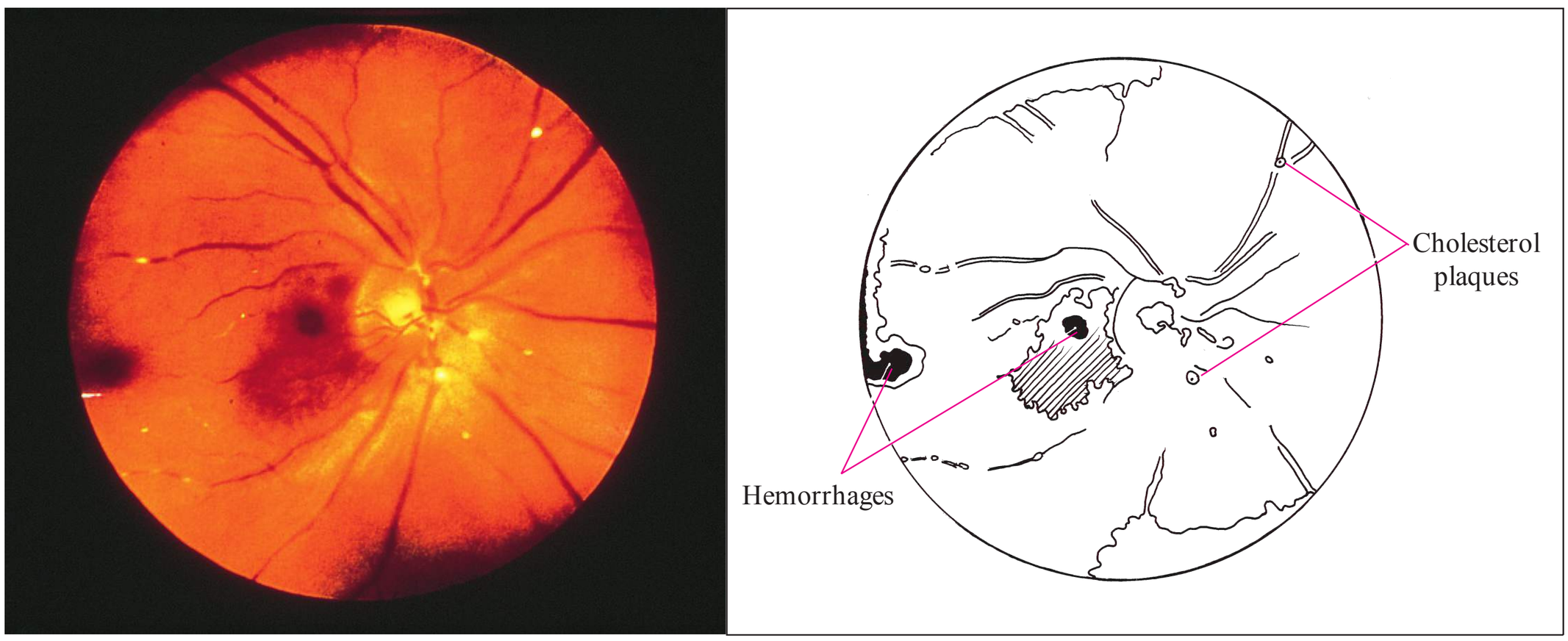


FIGURE 3.9 ■ **Emboli.** Refractile cholesterol plaques usually lodge at vessel bifurcations. (Photo contributor: William E. Cappaert, MD.)

Clinical Summary

In central retinal artery occlusion (CRAO), the typical patient experiences a sudden painless monocular loss of vision, either segmental or complete. Visual acuity may range from finger counting or light perception to complete blindness. Fundal findings may include the following: fundal paleness caused by retinal edema; the fovea does not have the edema and thus appears as a cherry-red spot; narrow and irregular retinal arterioles; and a "boxcar" appearance of the retinal venules.

Management and Disposition

Attempts to restore retinal blood flow may be beneficial if performed in a very narrow time window after the acute event. This may be accomplished by (1) decreasing intraocular pressure (IOP) with topical β-blocker eye drops or intravenous acetazolamide; (2) ocular massage, applied with cyclic pressure on the globe for 10 seconds, followed by release and then repeated. Urgent consultation with an ophthalmologist is indicated to determine if more aggressive acute therapy (paracentesis) is warranted. However, such aggressive treatment rarely alters the poor prognosis. Medical evaluation and treatment of associated findings may be warranted. Tissue plasminogen activator may be considered for lysis of an occluding thrombus.

Pearls

1. Visual decrement may be caused by a "low-flow" state (vs total occlusion). As this cannot be identified on presentation and can present hours later, immediate treatment and consultation is indicated regardless of the time of onset.
2. Sudden, painless monocular vision loss is typical.
3. CRAO may be associated with temporal arteritis. This diagnosis should be strongly considered in all patients presenting with signs and symptoms of CRAO who are older than 55 years.



FIGURE 3.10 ■ **Central Retinal Artery Occlusion.** The retinal pallor caused by retinal edema is well demonstrated, contrasting with the "cherry-red spot" of the nonedematous fovea. Note the vascular narrowing and the "boxcar" appearance of the venules. (Photo contributor: Aaron Sobol, MD.)

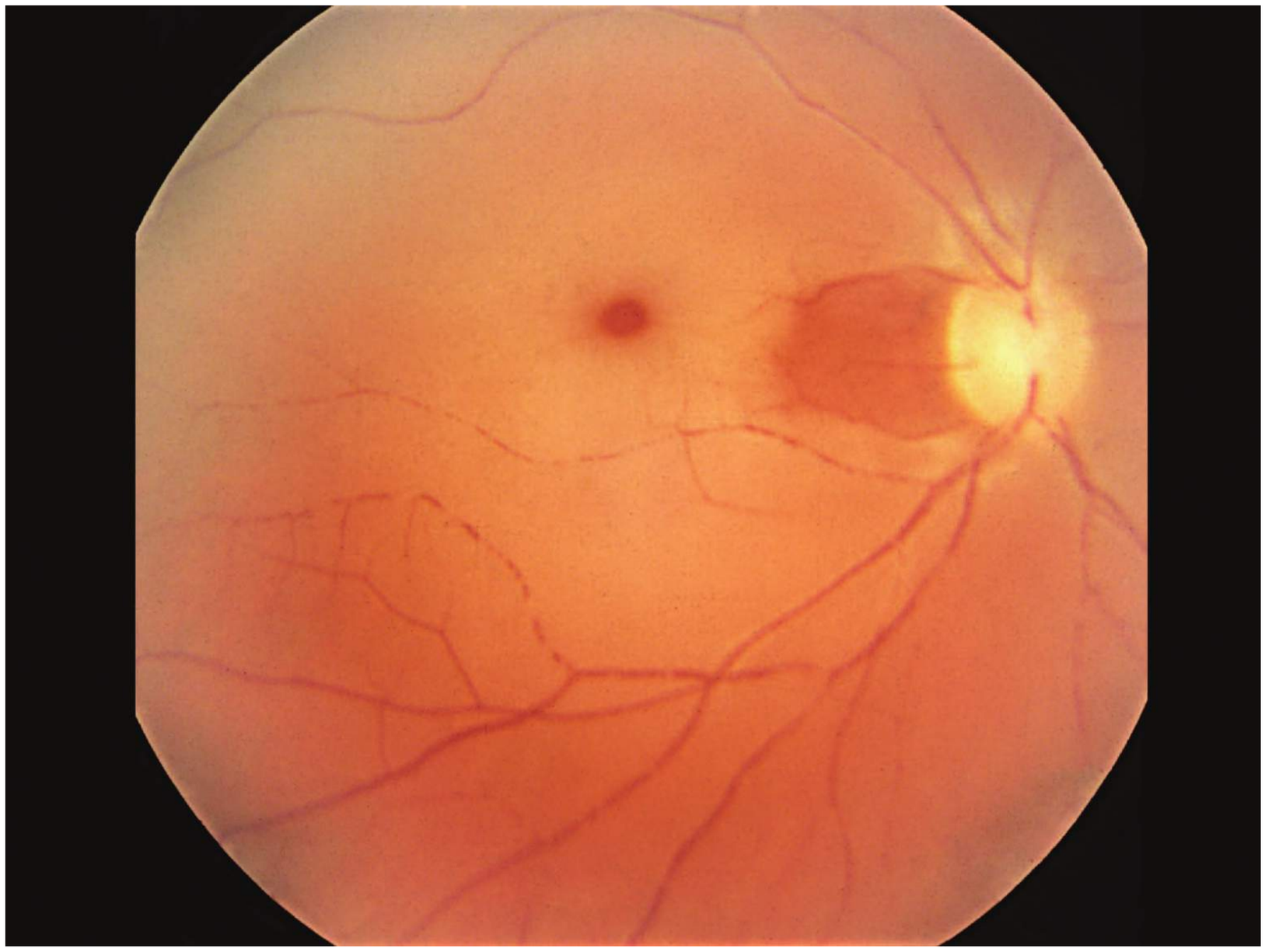


FIGURE 3.11 ■ **CRAO with Cilioretinal Artery Sparing**. "Hyperemia" of the fundus on the temporal side of the disc and sparing of the macular region owing to the presence of a patent cilioretinal artery. (Photo contributor: Thomas R. Hedges III, MD.)

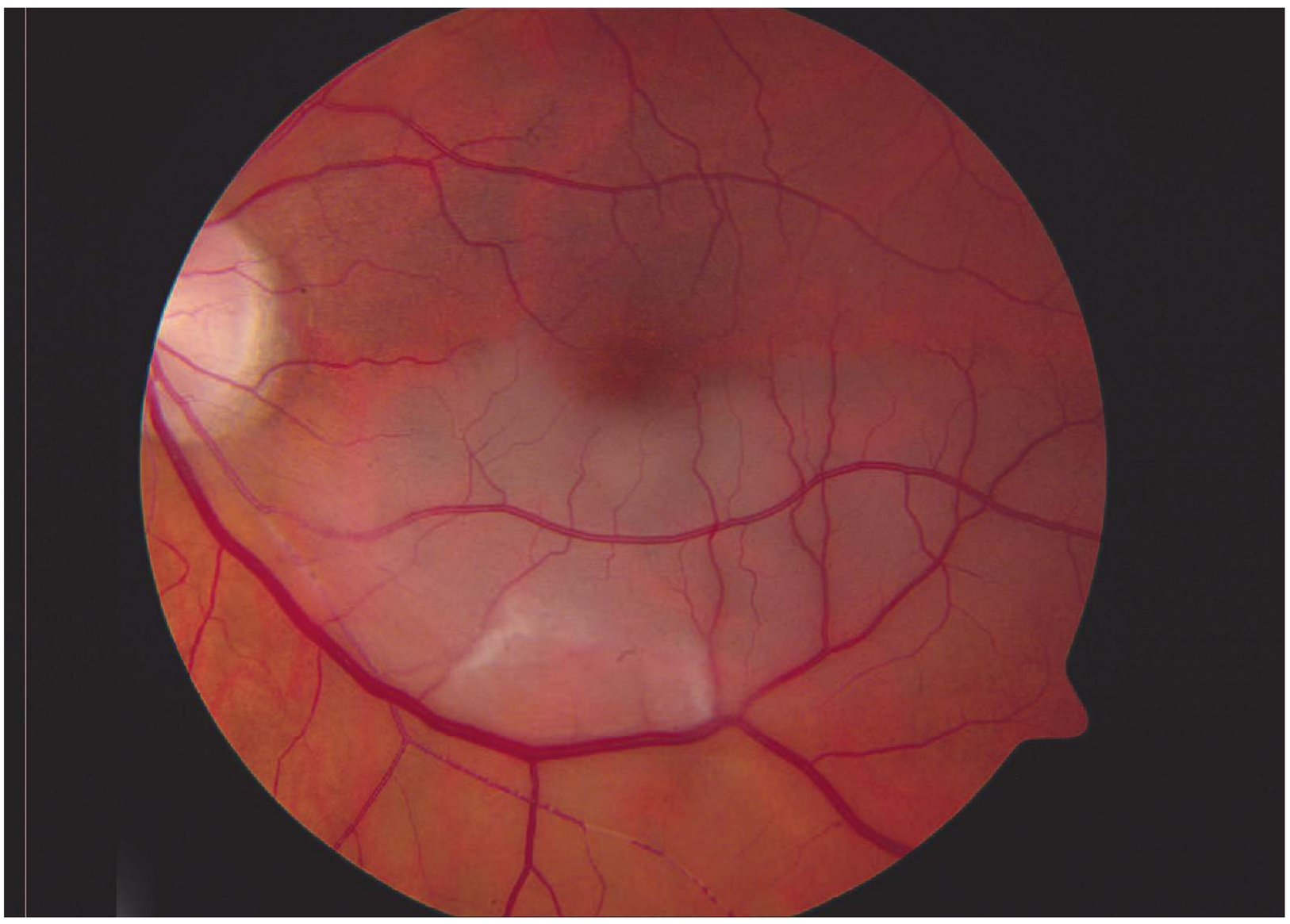


FIGURE 3.12 ■ **Branch Central Retinal Artery Occlusion.** Retinal whitening (owing to ischemia) and absence of blood flow in the infero-termporal branch retinal arteriole. (Photo contributor: Arun D. Singh, MD.)

Clinical Summary

Patients are usually older individuals and complain of sudden, painless visual loss in one eye. The vision loss is usually not as severe as CRAO and may vary from normal to hand motion. Funduscopy in a classic, ischemic central retinal vein occlusion (CRVO) shows a "blood and thunder" fundus: hemorrhages (including flame, dot, or blot, preretinal, and vitreous) and dilation and tortuosity of the venous system. The arterial system often shows narrowing. The disk margin may be blurred. Cotton wool spots and edema may be seen.

Management and Disposition

Treatment is rarely effective in preventing or reversing the damage done by the occlusion and is directed toward systemic evaluation to identify and treat contributing factors, hopefully decreasing the chance of contralateral CRVO. Ophthalmologic evaluation is necessary to confirm the diagnosis, estimate the amount of ischemia, and follow the patient so as to minimize sequelae of possible complications such as neovascularization and neovascular glaucoma.

Pearls

1. Sudden, painless visual loss in one eye should be evaluated promptly to determine its etiology.
2. Look for the classic "blood and thunder" funduscopic findings.
3. Consider the differential diagnosis of acute painful (glaucoma, retrobulbar neuritis) versus painless vision loss (CRAO, anterior ischemic optic neuropathy [AION], retinal detachment, subretinal neovascularization, and vitreous hemorrhage).

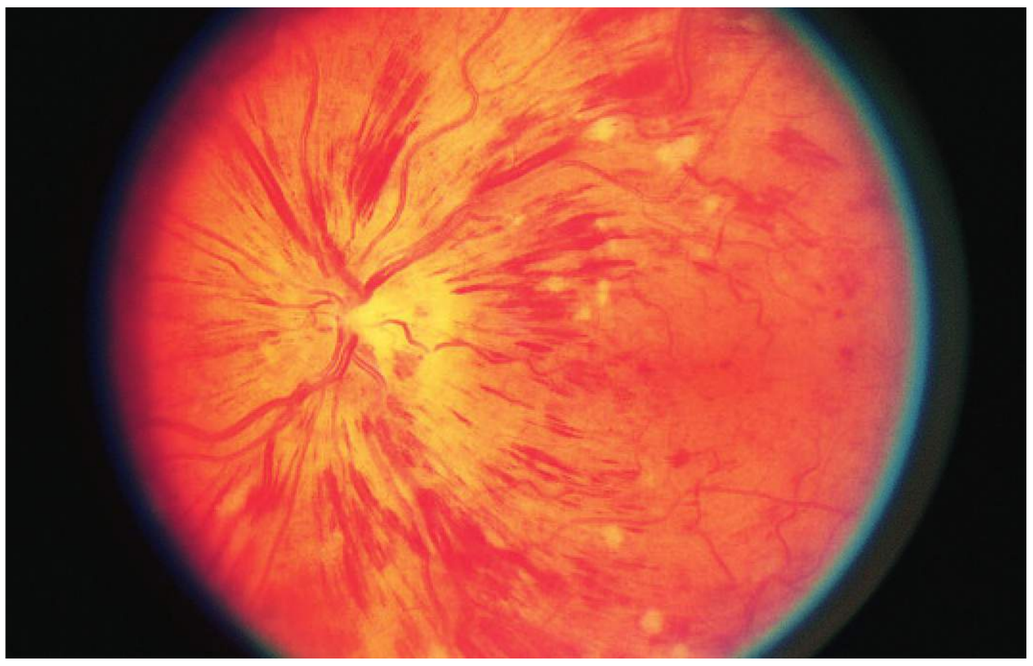


FIGURE 3.13 ■ **Central Retinal Vein Occlusion.** The amount of hemorrhage is the most striking feature in this photograph. Also note the blurred disk margin, the dilation and tortuosity of the venules, and the cotton wool spots. Retinal edema is suggested by blurring of the retinal details. (Photo contributor: Department of Ophthalmology, Naval Medical Center, Portsmouth, VA.)



FIGURE 3.14 ■ **Central Retinal Vein Branch Occlusion.** The hemorrhage seen is limited to a sector of the fundus, indicating that a branch occlusion has occurred. There is less edema, and a large portion of the fundus is unaffected. (Photo contributor: Richard E. Wyszynski, MD.)

HYPERTENSIVE RETINOPATHY

Clinical Summary

Fundus changes that may be seen with hypertension include generalized and focal narrowing of arterioles, generalized arteriolar sclerosis (resembling copper or silver wiring), arteriovenous crossing changes, hemorrhages (usually flame-shaped), retinal edema and exudation, cotton wool spots, microaneurysms, and disk edema.

Diabetic retinopathy, many hematologic and vascular diseases, traumas, localized ocular pathology, and papilledema should all be considered.

Management and Disposition

The patient's hypertension should be appropriately treated, and a search for other end-organ damage should be considered. The patient should be referred for appropriate long-term blood pressure management.

Pearls

1. Hypertensive arteriolar findings may be reversible if organic changes have not occurred in the vessel walls.
2. Always consider hypertensive retinopathy in the differential diagnosis of papilledema.



FIGURE 3.15 ■ **Hypertension.** Chronic, severe systemic hypertensive changes are demonstrated by hard exudates, increased vessel light reflexes, and sausage-shaped veins. (Photo contributor: Richard E. Wyszynski, MD.)

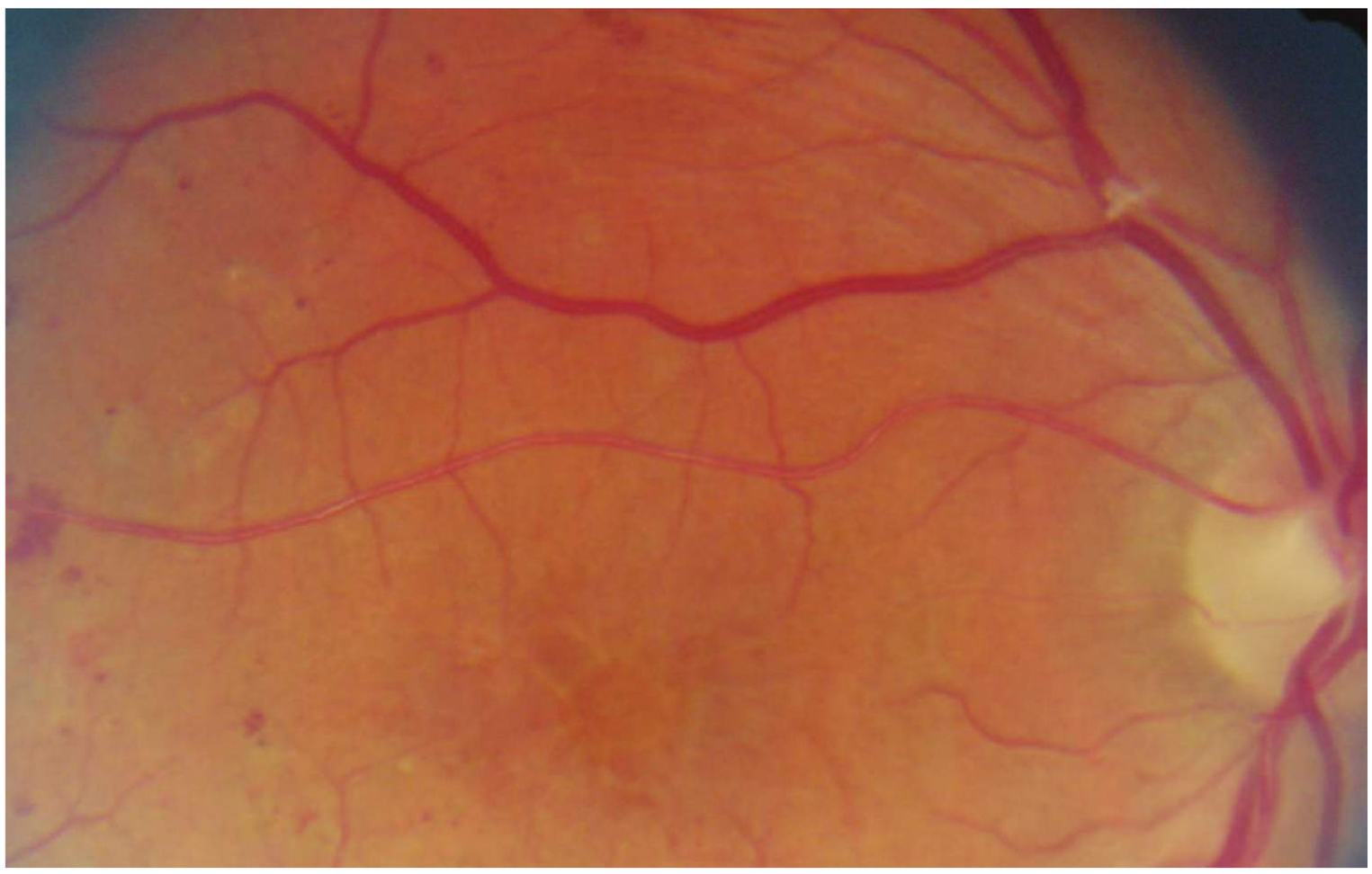


FIGURE 3.16 ■ **Copper and Silver Wiring.** Arteriolar changes seen in hypertensive retinopathy resemble copper (light reflex occupies most of the width) and silver (light reflex occupies the entire width of the arteriole) wiring. (Photo contributor: Aaron Sobol, MD.)

Clinical Summary

The early ocular manifestations of diabetes mellitus are referred to as background diabetic retinopathy (BDR). Fundus findings include flame or splinter hemorrhages (located in the superficial nerve fiber layer) or dot and blot hemorrhages (located deeper in the retina), hard exudates, retinal edema, and microaneurysms. If these signs are located in the macula, the patient's visual acuity may be decreased or at risk of becoming compromised, requiring laser treatment. Preproliferative diabetic retinopathy can show BDR changes plus cotton wool spots, intraretinal microvascular abnormalities, and venous beading. Proliferative diabetic retinopathy is demonstrated by neovascularization at the disk (NVD) or elsewhere (NVE). These require laser therapy owing to risk of severe visual loss from sequelae: vitreous hemorrhage, tractional retinal detachment, and severe glaucoma.

Many vascular and hematologic diseases—such as collagen vascular disease, sickle cell trait, hypertension, hypotension, anemia, leukemia, and inflammatory and infectious states—and ocular conditions can be associated with some or all of the above signs.

Management and Disposition

Routine ophthalmologic referral for laser or surgical treatment is indicated.

Pearls

1. Periodic ophthalmologic evaluations are recommended for all diabetic patients.
2. Microaneurysms typically appear 10 years after the initial onset of diabetes, although they may appear earlier in patients with juvenile diabetes.
3. Control of blood sugar alone does not prevent the development of retinopathy.
4. Blurred vision can also occur from acute increases in serum glucose, causing lens swelling and a refractive shift even in the absence of retinopathy.



FIGURE 3.17 ■ **Background Diabetic Retinopathy.** An example of diabetic maculopathy with a typical circinate lipid ring. (Photo contributor: Richard E. Wyszynski, MD.)

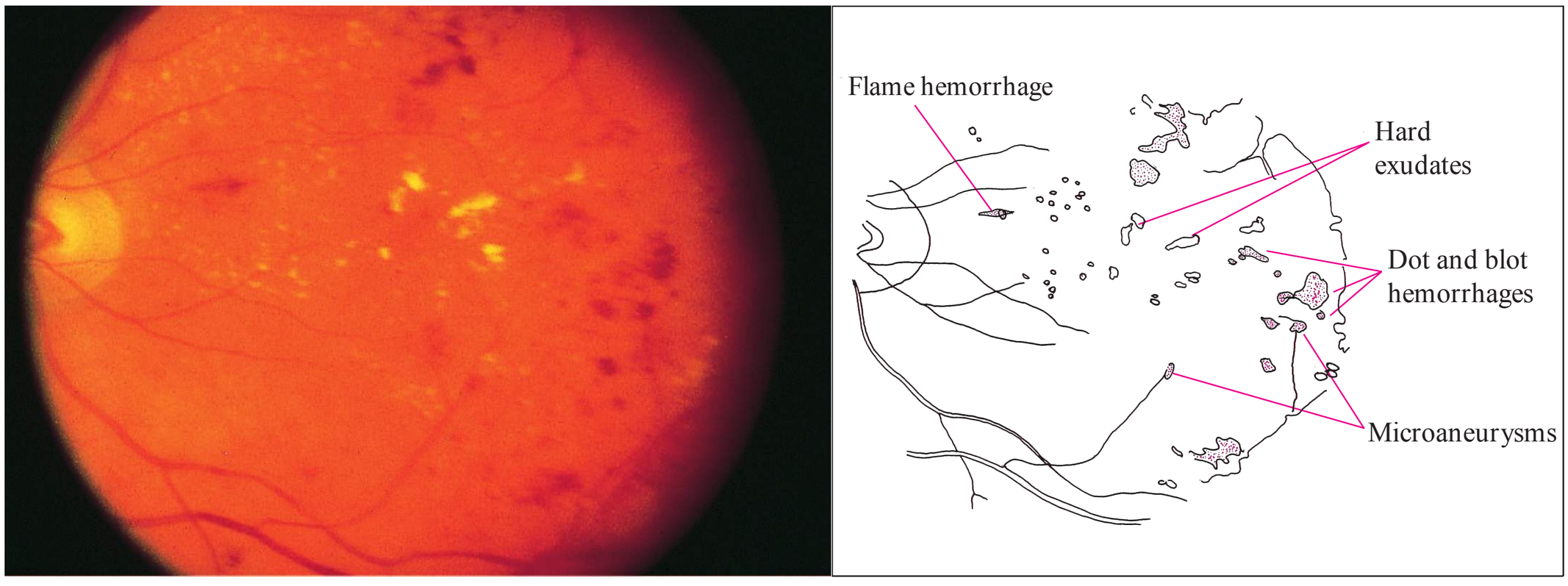


FIGURE 3.18 ■ **Background Diabetic Retinopathy.** Hard exudates, dot hemorrhages, blot hemorrhages, flame hemorrhages, and microaneurysms are present. Because these changes are located within the macula, this is classified as diabetic maculopathy. (Photo contributor: Richard E. Wyszynski, MD.)

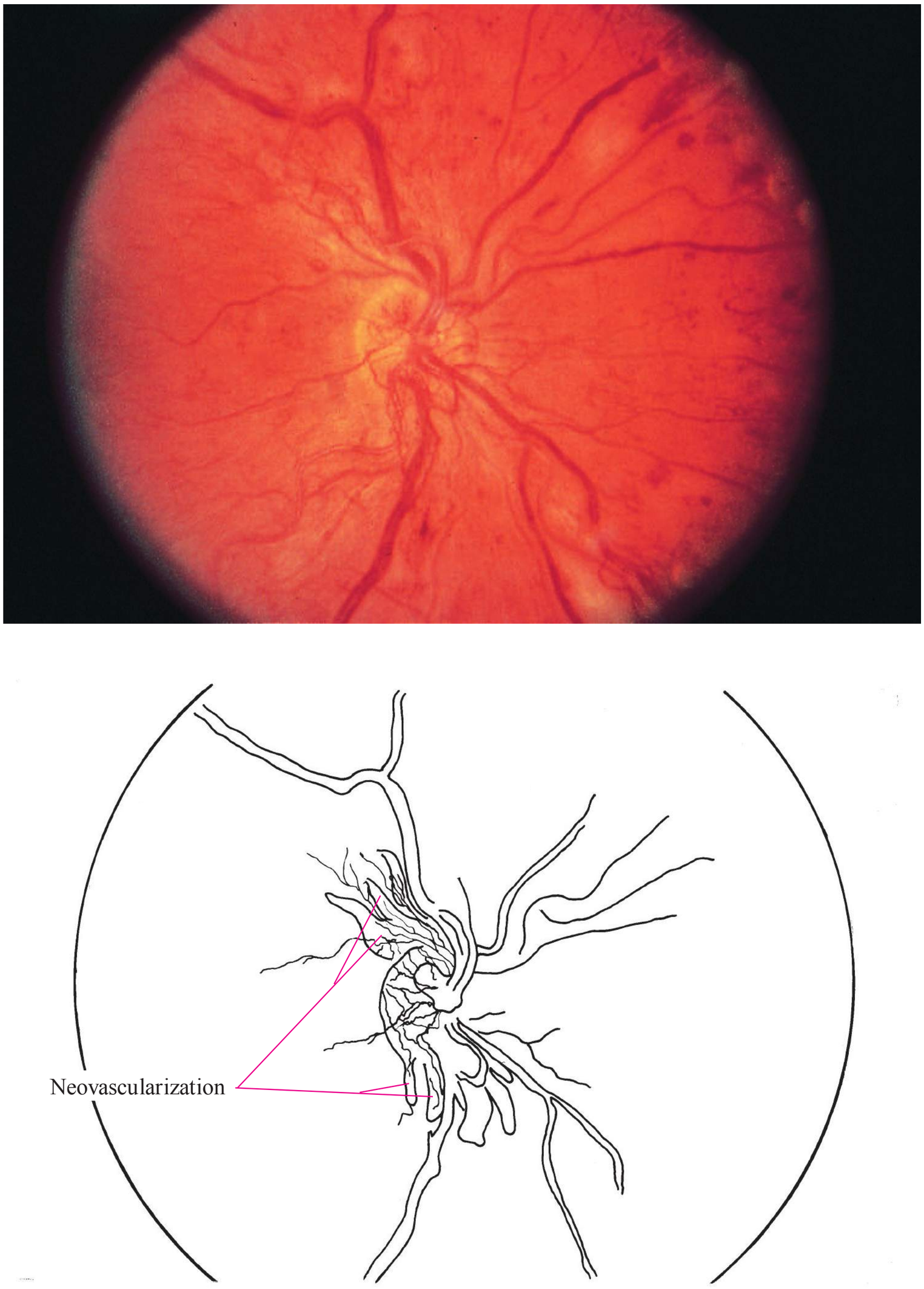


FIGURE 3.19 ■ **Proliferative Diabetic Retinopathy.** In addition to the signs seen in background and preproliferative diabetic retinopathy, neovascularization is seen here coming off the disk. (Photo contributor: Richard E. Wyszynski, MD.)

Clinical Summary

Patients may complain of floaters followed by the sudden loss or deterioration of vision in the affected eye, although bilateral hemorrhage can occur. The red reflex is diminished or absent, and the retina is obscured because of the bleeding. Large sheets or three-dimensional collections of red to red-black blood may be detected.

Multiple underlying etiologies include proliferative diabetic retinopathy, retinal or vitreous detachments, hematologic diseases, trauma (ocular or shaken impact syndrome), subarachnoid hemorrhage (SAH), collagen vascular disease, infections, macular degeneration, and tumors.

Management and Disposition

Refer to an ophthalmologist and an appropriate physician for associated conditions. Ophthalmic observation, photocoagulation, and surgery are all therapeutic options. Bed rest may help to increase visualization of the fundus.

Pearl

1. The patient's vision may improve somewhat after a period of sitting or standing as the blood layers out.

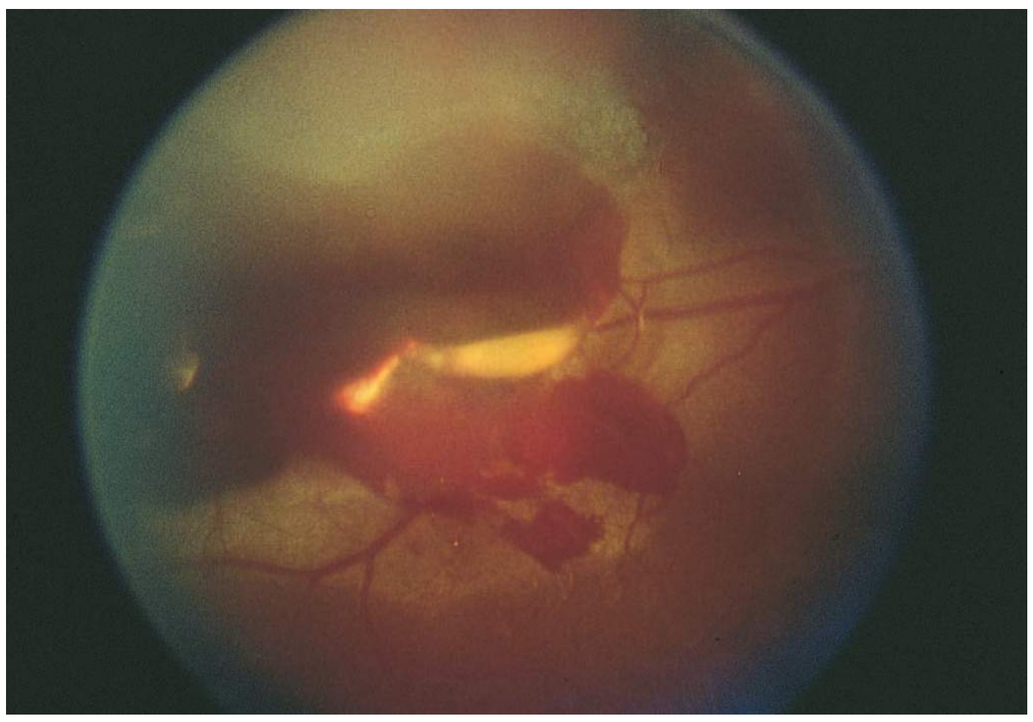


FIGURE 3.20 ■ **Vitreous Hemorrhage.** Large amount of vitreous hemorrhage associated with metallic intraocular foreign body. The large quantity of blood obscures visualization of retinal details. (Photo contributor: Richard E. Wyszynski, MD.)

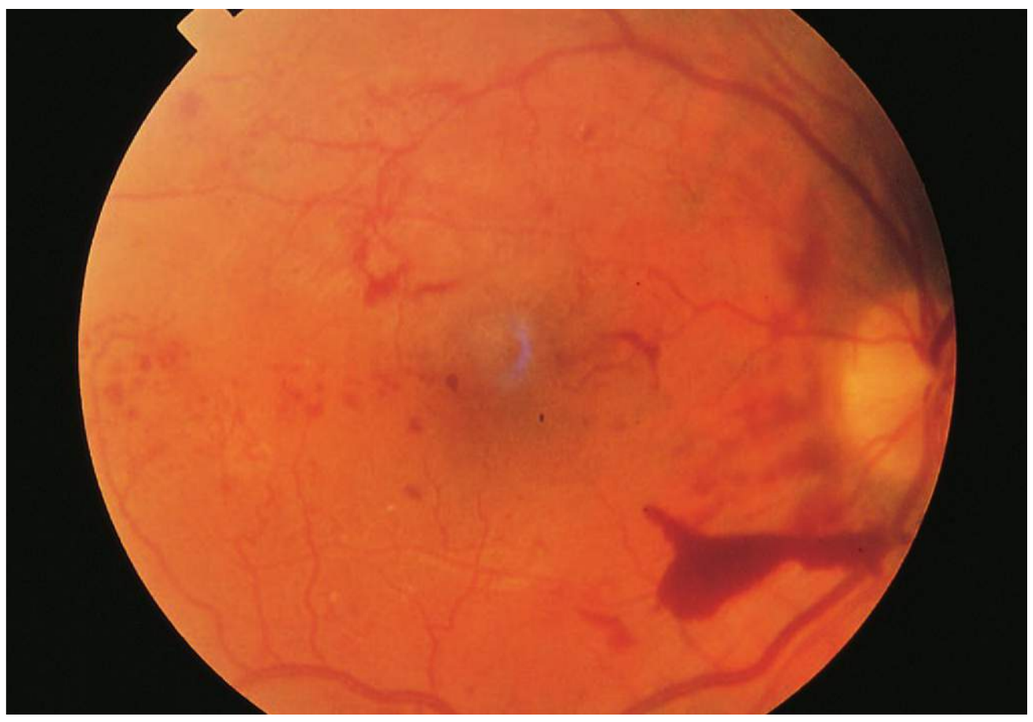


FIGURE 3.21 ■ **Vitreous Hemorrhage.** A smaller amount of vitreous hemorrhage is more easily photographed. Gravitational effect on the vitreous blood creates the appearance of a flat meniscus (keel-shaped blood) in this patient with vitreous hemorrhage associated with proliferative diabetic retinopathy. (Photo contributor: Richard E. Wyszynski, MD.)

RETINAL DETACHMENT

Clinical Summary

Patients often complain of monocular, decreased visual function and may describe a shadow or curtain descending over the eye. Other complaints include cloudy or smoky vision, floaters, or momentary flashes of light. Monocular visual field defects may be noted, and central visual acuity is diminished with macular involvement. Fundal examination may reveal a billowing or tentlike elevation of retina compared with adjacent areas. The elevated retina often appears gray. Retinal holes and tears may be seen, but often the holes, tears, and retinal detachment cannot be seen without indirect ophthalmoscopy.

Retinal detachments caused by retinal tears or holes can be associated with trauma, previous ocular surgery, nearsightedness, family history of retinal detachment, and Marfan disease. Retinal detachments caused by traction on the retina by an intraocular process can be because of systemic influences in the eye, such as diabetes mellitus or sickle cell trait. Occasionally retinal detachments are caused by tumors or exudative processes that elevate the retina. Symptoms of "light flashes" may occur with vitreous changes in the absence of retinal pathology. Patients may note flashes of light occurring only in a darkened environment because of the mechanical stimulation of the retina from the extraocular muscles, usually in a nearsighted individual.

Management and Disposition

Urgent ophthalmologic evaluation and treatment are warranted.

Pearls

1. Often patients have had sensation of flashes of light that occur in a certain area of a visual field in one eye, corresponding to the pathologic pulling on the corresponding retina.
2. Visual loss may be gradual or sudden.

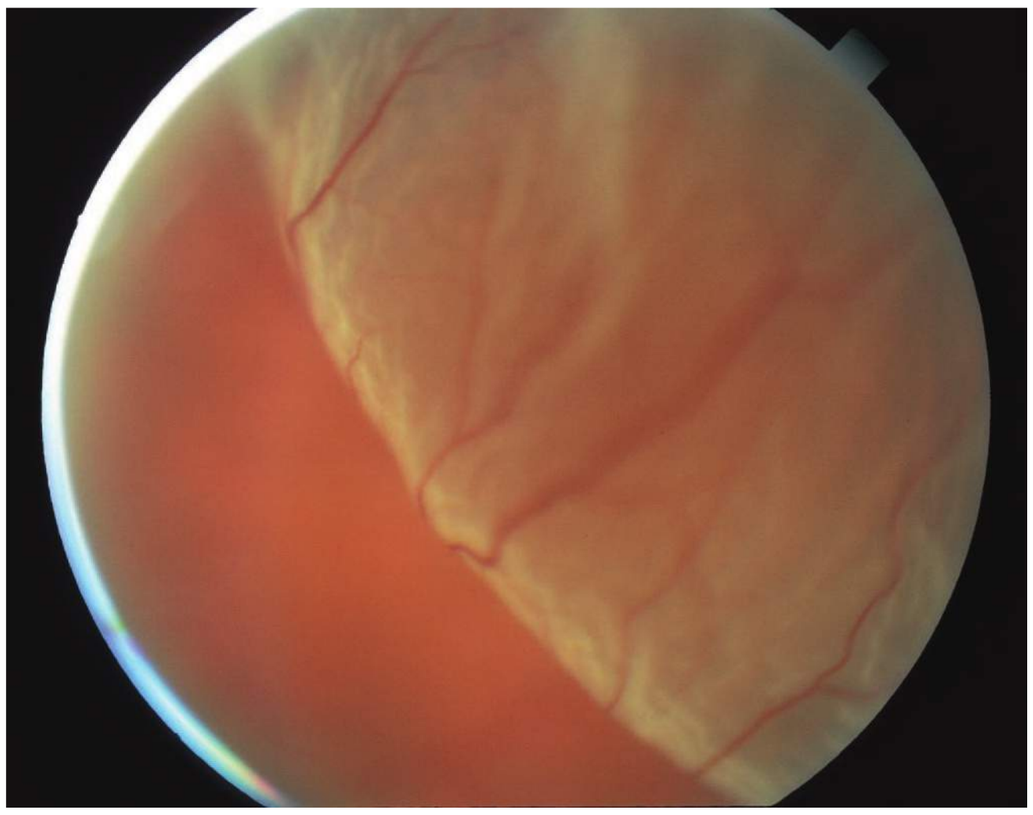


FIGURE 3.22 ■ **Retinal Detachment**. A fold of detached retina is seen drooping well in front of the posterior pole giving the appearance of a "three-dimensional" fundus. (Photo contributor: Department of Ophthalmology, Naval Medical Center, Portsmouth, VA.)

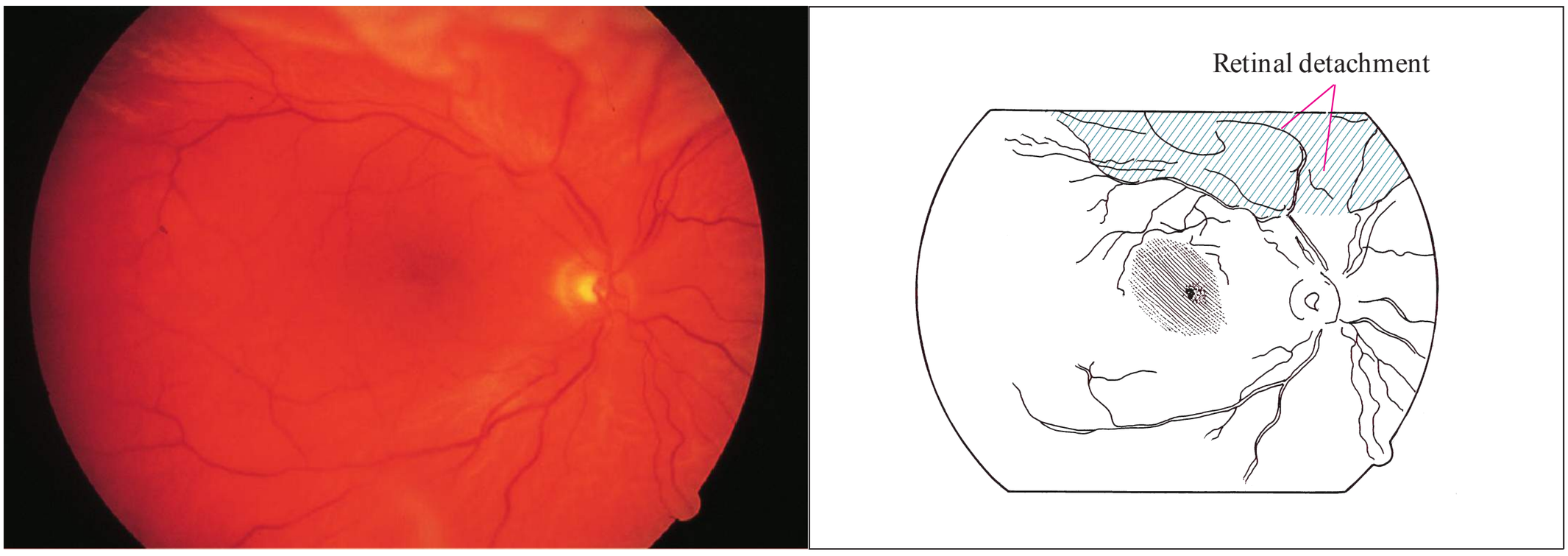


FIGURE 3.23 ■ **Retinal Detachment.** Undulating, out-of-focus, elevated retina is seen with few vessels in focus. (Photo contributor: Richard E. Wyszynski, MD.)

Clinical Summary

Patients may complain of the gradual and usually painless onset of the following visual sensations: floaters, scintillating scotomas (quivering blind spots), decreased peripheral visual field, and metamorphopsia (wavy distortion of vision). Cytomegalovirus (CMV) infiltrates appear as focal, small (but may be larger, confluent) white lesions in the retina that look like cotton wool spots. CMV is a necrotizing virus that is spread hematogenously, so that damage is concentrated in the retina adjacent to the major vessels and the optic disk. Often hemorrhage is involved with significant retinal necrosis (dirty white with a granular appearance), giving the "pizza pie" or "cheese and ketchup" appearance. Optic nerve involvement and retinal detachments can be present.

The differential includes other infections such as toxoplasmosis, other herpesviruses, syphilis, and occasionally other opportunistic infections.

Management and Disposition

Reversal, if possible, of immunosuppression; antiviral agents have been used effectively to threat this condition.

Pearls

1. HIV retinopathy consists of scattered retinal hemorrhages and scattered, multiple cotton wool spots that resolve over time, whereas CMV lesions will typically progress.
2. Although exposure to the CMV virus is widespread, the virus rarely produces a clinically recognized disease in nonimmunosuppressed individuals.

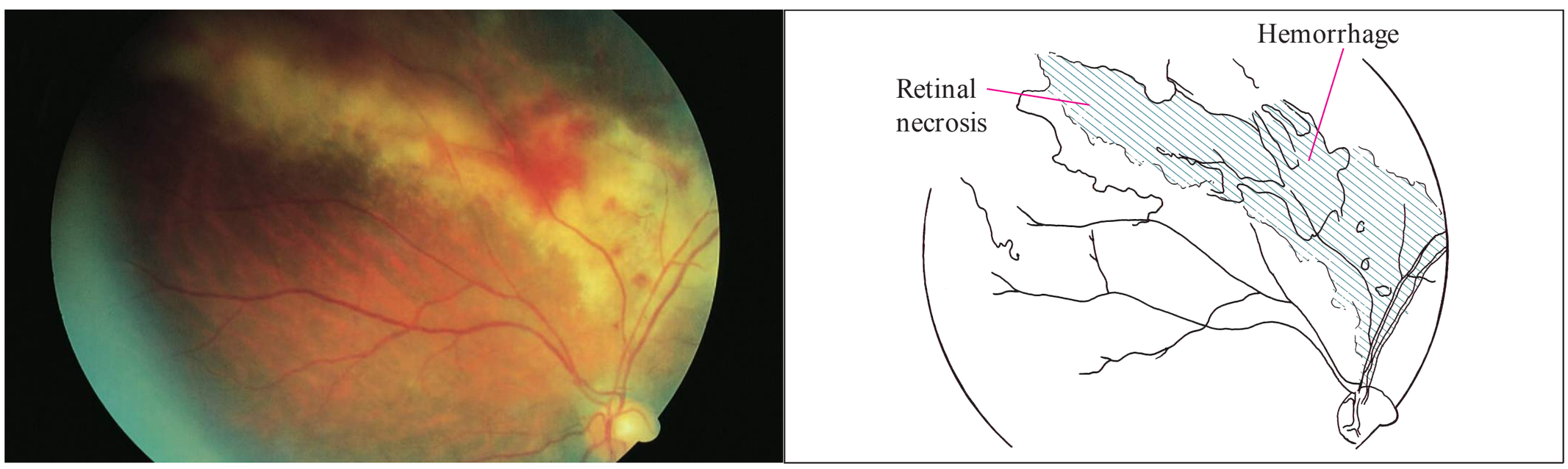


FIGURE 3.24 ■ **CMV Retinitis.** "Pizza pie" or "cheese and ketchup" appearance is demonstrated by hemorrhages and the dirty, white, granular-appearing retinal necrosis adjacent to major vessels. (Photo contributor: Richard E. Wyszynski, MD.)

Clinical Summary

Papilledema involves swelling of the optic nerve head, usually in association with elevated intracranial pressure. The optic disks are hyperemic with blurred disk margins; the venules are dilated and tortuous. The optic cup may be obscured by the swollen disk. There may be flame hemorrhages and infarctions (white, indistinct cotton wool spots) in the nerve fiber layer and edema in the surrounding retina.

Ocular inflammation (eg, papillitis), tumors or trauma, central retinal artery or vein occlusion, optic nerve drusen, and marked hyperopia may present with similar findings.

Management and Disposition

Expeditious ophthalmologic and medical evaluation is warranted.

Pearls

1. The top of a swollen disk and the surrounding unaffected retina will not both be in focus on the same setting on direct ophthalmoscopy.
2. Papilledema is a bilateral process, though it may be slightly asymmetric. A unilateral swollen disk suggests a localized ocular or orbital process.
3. Vision is usually normal acutely, though the patient may complain of transient visual changes. The blind spot is usually enlarged.
4. Diplopia from sixth cranial nerve palsy can be associated with increased intracranial pressure and papilledema.



FIGURE 3.25 ■ **Papilledema.** Note blurred disk margins and congested disk. (Photo contributor: Arun D. Singh, MD.)

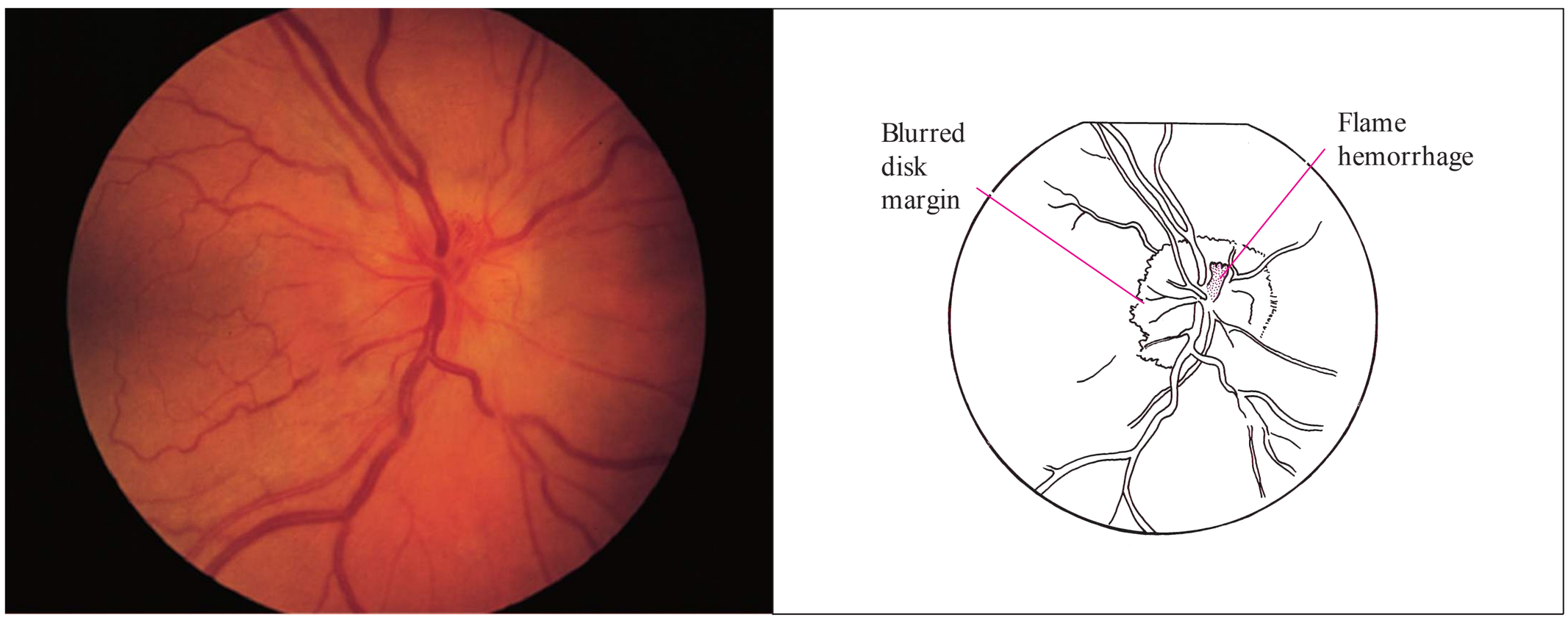


FIGURE 3.26 ■ **Papilledema.** Disk is hyperemic and swollen with loss of sharp margins. The venules are dilated and tortuous. The cup is obscured. A small flame hemorrhage is seen at 12- to 1-o'clock position on the disk margin. (Photo contributor: Department of Ophthalmology, Naval Medical Center, Portsmouth, VA.)

Clinical Summary

Most cases of optic neuritis are retrobulbar and involve no changes in the fundus, or optic disk, during the acute episode. With time, variable optic disk pallor may develop. Typical retrobulbar optic neuritis presents with sudden or rapidly progressing monocular vision loss in patients younger than 50 years. There is a central visual field defect that may extend to the blind spot. Pain on movement of the globe is common. The pupillary light response is diminished in the affected eye. Over time the vision improves partially or completely; minimal or severe optic atrophy may develop. Papillitis, inflammation of the intraocular portion of the optic nerve, will accompany disk swelling, with a few flame hemorrhages and possible cells in the vitreous.

Optic neuritis must be differentiated from papilledema (bilateral disk swelling, typically with no acute visual loss with the exception of transient visual changes), ischemic neuropathy (pale, swollen disk in an older individual with sudden monocular vision loss), tumors, and metabolic or endocrine disorders. Most cases of optic neuritis are of unknown etiology. Some known causes of optic neuritis include demyelinating disease, infections (including viral, syphilis, tuberculosis, sarcoidosis), or inflammations from contiguous structures (sinuses, meninges, orbit).

Management and Disposition

Treatment is controversial; often none is recommended. Oral steroids may worsen prognosis in certain cases. Intravenous steroids should be considered after consultation with an ophthalmologist.

Pearls

1. Monocular vision loss with pain on palpation of the globe or with eye movement and decreased color vision and color desaturation with loss of contrast are clinical clues to the diagnosis.
2. Sudden or rapidly progressing central vision loss is characteristic.
3. Most cases of acute optic neuritis are retrobulbar. Thus, ophthalmoscopy shows a normal fundus.
4. Suspect temporal arteritis in older patients.

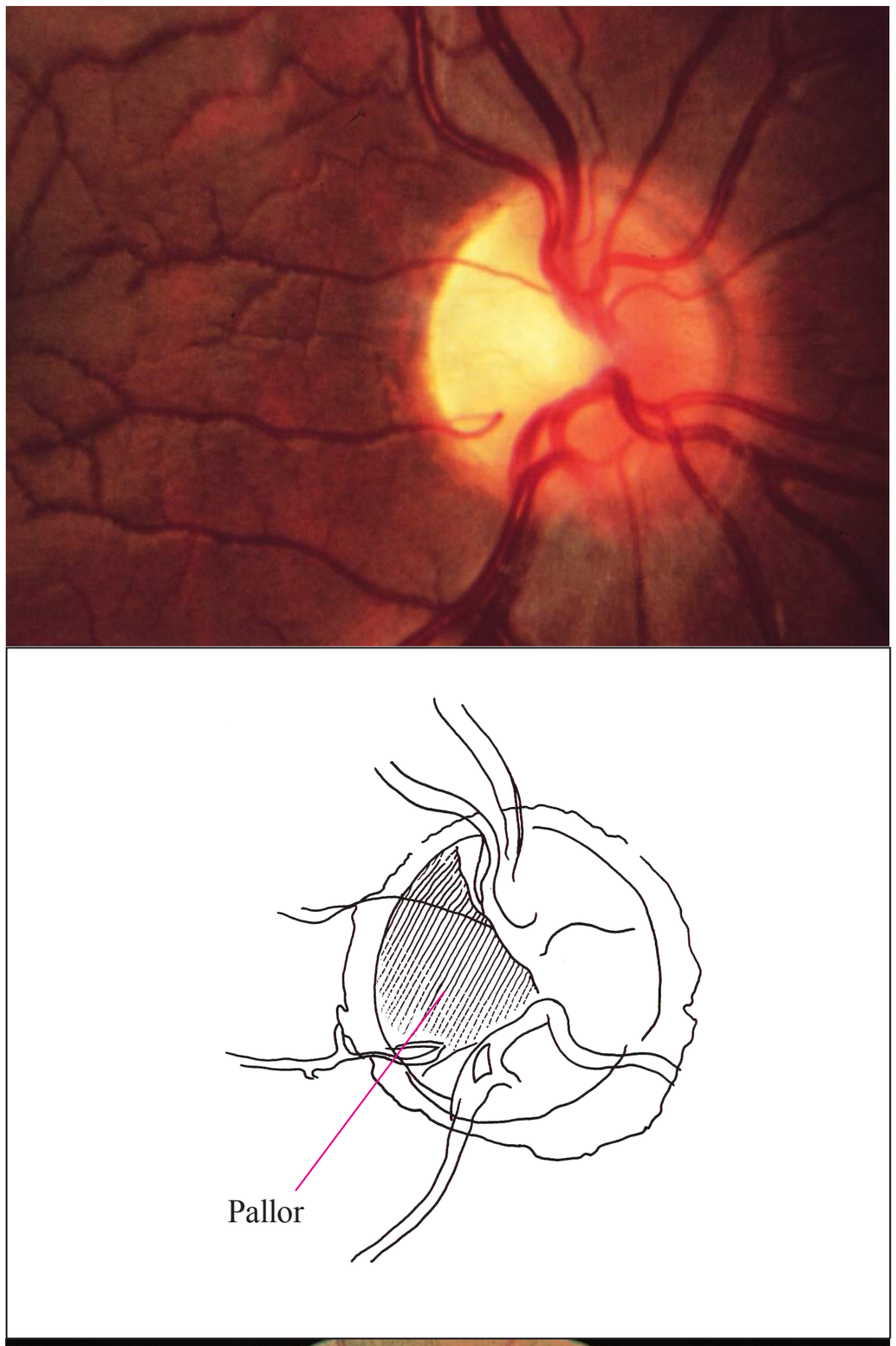


FIGURE 3.27 ■ **Optic Nerve Pallor.** Optic nerve pallor, either segmental (top) or generalized (bottom), is a nonspecific change that may be associated with a previous episode of optic neuritis or other insults to the optic nerve. (Photo contributor: Richard E. Wyszynski, MD.)

ANTERIOR ISCHEMIC OPTIC NEUROPATHY

Clinical Summary

Anterior ischemic optic neuropathy (AION) presents with a sudden loss of visual field (often altitudinal), usually involving fixation, in an older individual. The loss is usually stable after onset, with no improvement, and only occasionally, progressive over several days to weeks. Pale disk swelling is present involving a sector or the full disk, with accompanying flame hemorrhages. The cup to disc ratio is typically small (0.1-0.2) bilaterally.

The common, nonarteritic causes of AION (probably arteriosclerosis) need to be differentiated from arteritic ones, such as giant cell arteritis. If untreated, the latter will involve the other eye in 75% of cases, often in a few days to weeks. These elderly individuals often have weight loss, masseter claudication, weakness, myalgias, elevated sedimentation rate, and painful scalp, temples, or forehead.

Management and Disposition

Routine ophthalmologic and medical evaluation is appropriate.

Pearls

1. Consider AION in an elderly patient with sudden, usually painless, visual field loss.
2. Rule out giant cell arteritis. These patients tend to be older (age > 55 years) and may have associated CRAO or cranial nerve palsies (III, IV, or VI) with diplopia.

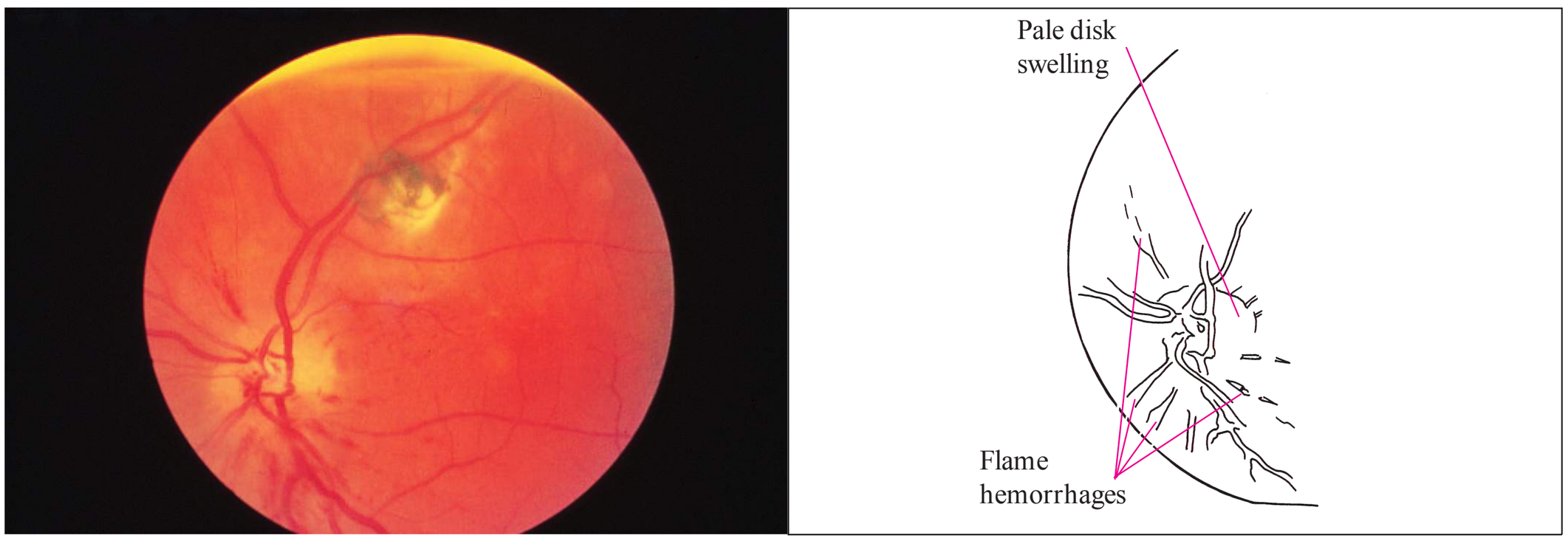


FIGURE 3.28 ■ **Anterior Ischemic Optic Neuropathy.** Pale disk swelling and flame hemorrhages are present. This patient also has an unrelated retinal scar owing to toxoplasmosis. (Photo contributor: William E. Cappaert, MD.)

Clinical Summary

Acute narrow or closed-angle glaucoma (ACG) results from a physical impedance of aqueous humor outflow. Symptoms range from colored halos around lights and blurred vision to severe pain (described as a headache or brow ache) with nausea and vomiting. IOP is markedly elevated. Perilimbal vessels are injected, the pupil is mid-dilated and poorly reactive to light, and the cornea may be hazy and edematous.

Two-thirds of glaucoma patients have open-angle glaucoma. Often they are asymptomatic. They may have a family history of glaucoma. Funduscopy may show asymmetric cupping of the optic nerves. The optic nerve may show notching, local thinning of tissue, or disk hemorrhage. Optic cups enlarge, especially vertically, with progressive damage. Tissue loss is associated with visual field abnormalities. The IOP is often but not always greater than 21 mm Hg.

Management and Disposition

Acute ACG requires emergent ophthalmologic consultation and administration of medications to decrease IOP such as β-blocker drops (timolol), carbonic anhydrase inhibitors (acetazolamide), cholinergic-stimulating drops (pilocarpine), hyperosmotic agents (osmoglyn), and α-adrenergic agonists (apraclonidine). Open-angle glaucoma is treated with long-term ophthalmic evaluation and treatment with medications and laser or surgery.

Pearls

1. Nausea, vomiting, and headache may obscure the diagnosis. Use digital globe palpation routinely in patients with these complaints.
2. Open-angle glaucoma usually causes no symptoms other than gradual loss of vision.
3. Congenital glaucoma is rare. However, because of prognosis if diagnosis is delayed, consider congenital glaucoma in infants and children with tearing, photophobia, enlarged eyes, or cloudy corneas.
4. Asymmetric cupping, enlarged cups, and elevated IOP are hallmarks of open-angle glaucoma.



FIGURE 3.30 ■ **Glaucomatous Cupping.** The cup is not central; it is elongated toward the rim superotemporally. (Photo contributor: Department of Ophthalmology, Naval Medical Center, Portsmouth, VA.)

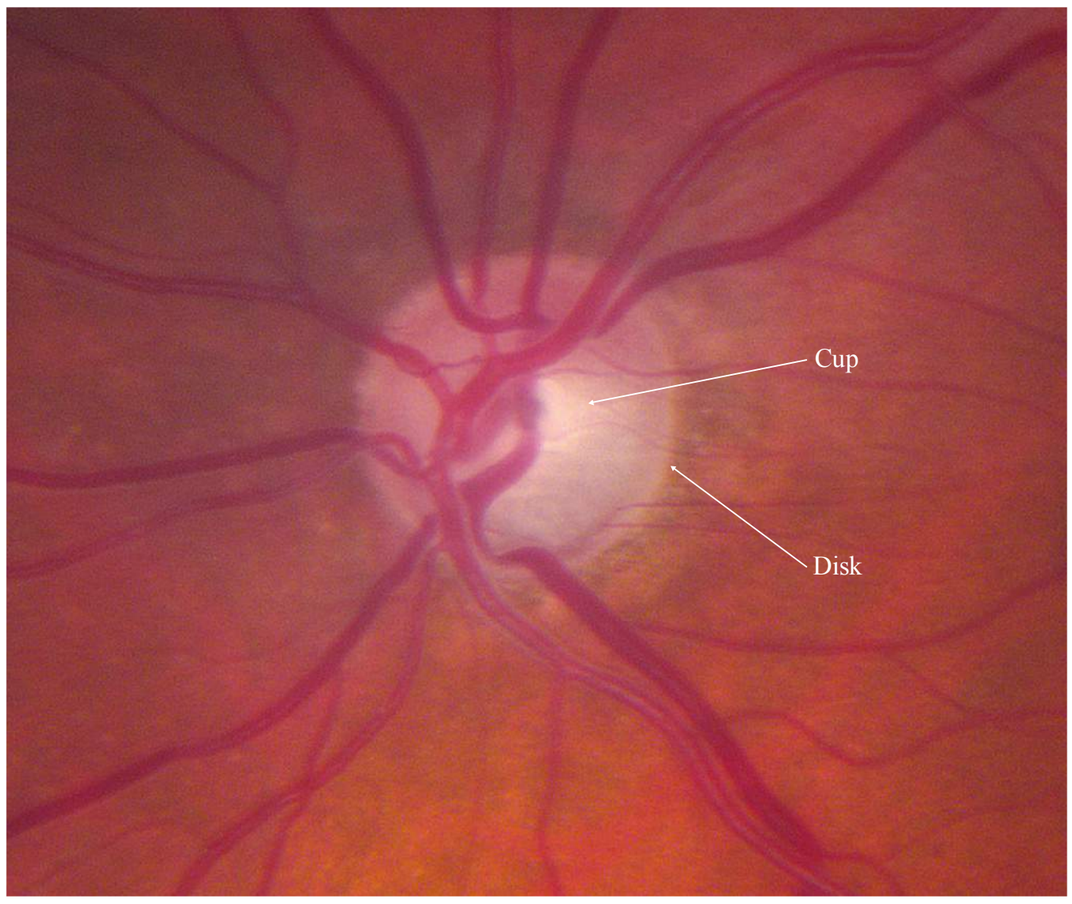


FIGURE 3.29 ■ **Normal Cupping.** The cup is central and is less than one half the diameter of the disc. (Photo contributor: Jeffrey Goshe, MD.)

Clinical Summary

Subhyaloid hemorrhage appears as extravasated blood beneath the retinal layer. These are often described as "boat-shaped" hemorrhages to distinguish them from the "flame-shaped" hemorrhages on the superficial nerve fiber layer of the retina. They may occur as a result of blunt trauma but are perhaps best known as a marker for subarachnoid hemorrhage (SAH). In SAH, the hemorrhages appear as a "puff" of blood emanating from the central disk.

SAH, shaken impact syndrome, hypertensive retinopathy, and retinal hemorrhage should all be considered and aggressively evaluated.

Management and Disposition

No specific treatment is required for subhyaloid hemorrhage. Treatment is dependent on the underlying etiology. Appropriate specialty referral should be made in all cases.

Pearls

1. A funduscopic examination looking for subhyaloid hemorrhage should be included in all patients with severe headache, unresponsive pediatric patients, or those with altered mental status.
2. The appearance of a retinal hemorrhage indicates its anatomic location. A subhyaloid hemorrhage lies over the retinal vessels—thus, they cannot be seen on funduscopic examination. In a subretinal hemorrhage (see Fig. 3.4), the vessels lie superficial to the hemorrhage and thus are easily seen.

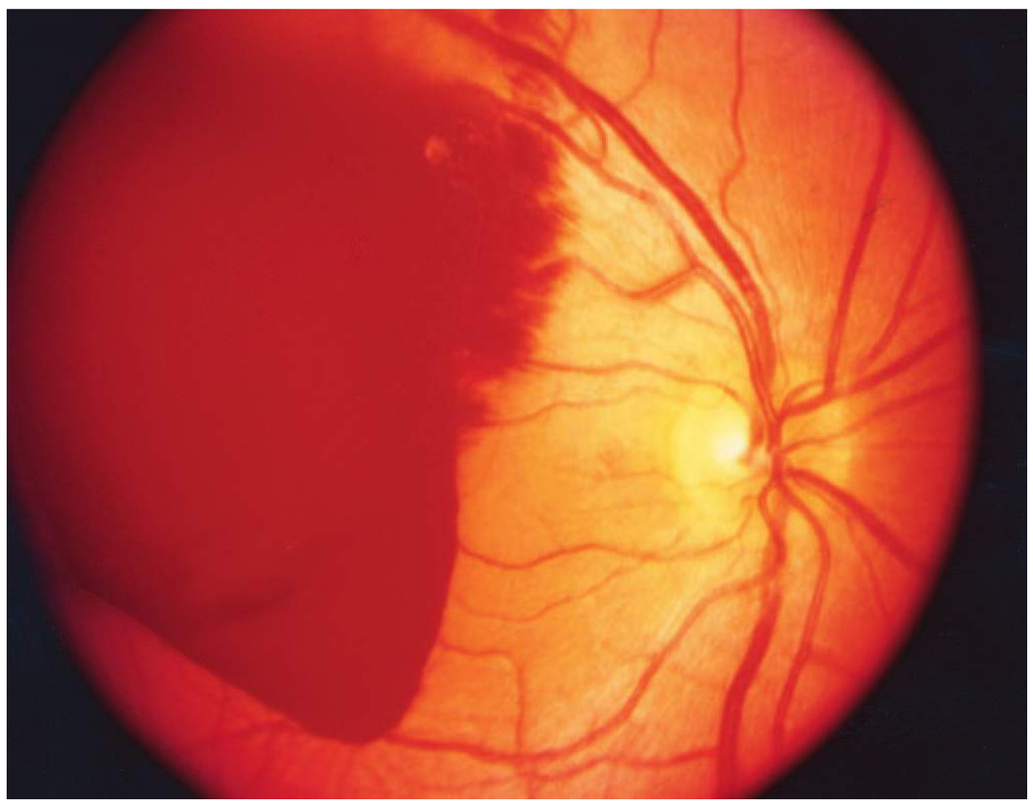


FIGURE 3.31 ■ **Subhyaloid Hemorrhage.** Subhyaloid hemorrhage seen on funduscopic examination in a patient with subarachnoid hemorrhage. (From Edlow JA, Caplan LR. Primary care: avoiding pitfalls in the diagnosis of subarachnoid hemorrhage. N Engl J Med. 2000;342:29-36. Reproduced with permission from John J. Weiter, MD, PhD.)

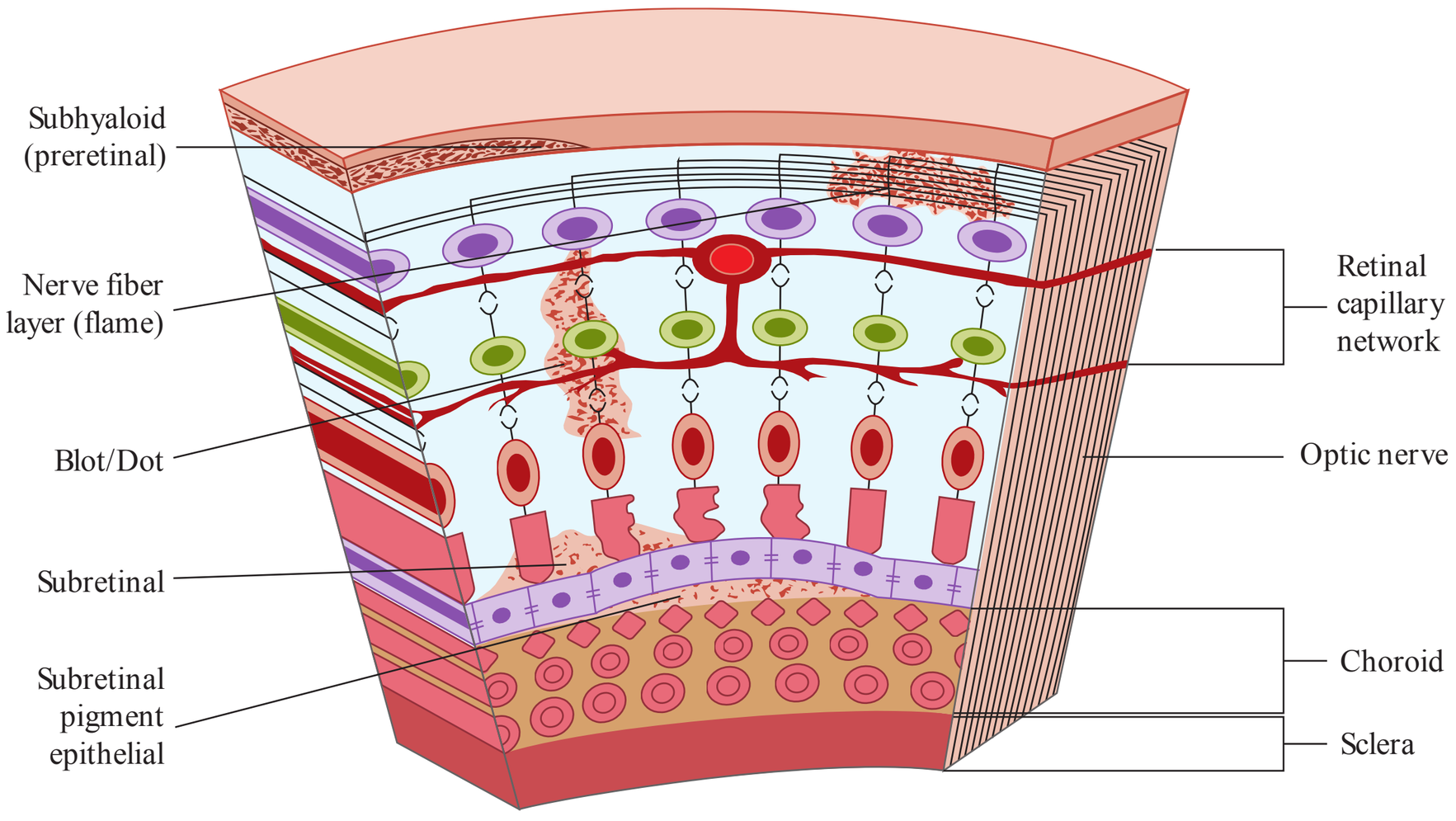


FIGURE 3.32 ■ **Retinal Hemorrhages—Anatomic Location.**

Chapter 4

OPHTHALMIC TRAUMA

Christopher S. Weaver
Kevin J. Knoop

Scan QR code or go to www.ematlas.com for video related to chapter



Corneal Perforation and Rust Ring. This patient was using a compressed air wire wheel brush when a wire came loose and lodged in the cornea. He was not wearing safety glasses. The wire was removed in the operating room where it was found to penetrate fully through the cornea and required one corneal suture. Interestingly, the rust ring had formed around the wire only about 3 hours after this injury occurred. (Used with permission from Brice Critser, CRA, The University of Iowa and EyeRounds.org.)

The authors acknowledge the special contributions of Dallas E. Peak and Carey D. Chisholm for contributions to prior editions.

CORNEAL ABRASION

Clinical Summary

Corneal abrasions present with acute onset of eye discomfort, tearing, and often a foreign-body sensation. A "ciliary flush" (conjunctival injection hugging the limbus) may be seen. Large abrasions or those in the central visual axis may affect visual acuity. Photophobia and headache from ciliary muscle spasm may be present. Associated findings or complications include traumatic iritis, hypopyon, or a corneal ulcer. Fluorescein examination, preferably with a slit lamp, reveals the defect.

Management and Disposition

Instillation of topical anesthetic drops facilitates examination while relieving pain and blepharospasm. Consider using a short-acting cycloplegic (eg, cyclopentolate 1%, homatropine 5%) to reduce pain from ciliary spasm in patients who complain of headache or photophobia. Consider oral opioid analgesics for pain control. Nonsteroidal anti-inflammatory drug (NSAID) eye drops (eg, diclofenac or ketorolac) are equally effective and avoid risks of sedation. Neither treatment with topical antibiotics, nor patching, nor tetanus prophylaxis for uncomplicated corneal abrasions has scientific validation. Follow-up is advised for any patient with complications, or who is still symptomatic after 24 hours.

Pearls

1. Mucus may simulate fluorescein uptake, but its position changes with blinking.
2. Multiple linear corneal abrasions, the "ice-rink sign," may result from an embedded foreign body (FB) adhered to the upper lid. Always evert the lid to evaluate this.
3. Whenever the mechanism includes grinding or striking metal, or high-velocity injuries from mowers or string trimmers, maintain a high index of suspicion for penetrating injury. Fluorescein streaming away from an "abrasion" (Seidel test) may be an indication of a corneal perforation.
4. Routine prophylactic treatment with topical antibiotics remains controversial. When used, inexpensive, broad-spectrum antibiotic drops (sulfacetamide sodium or trimethoprim/polymyxin B) allow clearer vision than lubricating ointments, which may feel better, but blur vision. Avoid topical neomycin antibiotics because of a high risk of irritant allergy symptoms in many people.
5. An "abrasion" in a contact lens wearer should alert one to suspect a corneal ulcer. Consult ophthalmology while the patient is in the emergency department.

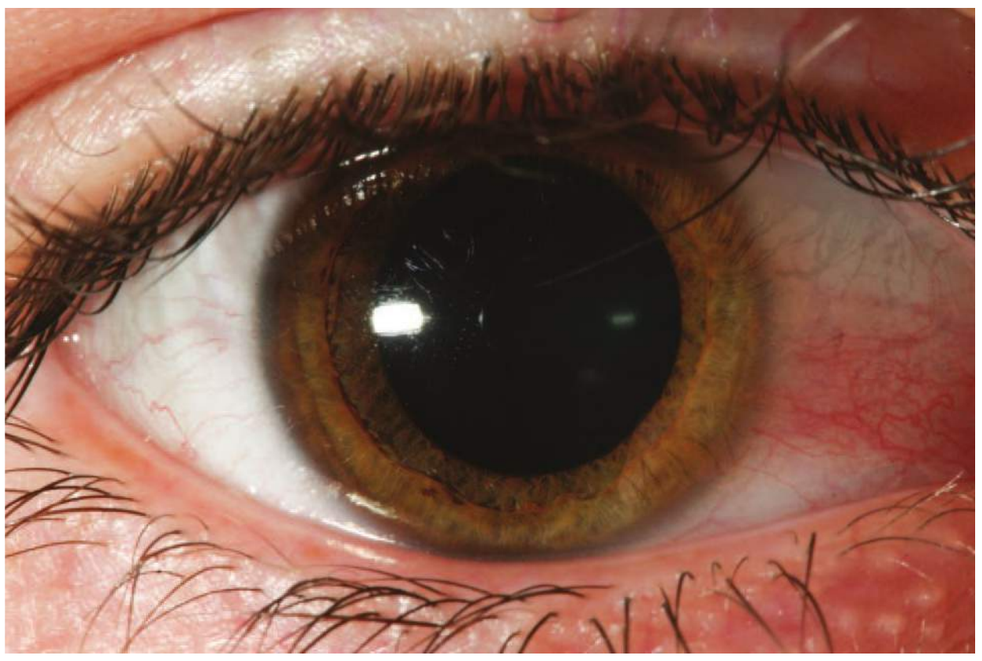


FIGURE 4.1 ■ **Corneal Abrasion.** A small abrasion is seen at the 3-o'clock position, just across the larger white reflection from the flash. Note the brisk localized conjunctival inflammatory reaction and ciliary flush. (Photo contributor: Kevin J. Knoop, MD, MS.)

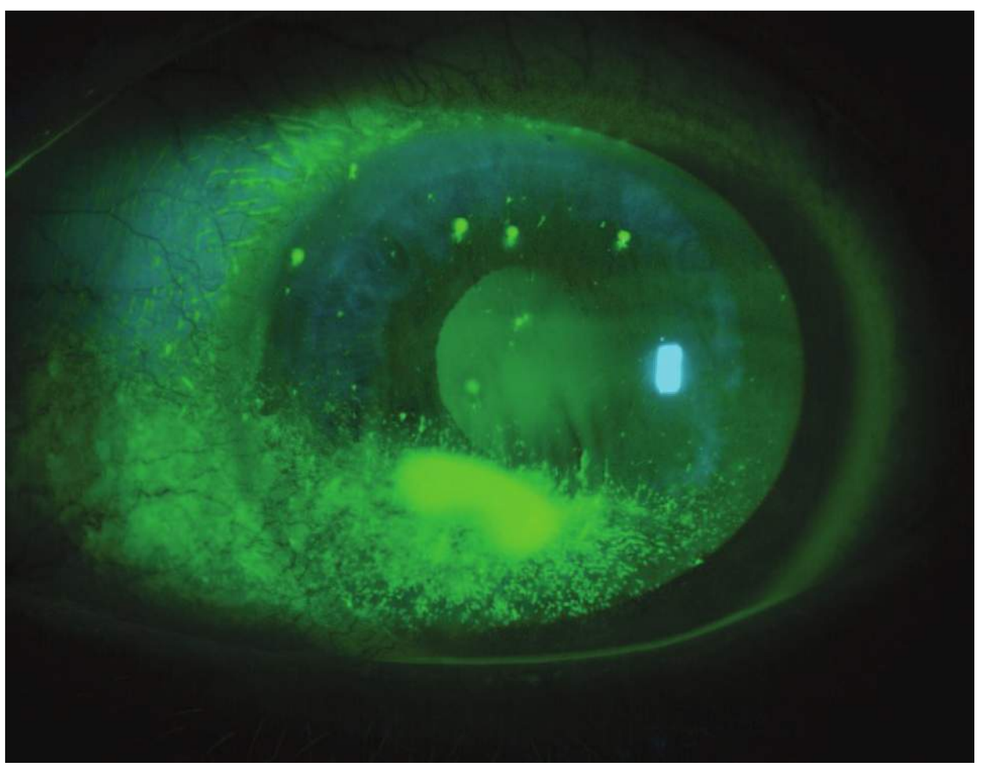


FIGURE 4.2 ■ **Corneal Abrasion.** Multiple punctate and one large traumatic abrasion are seen with fluorescein uptake. (Used with permission from Brice Critser, CRA, The University of Iowa and EyeRounds.org.)

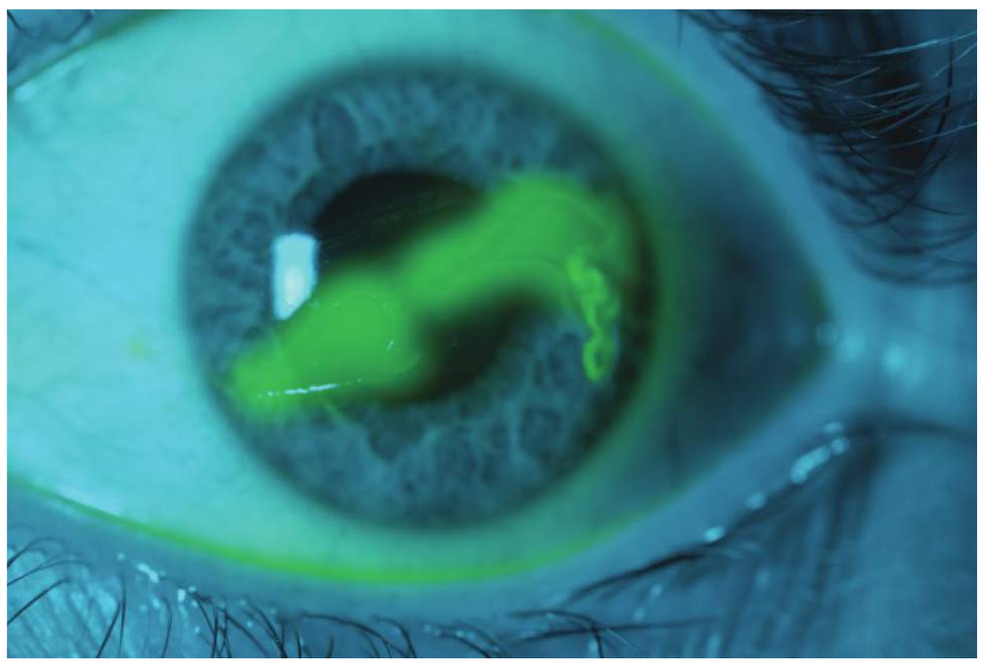


FIGURE 4.3 ■ **Corneal Abrasion.** Abrasions obscuring the visual axis benefit from close follow-up with an ophthalmologist to ensure adequate healing. (Photo contributor: Lawrence B. Stack, MD.)

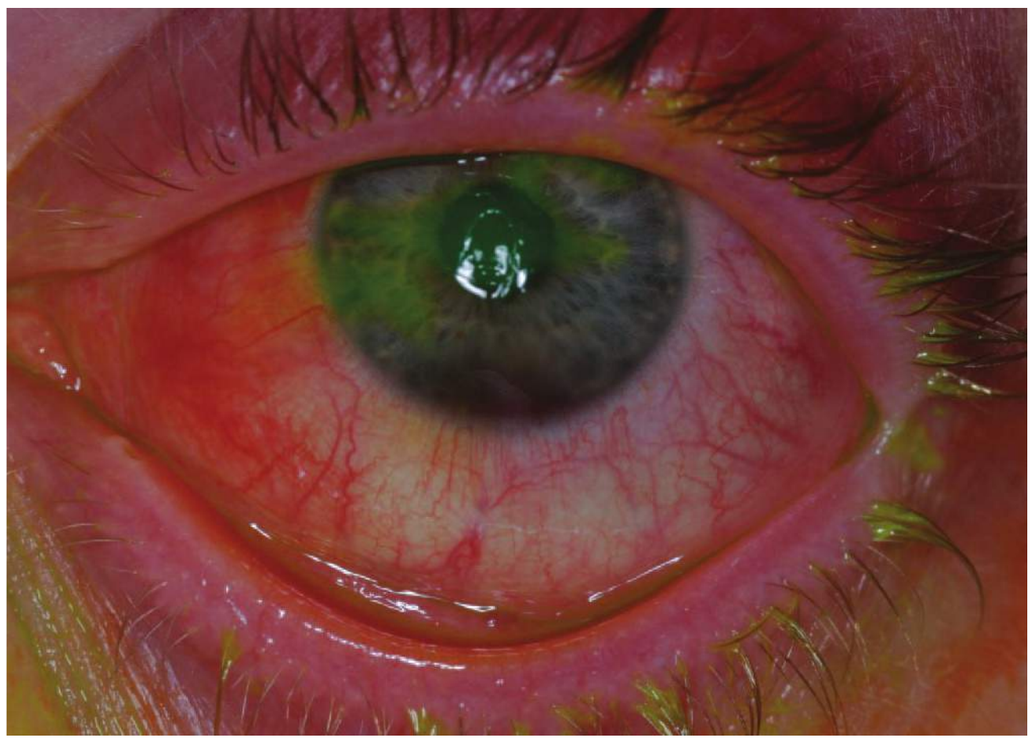


FIGURE 4.4 ■ **Corneal Abrasion.** This injury was due to a bungee cord impacting the eye. An irregular corneal light reflex indicates a disruption in the corneal epithelium (abrasion or perforation). A small hyphema is also seen. (Photo contributor: Kevin J. Knoop, MD, MS.)

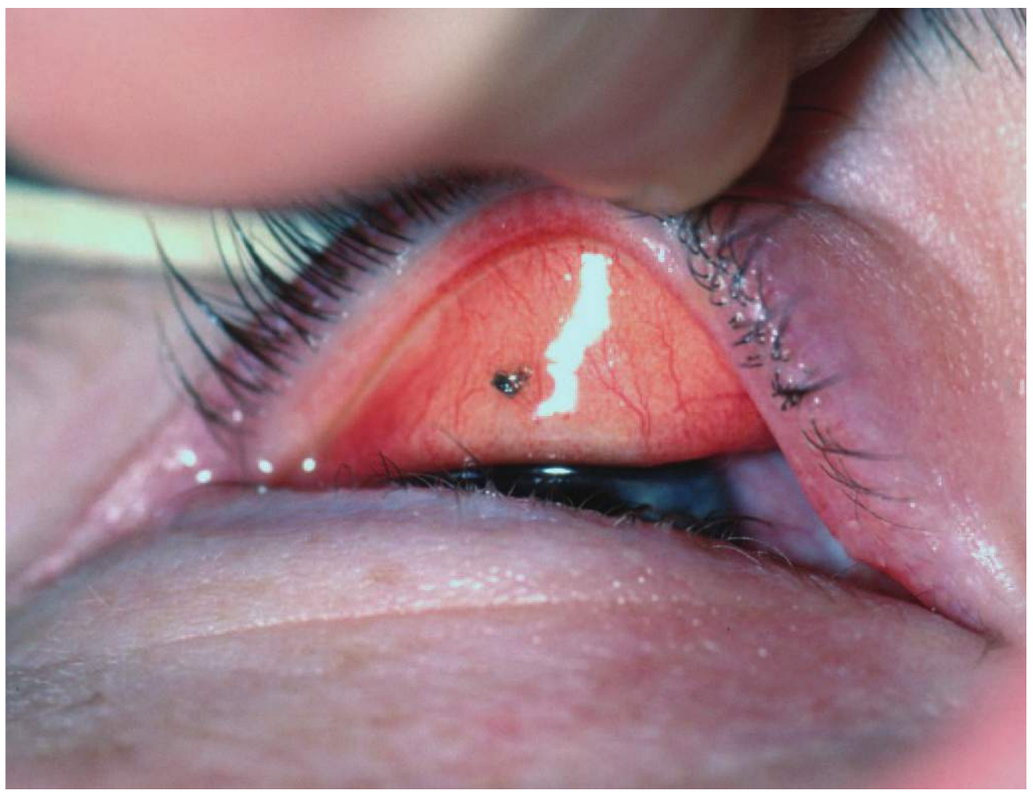


FIGURE 4.5 ■ **Foreign Body under the Upper Lid.** Lid eversion is an essential part of the eye examination to detect foreign bodies. (Photo contributor: Lawrence B. Stack, MD.)

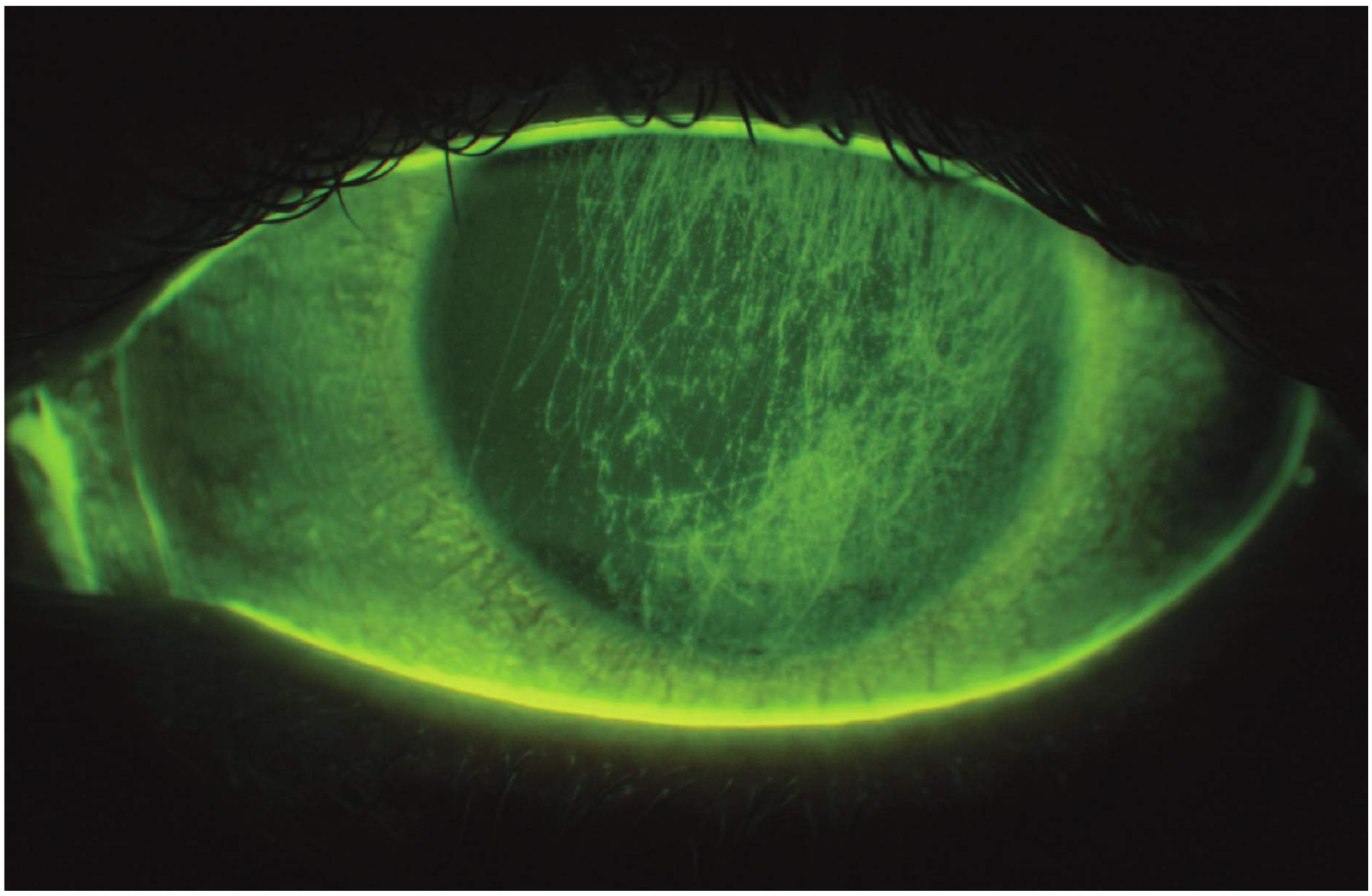


FIGURE 4.6 ■ **"Ice-Rink Sign."** Multiple linear abrasions caused by upper lid foreign body. (Photo contributor: Aaron Sobol, MD.)

SUBCONJUNCTIVAL HEMORRHAGE

Clinical Summary

A subconjunctival hemorrhage or hematoma occurs with often trivial events such as a cough, sneeze, Valsalva maneuver, or minor blunt trauma. The patient may present with some degree of duress secondary to the appearance of the bloody eye. The blood is usually bright red and appears flat. It is limited to the bulbar conjunctiva and stops abruptly at the limbus. This appearance is important to differentiate the lesion from bloody chemosis, which can occur with scleral rupture or nontraumatic conditions. Aside from appearance, this condition does not cause the patient any pain or diminution in visual acuity.



FIGURE 4.7 ■ **Subconjunctival Hemorrhage.** Subconjunctival hemorrhage from blunt trauma. The flat appearance of the hemorrhage suggests its benign nature. (Photo contributor: Dallas E. Peak, MD.)

Management and Disposition

No treatment is required. Instruct the patient to expect the blood to resorb in 2 to 3 weeks.

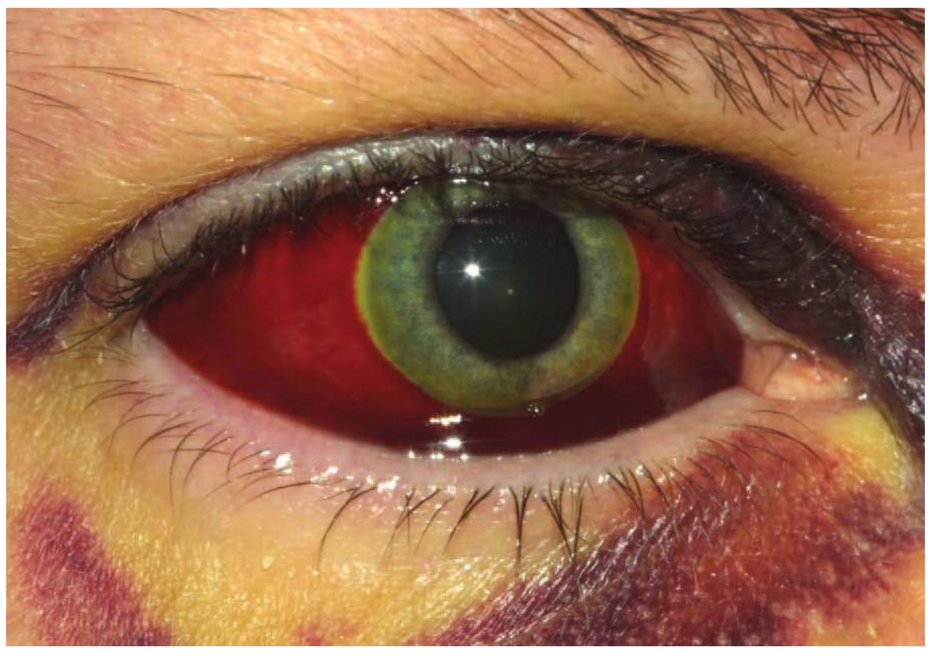


FIGURE 4.8 ■ **Subconjunctival Hemorrhage.** Subconjunctival hemorrhage that completely surrounds the eye, but stops abruptly at the limbus in a patient with blunt trauma. (Used with permission from Brice Critser, CRA, The University of Iowa and EyeRounds.org.)

Pearls

1. Subconjunctival hematoma differs from bloody chemosis with the flat appearance of the conjunctival membranes.
2. A subconjunctival hematoma involving the extreme lateral globe after blunt trauma is very suspicious for zygomatic arch fracture.
3. Evaluate patients with nontraumatic bloody chemosis for an underlying metabolic (coagulopathy) or structural (cavernous sinus thrombosis) disorder.

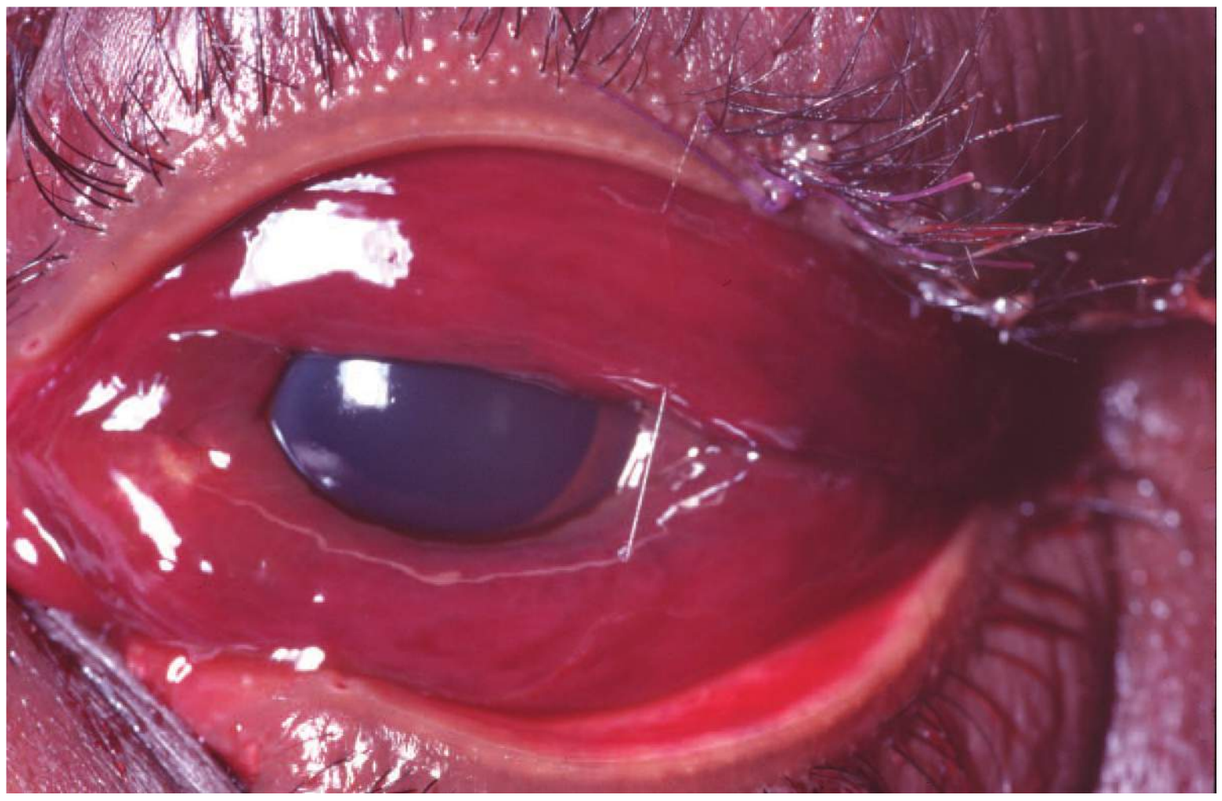


FIGURE 4.9 ■ **Traumatic Bloody Chemosis.** Suspicious for globe penetration, open globe was ruled out in this patient with CT. Dilated pupil is a result of pharmacologic agent. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Patients typically report getting something in the eye or complain of FB sensation. FBs that overlie the cornea may affect vision. Tearing, conjunctival injection, headache, and photophobia may also be present. The most important consideration is the possibility of a penetrating globe injury. One must elicit a meticulous history about the mechanism of injury.

Management and Disposition

Topical anesthetic drops (eg, 0.5% proparacaine or tetracaine) facilitate examination and removal. If superficial, removal with saline flush may be attempted before using a sterile eye spud or small (25-gauge) needle. Consider topical antibiotic drops or ointment for the residual corneal abrasion. Tetanus prophylaxis is indicated. A "short-acting" cycloplegic (eg, cyclopentolate 1% or homatropine 5%) may benefit patients with headache or photophobia. FB or "rust ring" removal should be conducted using slit-lamp microscopy, only by a physician skilled in rust ring removal due to the risk of corneal perforation or scarring.

Pearls

1. Always evert the upper lid and search carefully for an FB. An FB adherent to the upper lid abrades the cornea, producing the "ice-rink" sign, caused from multiple linear abrasions.
2. Vigorous attempts to remove the entire rust ring are not warranted. This may await emergency department or ophthalmology follow-up in 24 hours.
3. Use of cotton-tipped applicators to attempt FB removal should be discouraged (large surface area and potential to cause a larger corneal defect).

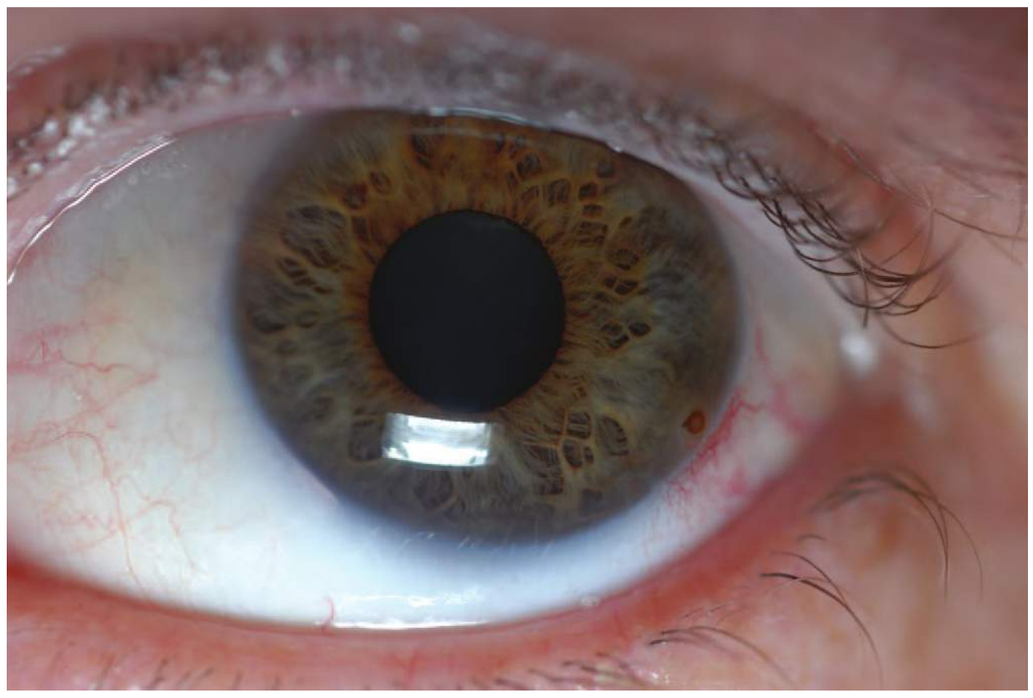


FIGURE 4.10 ■ **"Rust Ring."** A rust ring has formed from a foreign body (likely metallic) in this patient. A burr drill can be used for attempted removal, which, if unsuccessful, can be reattempted in 24 hours. (Photo contributor: R. Jason Thurman, MD.)

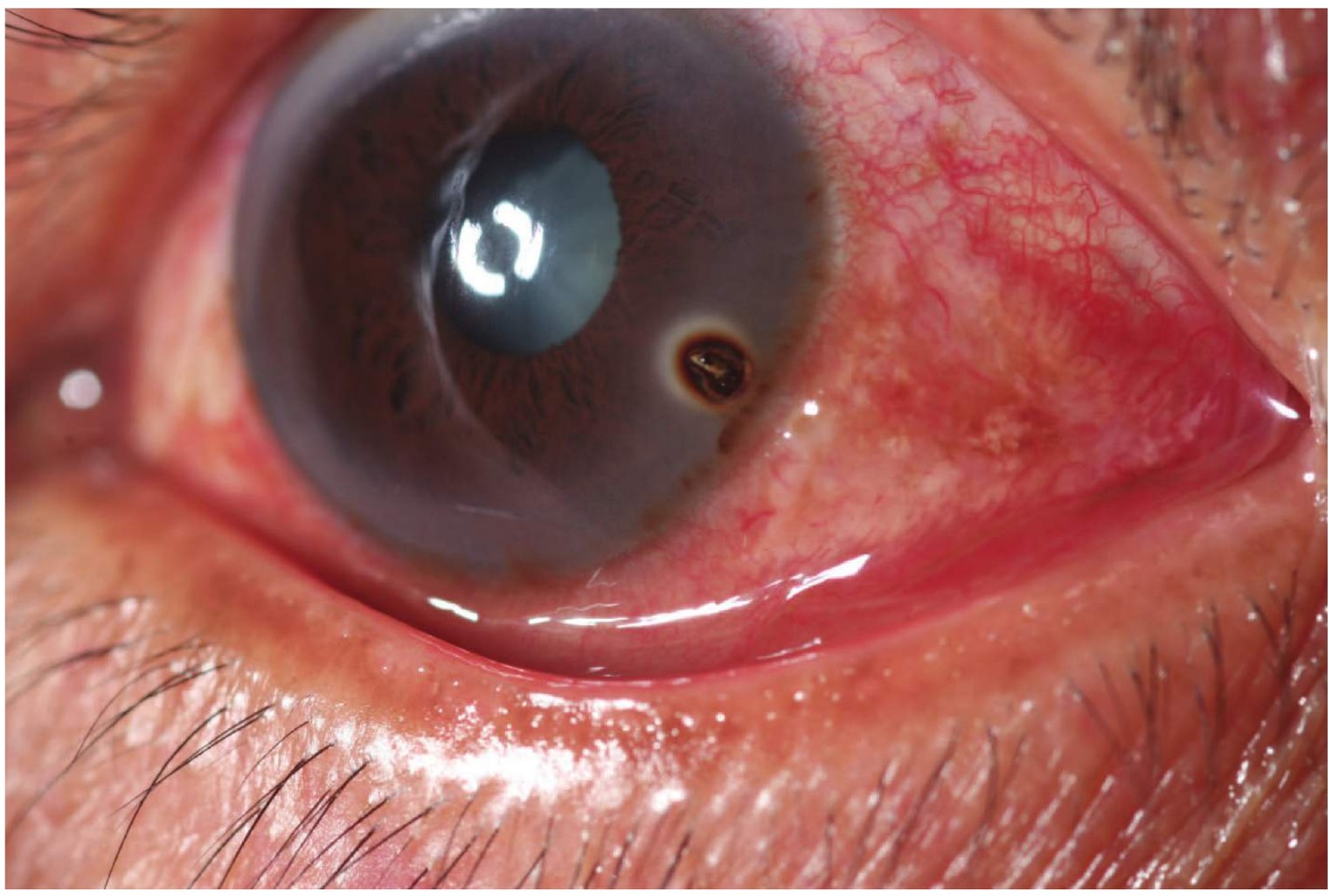


FIGURE 4.11 ■ **Foreign Body on the Cornea.** A small foreign body is lodged at 4-o'clock position on the cornea. (Photo contributor: Kevin J. Knoop, MD, MS.)

HYPHEMA

Clinical Summary

Injury to the anterior chamber that disrupts the vasculature supporting the iris or ciliary body results in a hyphema. The blood tends to layer with time, and if left undisturbed, gravity will form a visible meniscus. Symptoms can include pain, photophobia, and possibly blurred vision secondary to obstructing blood cells. Nausea and vomiting may signal a rise in intraocular pressure (glaucoma) caused by blood cells clogging the trabecular meshwork.

Management and Disposition

Prevention of further hemorrhage is the foremost treatment goal. Most rebleeding occurs within the first 72 hours and is usually more extensive than the initial event. Keep the patient at rest in the supine position with the head elevated slightly. Use a hard eye shield to prevent further trauma from manipulation. Avoid oral or parenteral pain medication and sedatives with antiplatelet activity such as NSAIDs. Use antiemetics if the patient has nausea. Further treatment at the discretion of specialty consultants may include topical and oral steroids, antifibrinolytics such as aminocaproic acid, or surgery. Measure intraocular pressure (IOP) unless there is a suspicion of penetrating injury to the globe. Treat elevated IOP with appropriate agents, including topical β-blockers, pilocarpine, and, if needed, osmotic agents (mannitol, sorbitol) and acetazolamide. Ophthalmologic consultation is warranted to determine local admission practices.

Pearls

1. Instruct patients specifically not to read or watch television, as these activities result in greater than usual ocular activity.
2. Rebleeding may occur in 10% to 20% of patients, most commonly in the first 2 to 5 days when the blood clots start to retract.
3. An "eight-ball" or total hyphema often leads to elevated IOP and corneal bloodstaining, and typically requires surgical evacuation.

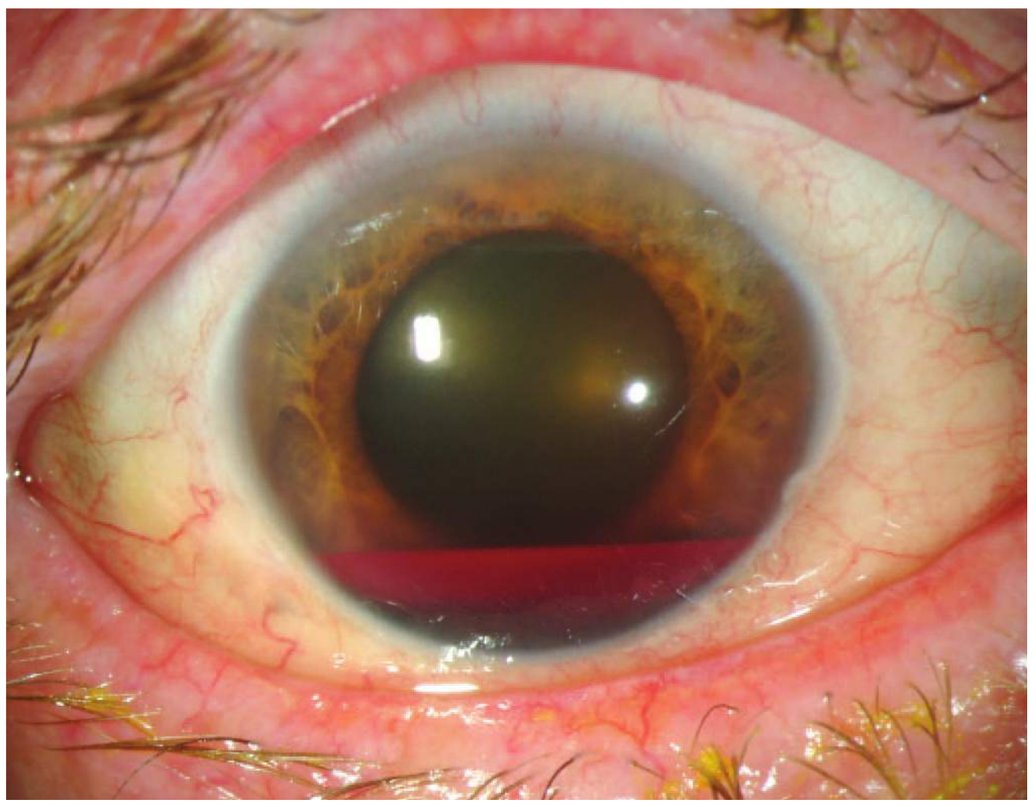


FIGURE 4.13. ■ **Hyphema.** This hyphema has completely layered out in the anterior chamber. (Used with permission from Brice Critser, CRA, The University of Iowa and EyeRounds.org.)

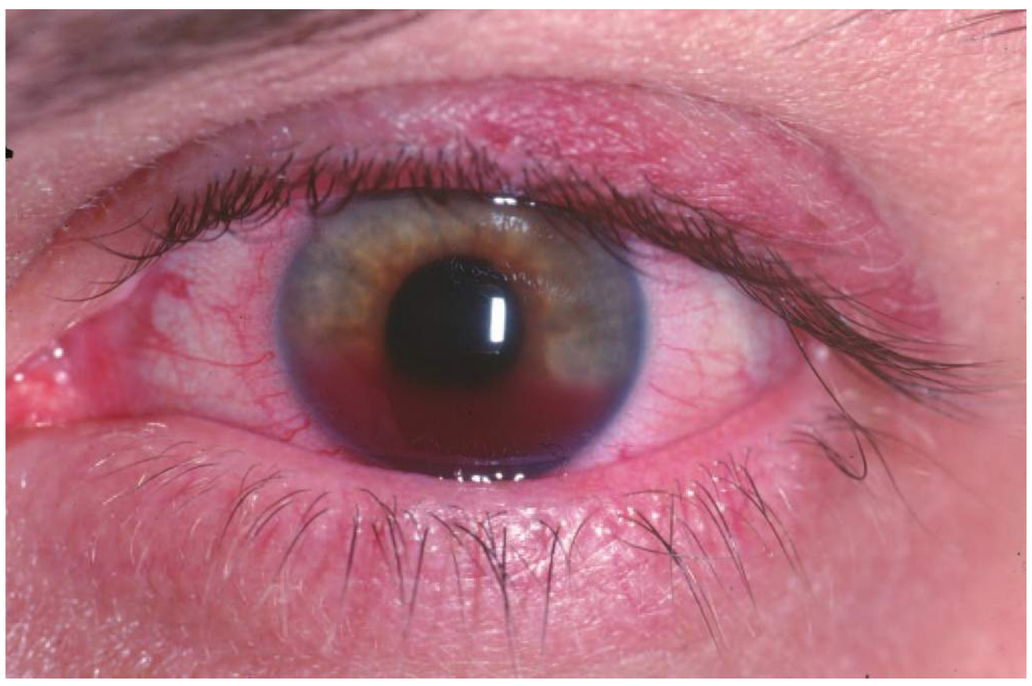


FIGURE 4.12 ■ **Hyphema.** This hyphema is just beginning to layer out, reflecting its acute nature. (Photo contributor: Lawrence B. Stack, MD.)

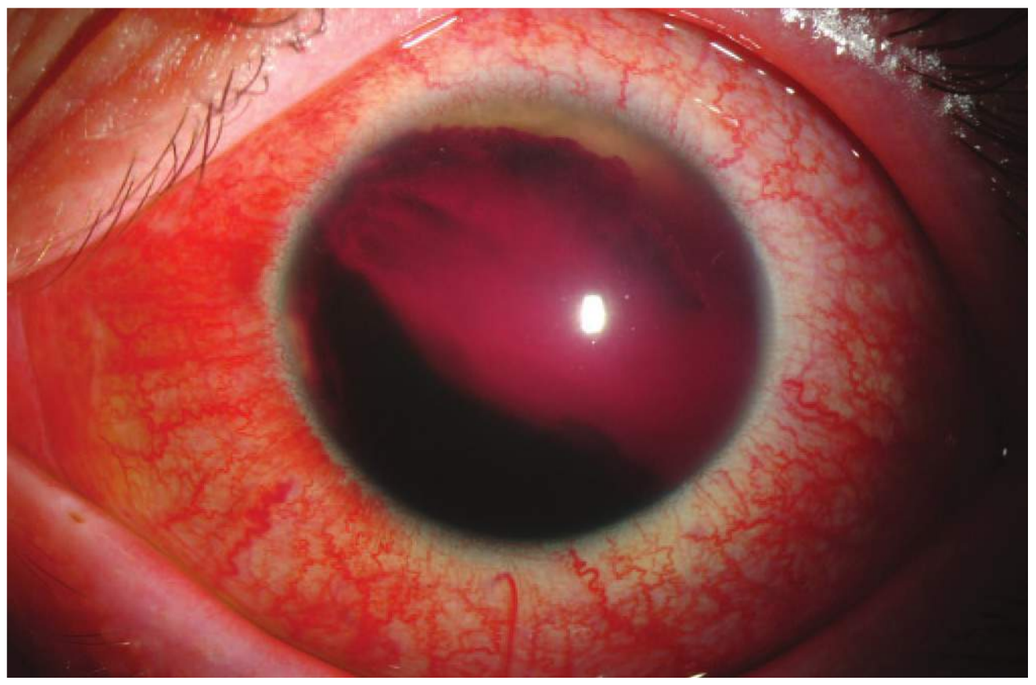


FIGURE 4.14 ■ **"Eight-Ball" Hyphema.** This hyphema completely fills the anterior chamber. (Used with permission from Brice Critser, CRA, The University of Iowa and EyeRounds.org.)

4. Patients with sickle cell and other hemoglobinopathies are at risk for sickling of blood inside the anterior chamber. This can cause a rise in IOP caused by obstruction of the trabecular meshwork even if only a small hyphema is present.
5. An abnormally low IOP should prompt consideration for presence of penetrating globe injury.
6. Evaluate supine trauma patients for slight differences in iris color to determine the presence of a hyphema.

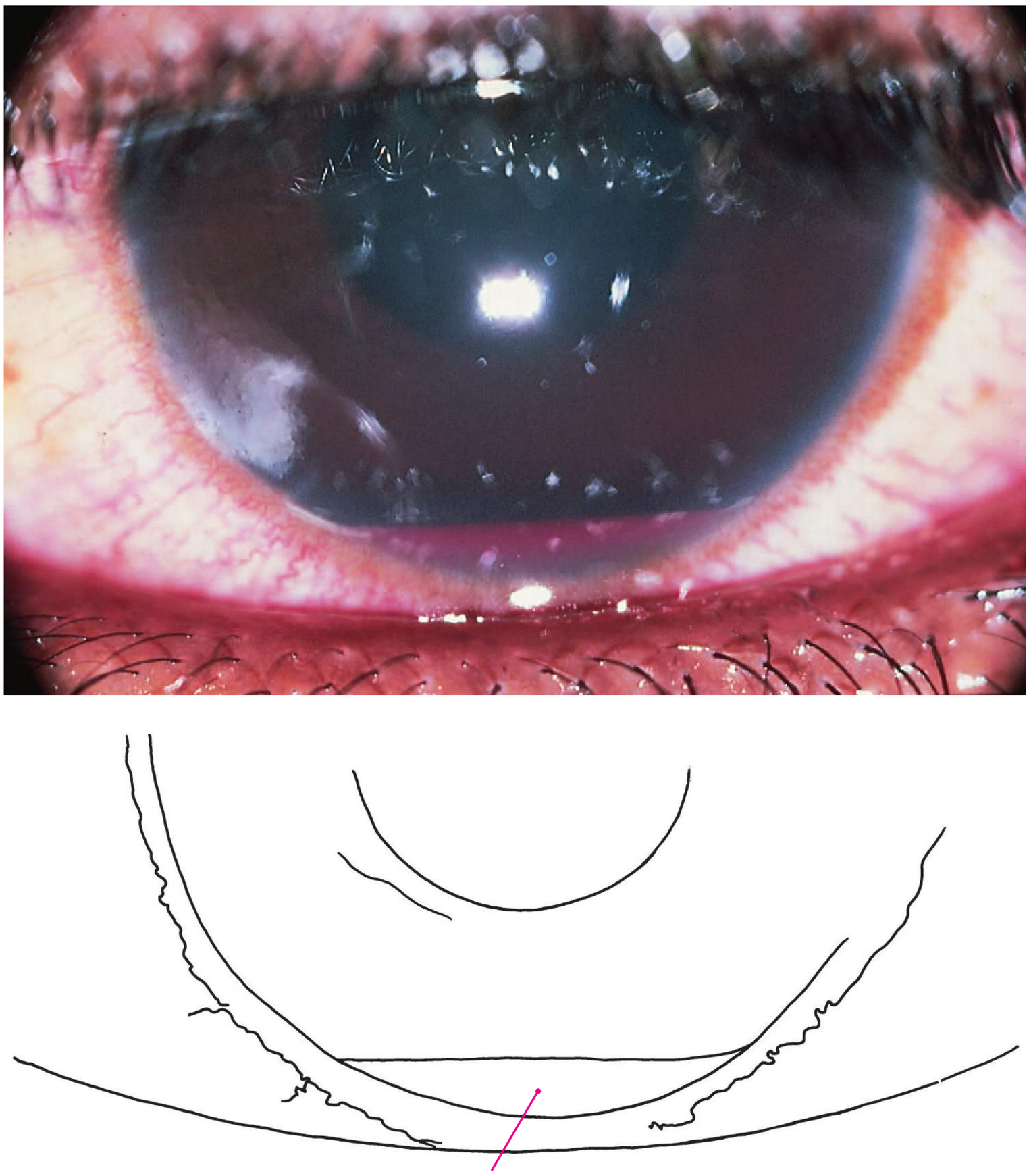


FIGURE 4.15 ■ **Hyphema.** A small hyphema (about 5%) in a patient with sickle cell disease. (Photo contributor: Dallas E. Peak, MD.)

INTRAOCULAR FOREIGN BODY

Clinical Summary

The most important consideration with any eye injury is the possibility of a penetrating globe injury with residual intraocular foreign body (IO FB). Patients may report FB sensation, but subtle presentations occur. A meticulous history about the mechanism of injury (grinding or metal on metal) must be elicited.

Management and Disposition

For suspected subtle injury, a careful examination is required. Bedside ultrasound can be a useful adjunct and allows rapid identification of an IO FB. Care must be taken to avoid any pressure on the globe. A slit-lamp examination with Seidel test (copious amounts of fluorescein instilled and observed for streaming away from the site of perforation) may reveal a microperforation.

Pearls

1. Always maintain a high index of suspicion for penetrating globe injury. Be particularly wary in mechanisms involving use of "metal on metal" such as grinding or hammering. A positive Seidel test demonstrates corneal microperforation.
2. If ocular penetration is suspected, a diligent search for a retained FB is indicated, beginning carefully with bedside ultrasound using a high-frequency transducer. Computed tomography (CT) is the diagnostic study of choice (avoid magnetic resonance imaging [MRI]) with indeterminate results or when confirmation is desired.

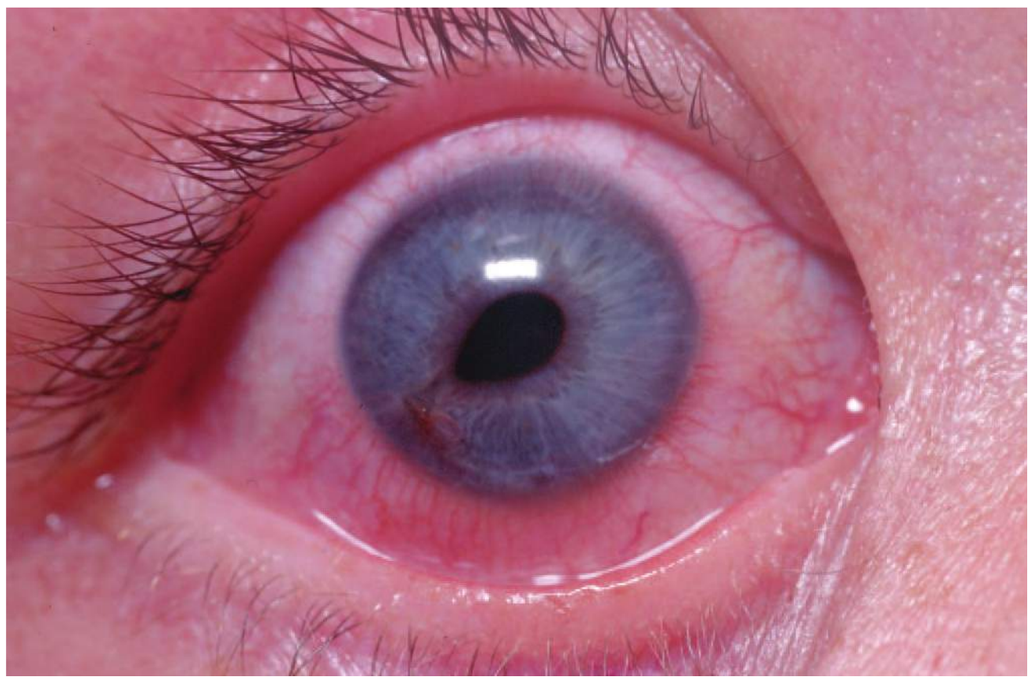


FIGURE 4.16 ■ **Anterior Chamber Foreign Body.** A shard from a nail is seen embedded in the anterior chamber. A "teardrop" pupil is present, indicating perforation. (Photo contributor: Lawrence B. Stack, MD.)

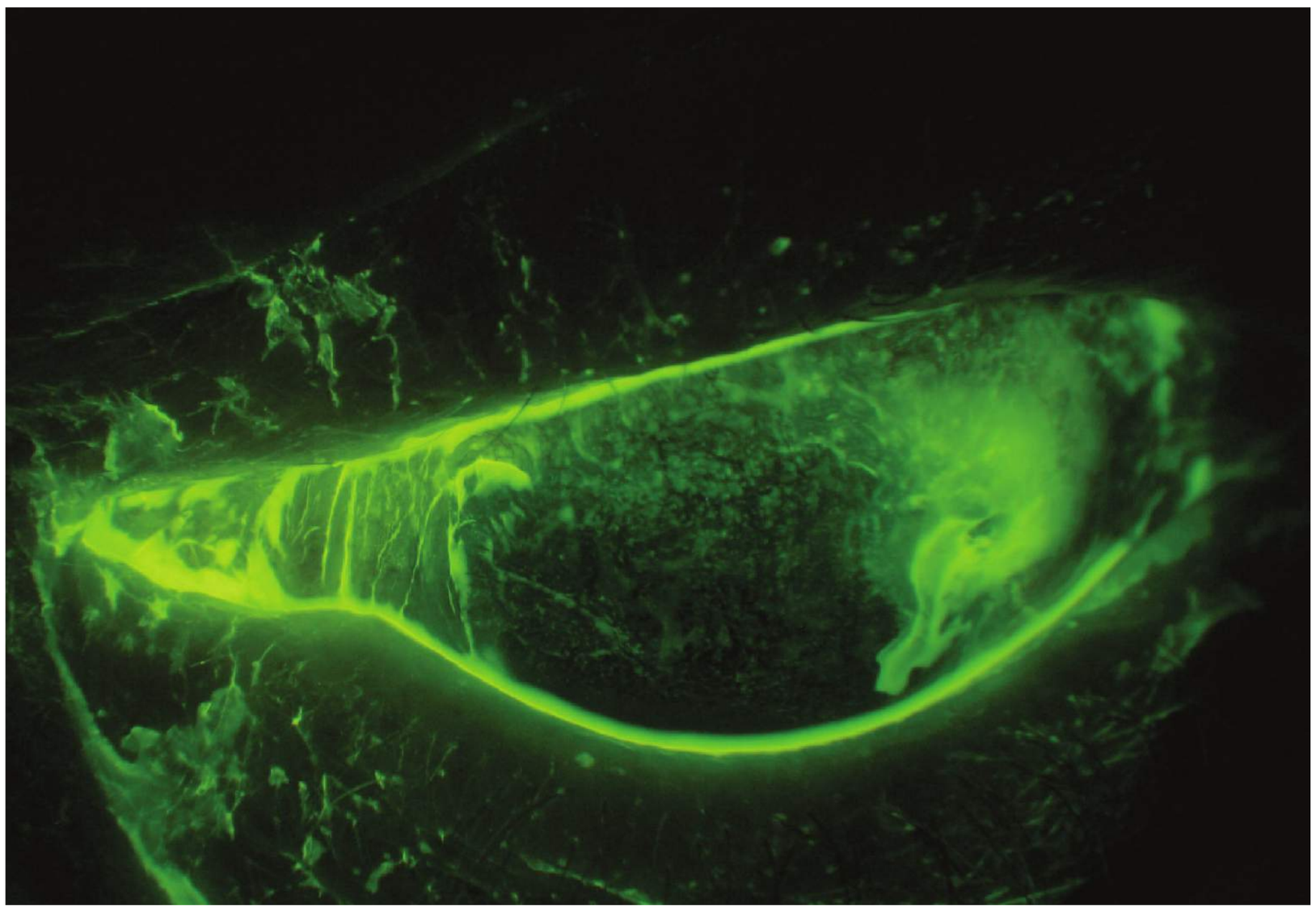


FIGURE 4.17 ■ **Seidel Test.** A positive Seidel test shows aqueous leaking through a corneal perforation while being observed with the slit lamp. (Photo contributor: Aaron Sobol, MD.)

Clinical Summary

Traumatic iridodialysis is the result of an injury, typically blunt trauma that pulls the iris away from the ciliary body. The resulting deformity appears as a lens-shaped defect at the outer margin of the iris. Patients may present complaining of a "second pupil." As the iris pulls away from the ciliary body, a small amount of bleeding may result. Look closely for associated traumatic hyphema.

Consider etiologies such as penetrating injury to the globe, scleral rupture, IO FB, and lens dislocation causing billowing of the iris.

Management and Disposition

A remote traumatic iridodialysis requires no specific treatment in the emergency department. Recent history of ocular trauma should prompt a diligent slit-lamp examination for associated hyphema or lens dislocation. If hyphema is present, treat it as discussed (see "Hyphema"). Refer cases of iridodialysis for specialty consultation to exclude other injuries; if the defect is large enough to result in monocular diplopia, surgical repair may be necessary.

Pearls

1. The examination should carefully exclude posterior chamber pathology and hyphema. Consider bedside ultrasonography to rule out posterior pole injuries (retinal detachment, vitreous hemorrhage, lens dislocation, or FB).
2. Carefully review the history to exclude penetrating trauma. If the history is unclear, CT scan may be used to exclude the presence of IO FB.
3. A careful examination includes searching for associated lens dislocation.

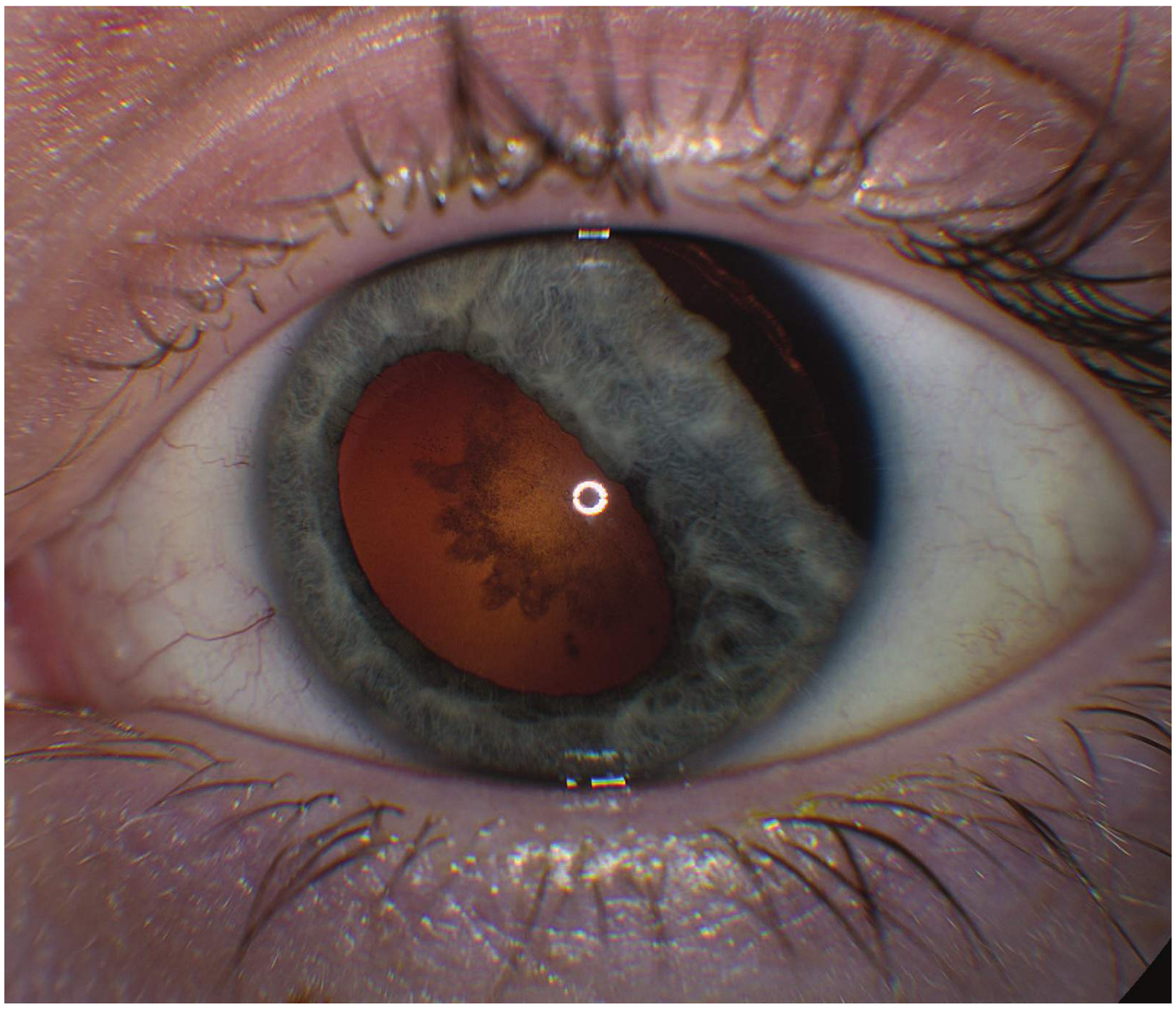


FIGURE 4.18 ■ **Traumatic Iridodialysis.** The iris has pulled away from the ciliary body as a result of blunt trauma. A traumatic cataract is also seen. The rosette pattern is classically seen after contusion injuries. It is due to separation of lens fibers around lens sutures. (Used with permission from Brice Critser, CRA, The University of Iowa and EyeRounds.org)

LENS DISLOCATION

Clinical Summary

Lens dislocation may result from blunt trauma to the globe. As the anterior surface of the eye is struck, there is compression in the anteroposterior dimension with resultant stretching of the globe along its equator in the medial-lateral plane. As this occurs, it stretches the zonule fibers, which suspend the lens in place, and they along with the lens capsule may become disrupted. The patient may experience symptoms of monocular diplopia or gross blurring of images, depending on the severity of the injury. Occasionally there can be dramatic visual fluctuations caused by the lens changing position with resultant phakic and aphakic vision. There is generally a lack of pain except if secondary angle closure glaucoma occurs from the lens causing pupillary block. On slit-lamp examination, the displaced crystalline lens appears as a crescent shape along its edge against a backdrop of the red reflex from the fundus. The edge of the subluxed lens may be visible only with pupillary dilatation. Use caution in dilating the pupil, as this may cause the lens to sublux into the anterior chamber, which occurs if all the zonule fibers are torn. Chronically, the lens may lodge in either the anterior chamber or the vitreous. Marfan syndrome, tertiary syphilis, and homocystinuria may be present and should be considered in patients presenting with lens dislocation.

Management and Disposition

A subluxed lens does not always require surgery; partial subluxations may require only a change in refraction. Surgery is required if anterior dislocation of the lens results in papillary block and angle closure glaucoma results.

Pearls

1. Patients may experience lens dislocation with seemingly trivial trauma if they have an underlying coloboma of the lens, Marfan syndrome, homocystinuria, or syphilis.
2. Iridodonesis is a trembling movement of the iris noted after rapid eye movements and is a sign of occult posterior lens dislocation.
3. Phacodonesis is a tremulousness of the lens itself caused by disruption of the zonule fibers.
4. Lens dislocation or subluxation is commonly associated with traumatic cataract formation.
5. Other associated injuries include hyphema, vitreous hemorrhage, and globe rupture.

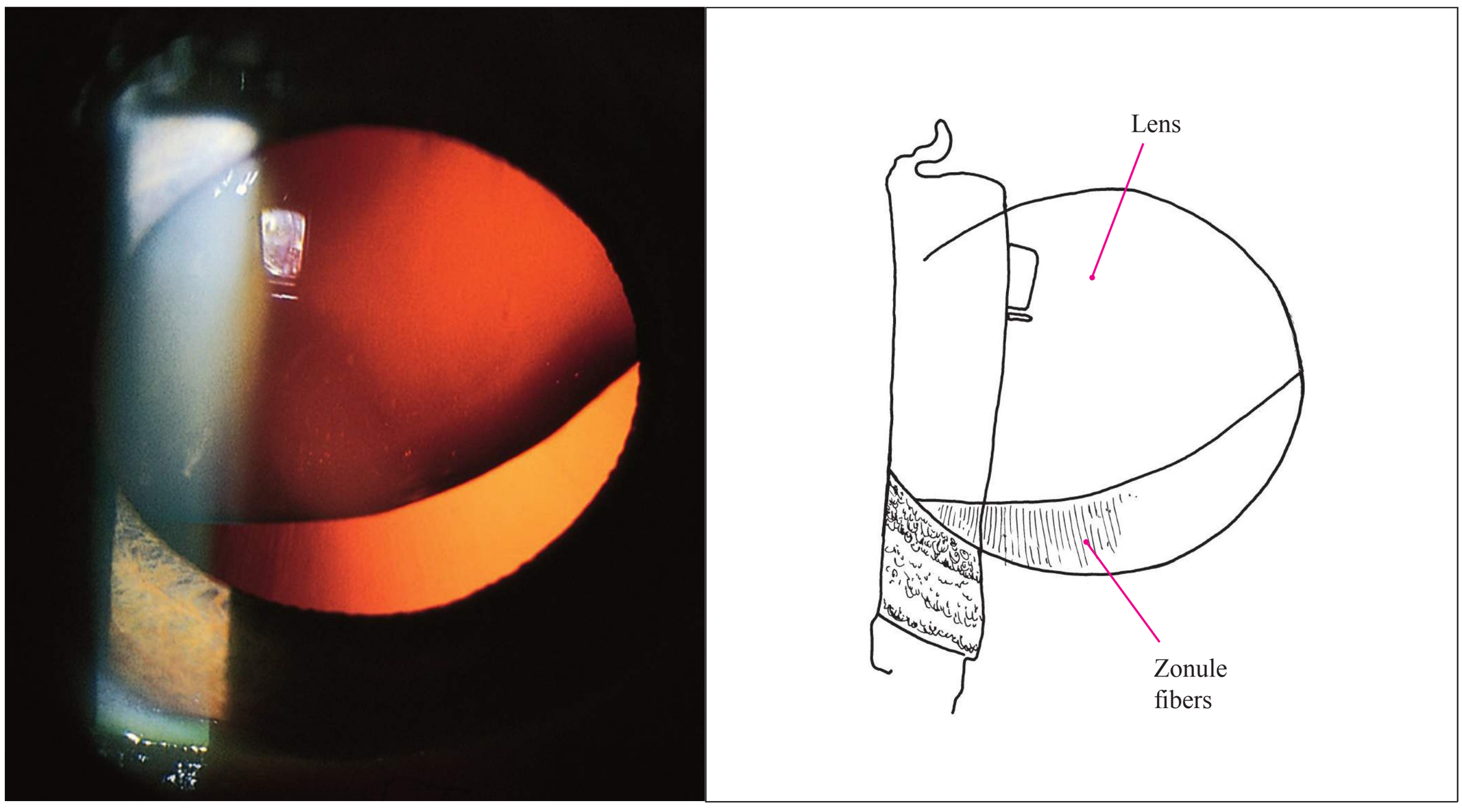


FIGURE 4.19 ■ **Lens Dislocation.** Lens dislocation revealed during slit-lamp examination. Note the zonule fibers, which normally hold the lens in place. (Photo contributor: Department of Ophthalmology, Naval Medical Center, Portsmouth, VA.)

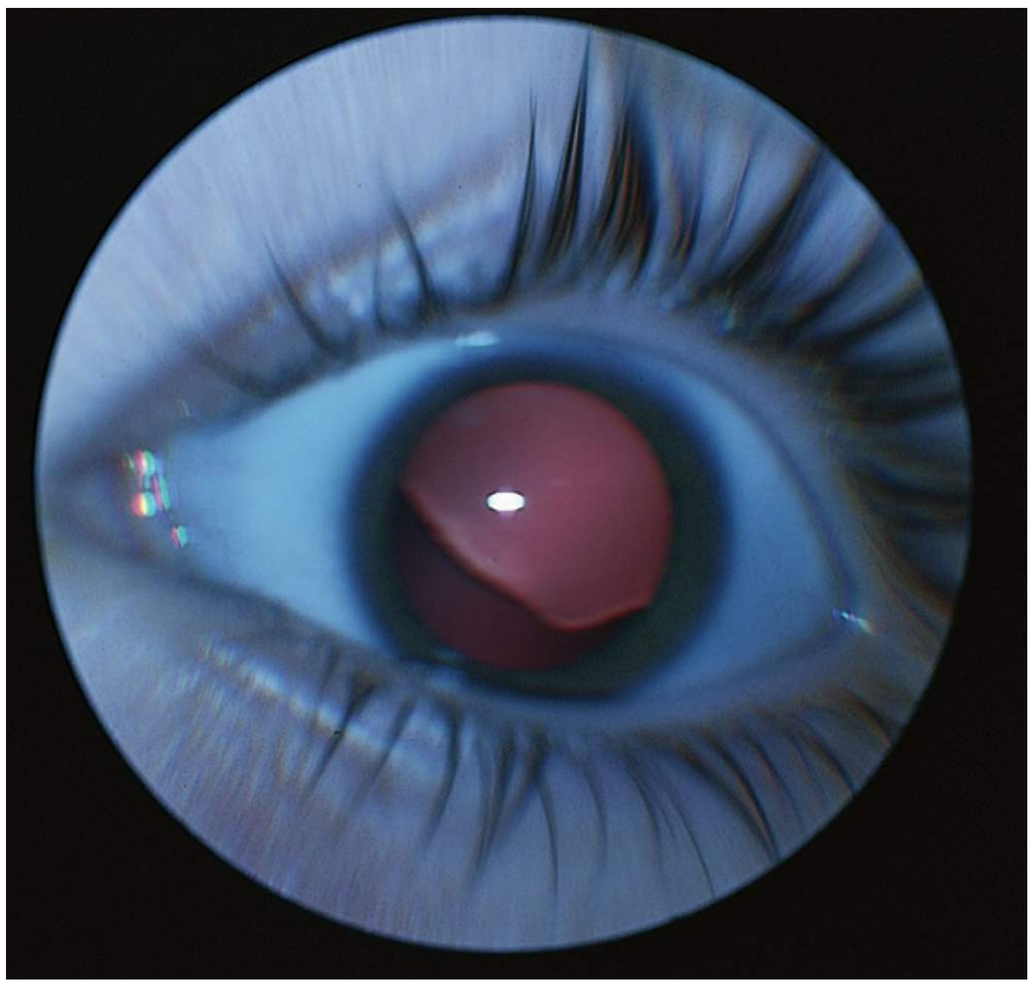


FIGURE 4.20 ■ **Lens Dislocation.** The edge of this dislocated lens is visible with the pupil dilated as an altered red reflex. (Photo contributor: Department of Ophthalmology, Naval Medical Center, Portsmouth, VA.)

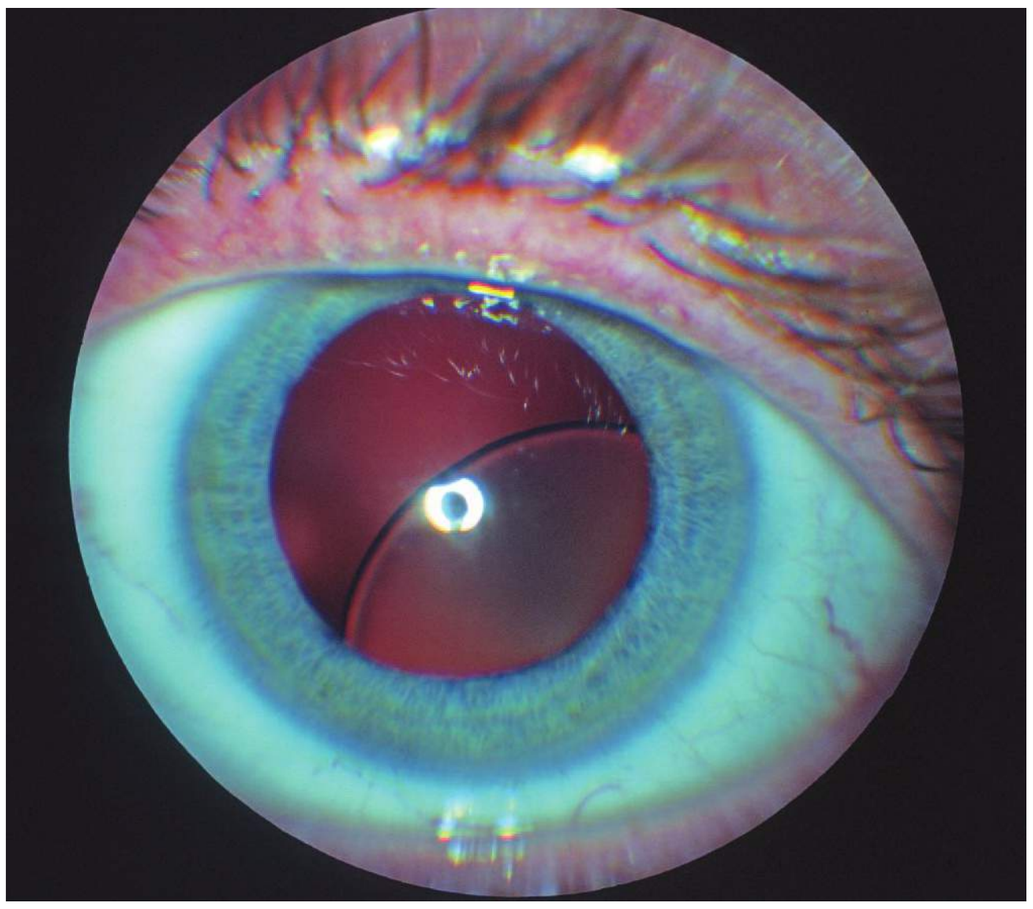


FIGURE 4.21 ■ **Lens Dislocation.** The crescentic edge of this dislocated lens is visible. Red reflex is abnormal. (Photo contributor: Thomas Egnatz, CRA.)

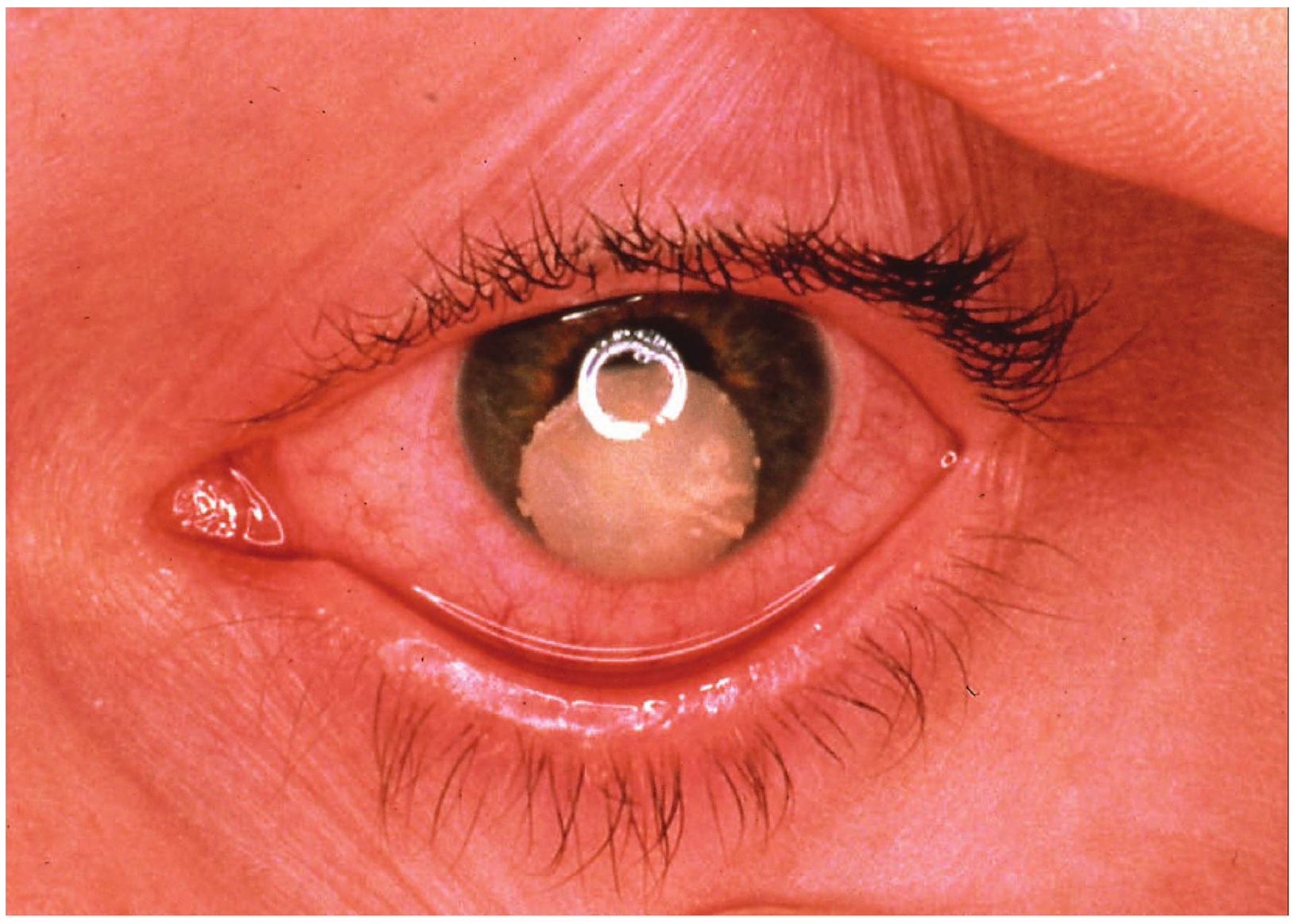


FIGURE 4.22 **Lens Dislocation.** Complete lens dislocation prolapsed into the anterior chamber. (Photo contributor: David Effron, MD.)

PENETRATING GLOBE INJURY

Clinical Summary

Penetrating globe injuries can be subtle and easily overlooked. All are serious injuries. Signs to look for are loss of anterior chamber depth caused by leakage of aqueous humor, a teardrop-shaped ("peaked") pupil, or prolapse of choroid through the wound.

Management and Disposition

All open globe injuries require urgent specialty consultation. Immediately place a rigid eye shield over the affected eye. If a specifically designed rigid shield (eg, Fox metal eye shield) is unavailable, the bottom of a Styrofoam cup may be used. Do not use a pressure patch. Tonometry to measure pressures is strictly contraindicated. It is imperative to avoid inadvertent pressure on the globe with resulting irreversible expulsion of choroid through the wound. Intravenous antibiotics appropriate to cover gram-positive organisms are indicated. Consider adding gram-negative coverage for injuries that involve organic foreign bodies. Sedation and aggressive pain management are crucial to prevent or decrease expulsion of intraocular contents caused by crying, activity, or vomiting. Antiemetics should be given as prophylaxis, and update tetanus status. Other significant blunt trauma may accompany penetrating globe. Always consider the possibility that foreign bodies may have penetrated through the globe into the posterior orbit and possibly extend into the cranial vault.

Pearls

1. Stabilize protruding foreign bodies without manipulation until definitively treated in the operating room.
2. Control of pain, activity, and nausea may be sight saving and requires proactive use of appropriate medications.
3. Use of lid retractors is preferred to open the eyelids of trauma victims with blepharospasm or massive swelling when such examination is indicated. Attempts to use fingers can inadvertently increase the pressure on the globe.
4. Emergency department ultrasound with a high-frequency probe using no pressure technique can be a useful adjunct to detect lens displacement, retinal detachment, and intraocular FB at the bedside. It is not useful to diagnose commotio retina, globe rupture, or retrobulbar hematoma.
5. Penetrating globe injuries are a relative contraindication to the sole use of depolarizing neuromuscular blockade (eg, succinylcholine). Consider a "de-fasciculating" dose of a nondepolarizing agent as a pretreatment to avoid increasing intraocular pressure.
6. CT scanning is the most readily available and sensitive modality to evaluate penetrating injuries. Some nonmetallic objects such as wood, glass, or plastic may be difficult to visualize with CT. MRI is preferable in these cases and is excellent at demonstrating associated soft-tissue injuries to the globe and orbit. MRI is contraindicated when a metallic FB is suspected.

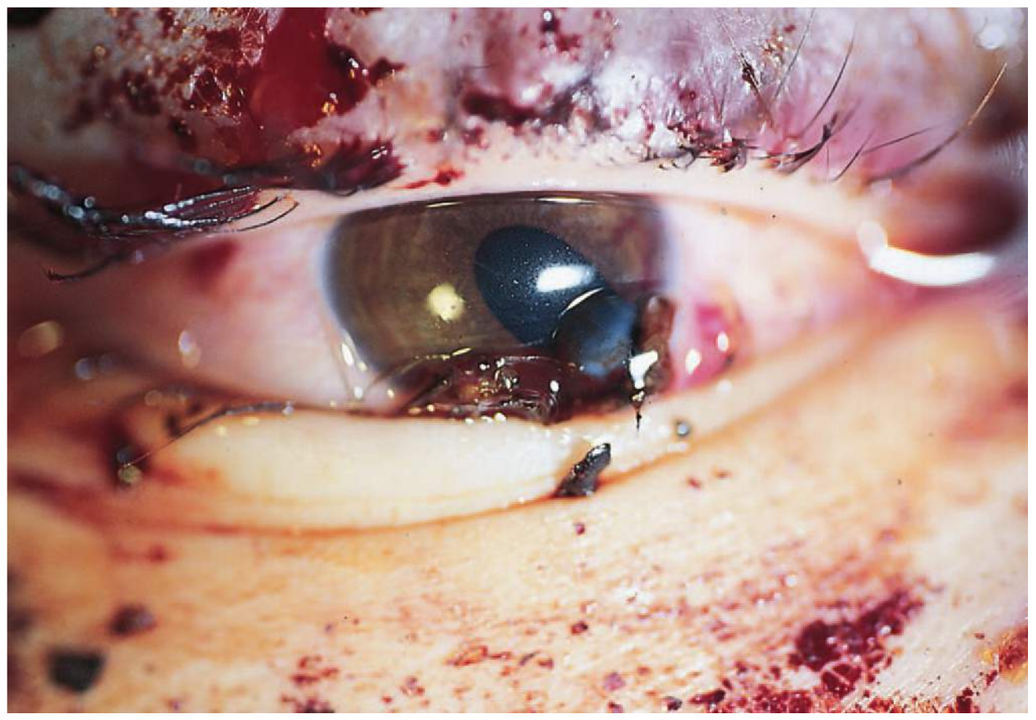


FIGURE 4.23 ■ **Open Globe.** This injury is not subtle; extruded ocular contents (vitreous) can be seen; a teardrop pupil is also present. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 4.24 ■ **Open Globe.** An obvious penetrating globe injury is seen once lids are carefully retracted. The lower lid laceration makes this a high risk for globe penetration. Intraocular foreign body should be suspected. (Photo contributor: David Effron, MD.)





FIGURE 4.25 ■ **Penetrating Globe.** (**A**) This penetrating injury shows an "eight-ball" hyphema. (**B**) Bedside ultrasound shows both intraocular and anterior chamber hemorrhage. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 4.26 ■ **Orbital Trauma.** A stick is impaled 7 cm into the medial aspect of the eye. Globe penetration was ruled out in the operating room. (Photo contributor: Elena Geraymovych, MD.)



FIGURE 4.27 ■ **Eyelid Retractors.** Retractors fashioned from paper clips can safely be used when standard retractors are not available. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 4.28 ■ **Protective Metal (Fox) Shield.** A protective shield is used in the setting of a suspected or confirmed perforating injury. (Photo contributor: Kevin J. Knoop, MD, MS.)

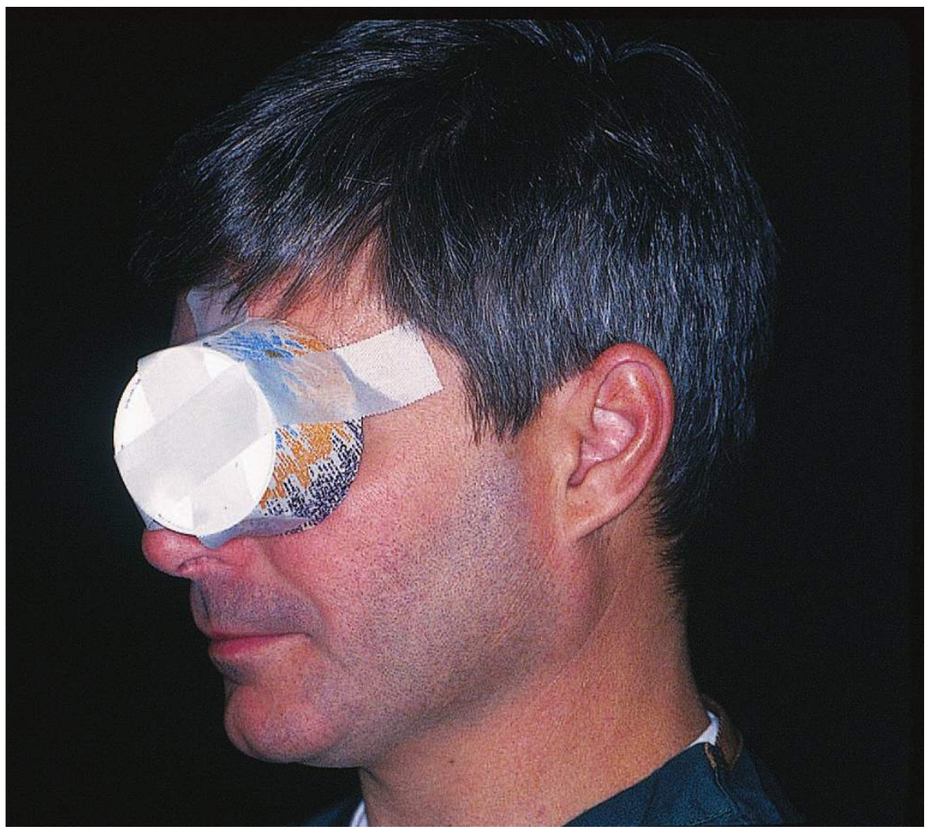


FIGURE 4.29 ■ **Protective Shield.** A protective shield is readily fashioned from a paper cup if a metal shield is not available. (Photo contributor: Kevin J. Knoop, MD, MS.)

GLOBE RUPTURE

Clinical Summary

A forceful blow to the eye may result in a ruptured globe. The diagnosis is obvious when orbital contents spill from the globe itself. Occult presentations occur when there is only a tiny rent in the sclera. Ruptures at the limbus, the margin between the cornea and sclera, may cause a small amount of iris to herniate, resulting in an irregularly shaped "teardrop pupil." A "peaked" or teardrop pupil may also result from a penetrating FB. Mechanism of injury is the key to distinguishing these two causes. Another associated finding is bloody chemosis involving the bulbar conjunctiva. Bulging of the conjunctiva over the wound distinguishes this from a simple subconjunctival hemorrhage. Suspect a rupture when severe conjunctival hemorrhage exists (covering 360° of the bulbar conjunctiva) following trauma.

Subconjunctival hemorrhage, nontraumatic bloody chemosis, corneal-scleral laceration, IO FB, iridodialysis, penetrating FB, and traumatic lens dislocation may have a similar presentation. A coloboma of the iris may appear similar to a teardrop pupil.

Management and Disposition

Urgent specialty consultation and operative management are mandatory. Protect the eye with a rigid eye shield, and all further examination and manipulation of the eye should be discouraged to prevent prolapse or worsening prolapse of choriouveal structures. Intravenous antibiotics to cover suspected organisms are appropriate. Adequate sedation and use of parenteral analgesics is encouraged. Also, address tetanus status. Since vomiting may result in further prolapse of intraocular contents, treat with antiemetics proactively. Consider CT scanning if the presence of an FB is suspected.

Pearls

1. The eyeball may appear deflated or the anterior chamber excessively deep. Intraocular pressure will likely be decreased, but measurement is contraindicated, since this may worsen herniation of intraocular contents.
2. Rupture usually occurs where the sclera is the thinnest, at the point of attachment of extraocular muscles and at the limbus.
3. A teardrop pupil may easily be overlooked in the triage process or in the setting of multiple traumatic injuries.
4. Seidel test (instillation of fluorescein and observing for fluorescein streaming away from the injury) may be used to diagnose subtle perforation.
5. Penetrating globe injuries are a relative contraindication to the sole use of depolarizing neuromuscular blockade (eg, succinylcholine). Consider a "de-fasciculating" dose of a nondepolarizing agent as a pretreatment to avoid increasing intraocular pressure.



FIGURE 4.30 ■ **Prolapsed Iris.** A teardrop pupil is present, with a small amount of iris herniating from a rupture at 8-o'clock position on the limbus. (Photo contributor: Lawrence B. Stack, MD.)

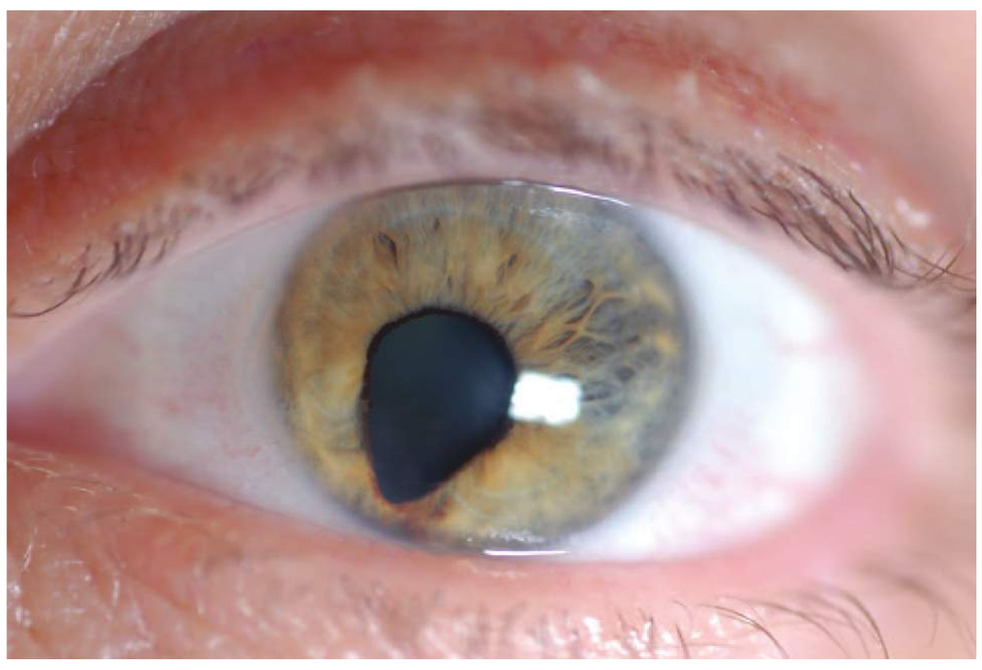


FIGURE 4.31 ■ **Iris Coloboma.** Iris coloboma is a congenital finding resulting from incomplete closure of the fetal ocular cleft. It appears as a teardrop pupil and may be mistaken for a sign of scleral rupture. (Photo contributor: R. Jason Thurman, MD.)

TRAUMATIC CATARACT

Clinical Summary

Any trauma to the eye that disrupts the normal architecture of the lens may result in the development of a traumatic cataract—a lens opacity. The mechanism behind cataract formation involves fluid infiltration into the normally avascular and acellular lens stroma. The lens may be observed to swell with fluid and become cloudy and opacified. The time course is usually weeks to months following the original insult. Cataracts that are large enough may be observed by the naked eye. Those that are within the central visual field may cause blurring of vision or distortion of light around objects (eg, halos).

Management and Disposition

No specific treatment is rendered in the emergency department for cases of delayed traumatic cataract. Most cases require routine ophthalmologic referral.

Pearls

1. Traumatic cataracts are frequent sequelae of lightning injury. Advise all victims of lightning strike of this possibility.
2. Cataracts may also occur as a result of electric current injury to the vicinity of the cranial vault.
3. Leukocoria results from a dense cataract, which causes loss of the red reflex.
4. If a cataract develops sufficient size and "swells" the lens, the trabecular meshwork may become blocked, producing glaucoma.

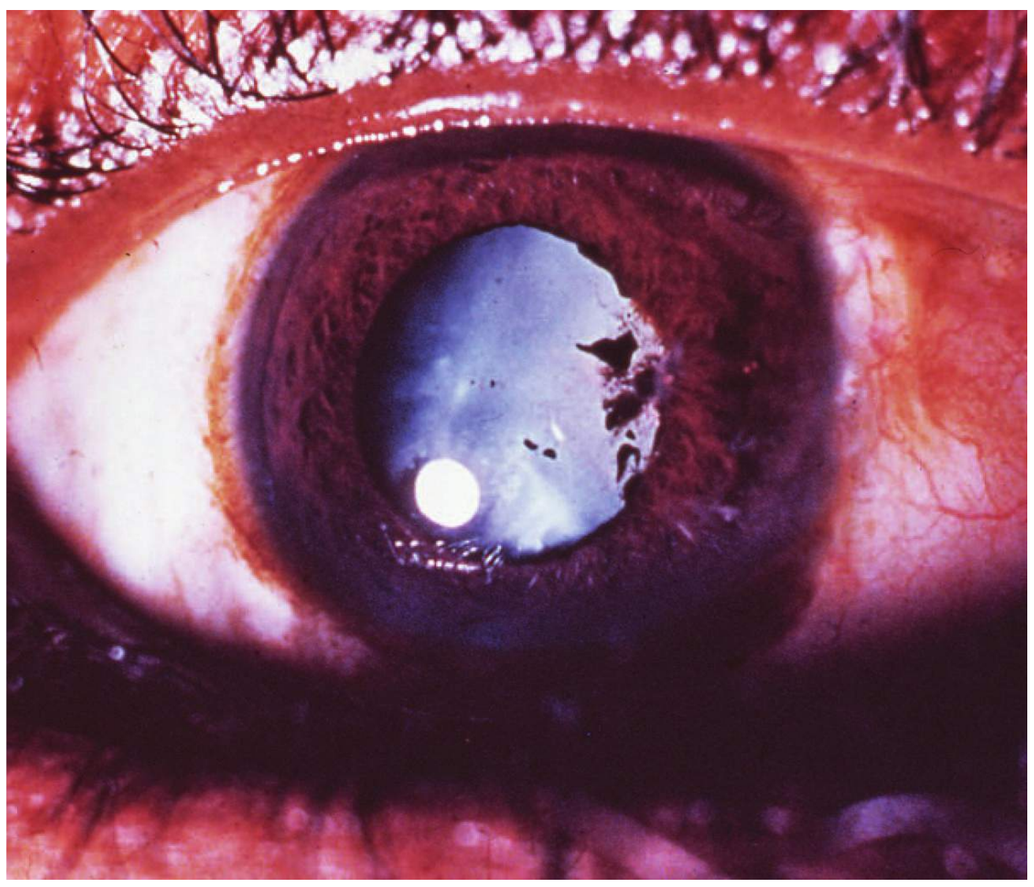


FIGURE 4.32 ■ **Traumatic Cataract.** This mature traumatic cataract is seen as a large lens opacity overlying the visual axis. A traumatic iridodialysis is also present. (Photo contributor: David Effron, MD.)

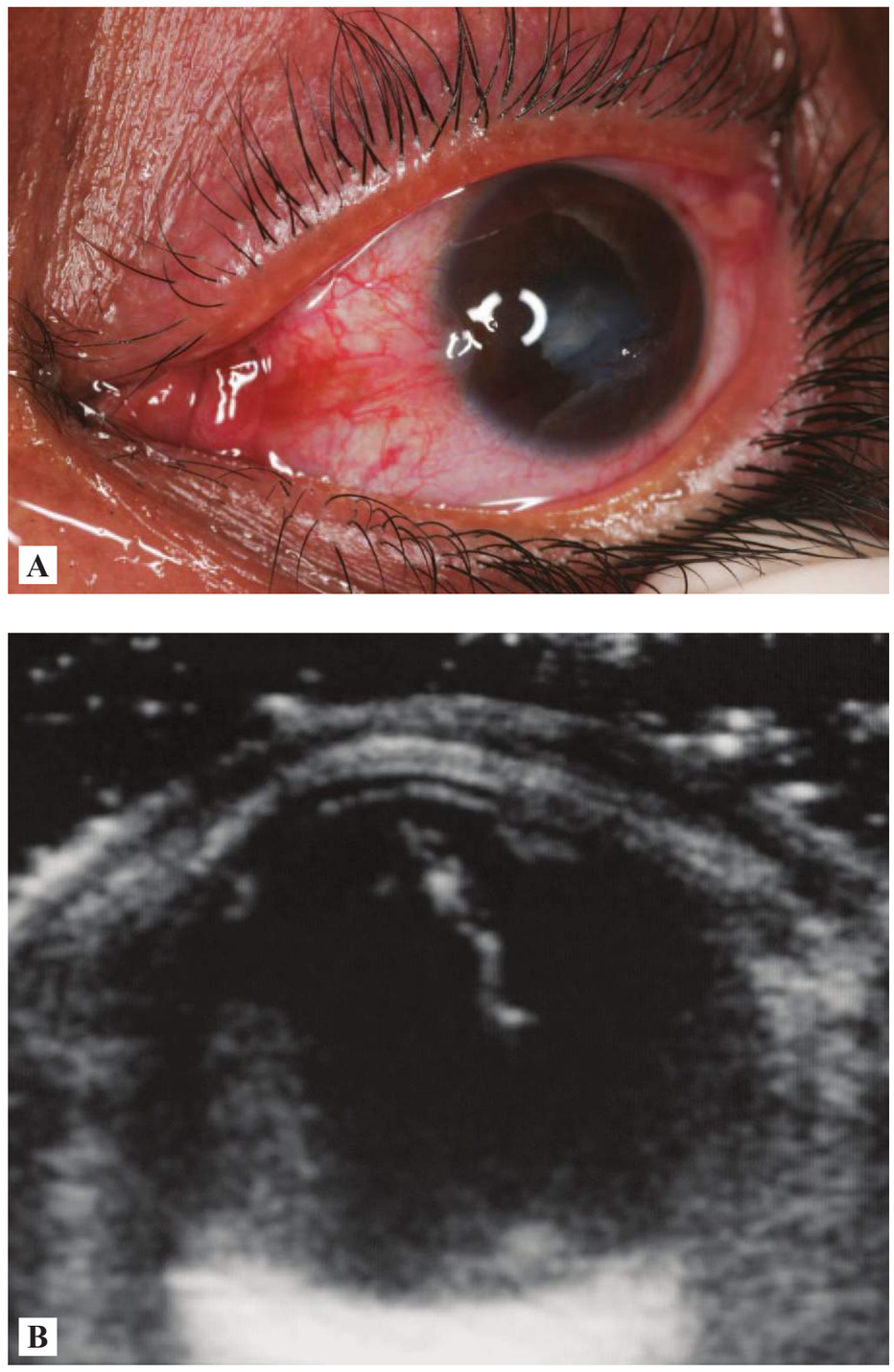


FIGURE 4.33 ■ **Traumatic Cataract.** (**A**) This acute traumatic cataract is seen as a milky cornea at the time of injury. (**B**) Bedside ultrasound shows a collapsed anterior chamber and intraocular foreign body. (Photo contributor: Kevin J. Knoop, MD, MS.)

EYELID LACERATION

Clinical Summary

Eyelid lacerations should always prompt a thorough search for associated injury to the globe, penetration of the orbit, or involvement of other adnexal structures (eg, lacrimal glands, canaliculi, puncta). Depending on the mechanism of injury, a careful exclusion of FB is important.

Management and Disposition

Repair eyelid lacerations involving superficial skin with 6-0 nonabsorbable interrupted sutures, which should remain in place for 3 to 5 days. Lacerations through an anatomic structure called the gray line, situated on the palpebral edge, require diligent reapproximation making referral prudent. Other injuries that require specialty consultation for repair include:

—Lacerations through the lid margins: these require exact realignment to avoid entropion or ectropion.

—Deep lacerations through the upper lid that divide the levator palpebrae muscles or their tendinous attachments: require repair with fine absorbable suture to avoid ptosis.

—Lacrimal duct injuries: these are repaired by stenting of the duct to avoid permanent epiphora.

—Medial canthal ligaments: these require repair to avoid drooping of the lids and telecanthus.

The most important objectives are to rule out injury to the globe and to search diligently for foreign bodies.

Pearls

1. Lacerations of the medial one-third of the lid or epiphora (tearing) raises suspicion for injury to the lacrimal system or the medial canthal ligament.
2. A small amount of adipose tissue seen within a laceration is a sign that perforation of the orbital septum has occurred (there is no subcutaneous fat in the eyelids).

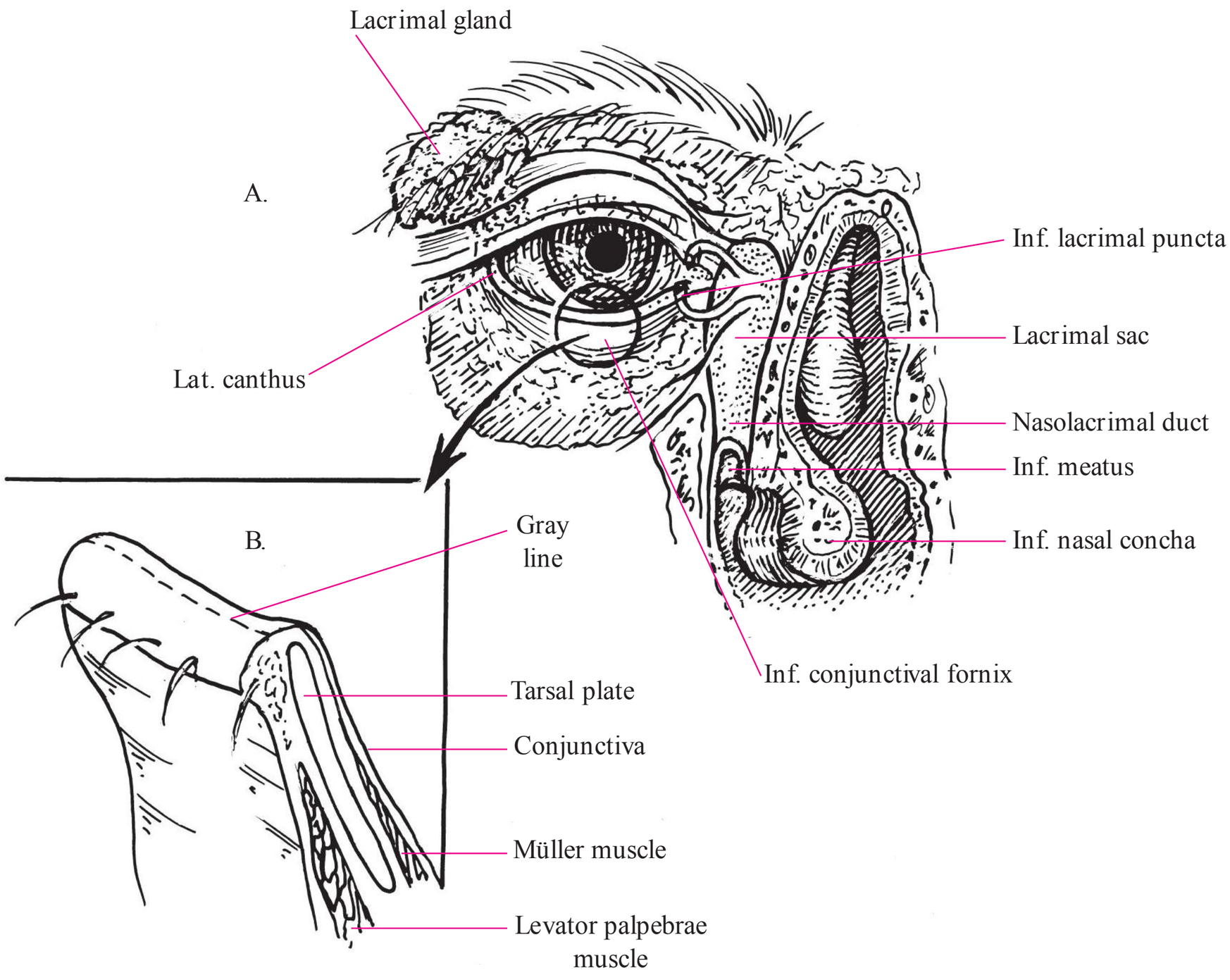


FIGURE 4.34 ■ **Eyelid and Adnexa Anatomy.** Ocular trauma should prompt examination of surrounding anatomic structures for associated injuries.

3. Laceration of the levator palpebrae musculature or tendinous attachments may result in traumatic ptosis.
4. Laceration of the canthal ligamentous support is suggested when there is rounding of the lid margins or telecanthus (widening of the distance between the medial canthi).
5. Anesthesia of the forehead may result from supraorbital nerve injury and should be sought prior to instilling local anesthetics.



FIGURE 4.35 ■ **Eyelid Laceration.** This complex laceration showing the displaced inferior punctum clearly violates the canalicular structures. (Photo contributor: Harold Lee, MD.)

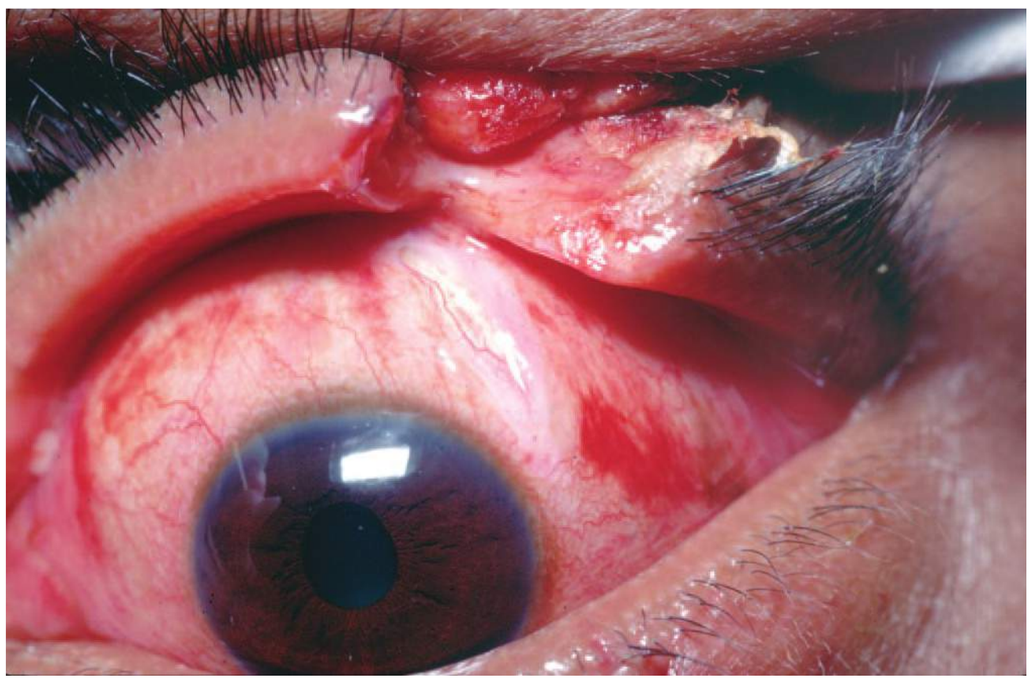


FIGURE 4.36 ■ **Eyelid Laceration.** Laceration involving the lid margin requires anatomic closure, ideally by a specialist. Careful approximation of the lid margins is required for adequate function. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 4.37 ■ **Eyelid Laceration.** Assault resulting in laceration to upper lid involving the tarsal plate. The assailant was wearing a ring. (Photo contributor: Kevin J. Knoop, MD, MS.)

Clinical Summary

Symptomatic ocular exposures involve either immediate or delayed onset of eye discomfort accompanied either by itching, tearing, redness, photophobia, blurred vision, and/or by foreign-body sensation. Conjunctival injection or chemosis may be seen. Exposure to defensive sprays or riot-control agents (eg, Mace or tear gas) causes immediate onset of severe burning, intense tearing, blepharospasm, and nasal and oropharynx irritation.

Management and Disposition

Begin copious irrigation immediately at the scene. Acute caustic exposures are triaged to immediate treatment. Determine the conjunctival sac pH with a broad-range pH paper without delaying treatment. Continue irrigation until achieving a tear film pH of 7.4. Topical anesthetic drops permit examination and facilitate irrigation. Evert upper lid conjunctiva to examine for concretions. Alkali exposures penetrate deeper into tissues, causing more severe injury (liquefaction necrosis). Acids are less damaging (coagulation necrosis), which creates a barrier to further penetration. The exception is hydrofluoric acid, which acts as an alkali exposure.

Irrigate liberally after all exposures. Many chemicals merely cause irritative symptoms; however, some may also denude the corneal epithelium and inflame the anterior chamber. Perform slit-lamp examination to document corneal epithelial defects or anterior chamber inflammation. Cycloplegics may reduce ciliary spasm and pain. Address tetanus status.

Pearls

1. Immediate onset of severe symptoms calls for immediate treatment and should prompt consideration of alkali or acid exposure.
2. Prolonged (up to 24 hours) irrigation may be needed for alkaline exposures.
3. Concretions from the exposure agent may form deep in the conjunctival fornices. Removal is critical to prevent ongoing injury.
4. A Morgan lens or other eye irrigation system is ideal for effective treatment as blepharospasm severely limits effectiveness of intravenous tubing alone.



FIGURE 4.38 ■ **Alkali Burn.** Diffuse opacification of the cornea occurred from a "lye" burn to the face. (Photo contributor: Stephen W. Corbett, MD.)



FIGURE 4.39 ■ **Alkali Burn.** Diffuse fluorescein uptake of the entire cornea is seen from an alkali exposure. (Photo contributor: Sarah M. Escott, MD.)



FIGURE 4.40 ■ **Caustic Burn Adhesions (Symblepharon).** Scarring of both palpebral and bulbar conjunctivae results in severe adhesions between the lids and the globe. (Photo contributor: Arden H. Wander, MD.)

Chapter 5

EAR, NOSE, AND THROAT CONDITIONS

Scan QR code or go to www.ematlas.com for video related to chapter

Edward C. Jauch
David R. White
Kevin J. Knoop



Mastoiditis. Postauricular redness, swelling, and proptosis in a young child with acute mastoiditis. (Photo contributor: Lawrence B. Stack, MD.)

The authors acknowledge Sean Barbabella and Francisco Fernandez for portions of this chapter written for the previous editions of this book.

Clinical Summary

Children between the ages of 6 months and 2 years are at highest risk of developing acute otitis media (AOM). Children at increased risk of recurrent AOM contract their first episode prior to 12 months, have a sibling with a history of recurrent AOM, are in day care, or have parents who smoke.

AOM is an acute inflammation and effusion of the middle ear. Viral, bacterial, and fungal pathogens may cause AOM. The pathogenesis of bacterial AOM is eustachian tube dysfunction, typically following a viral infection, allowing retention of secretions (serous otitis) and seeding of bacteria. The most common bacterial isolates are Streptococcus pneumoniae, Haemophilus influenzae, Moraxella catarrhalis, and Streptococcus pyogenes. There is an increased prevalence of antimicrobial resistance for S pneumoniae and β-lactamase-producing strains of H influenzae. Vaccination for strains of S pneumoniae has resulted in a modest decrease in incidence of AOM.

Patient presentations and complaints vary with age. Infants with AOM have vague, nonspecific symptoms (irritability, lethargy, and decreased oral intake). Young children can be irritable, often febrile, and frequently pull at their ears, but they may also be completely asymptomatic. Older children and adults note ear pain, impaired hearing, and occasionally otorrhea.

Otoscopy should focus on color, position, translucency, and mobility of the tympanic membrane (TM). Compared with the TM of a normal ear, AOM causes the TM to appear dull, erythematous or injected, bulging, and less mobile. The light reflex, normal TM landmarks, and malleus become obscured. Pneumatic otoscopy and tympanometry enhance accuracy in diagnosing AOM.

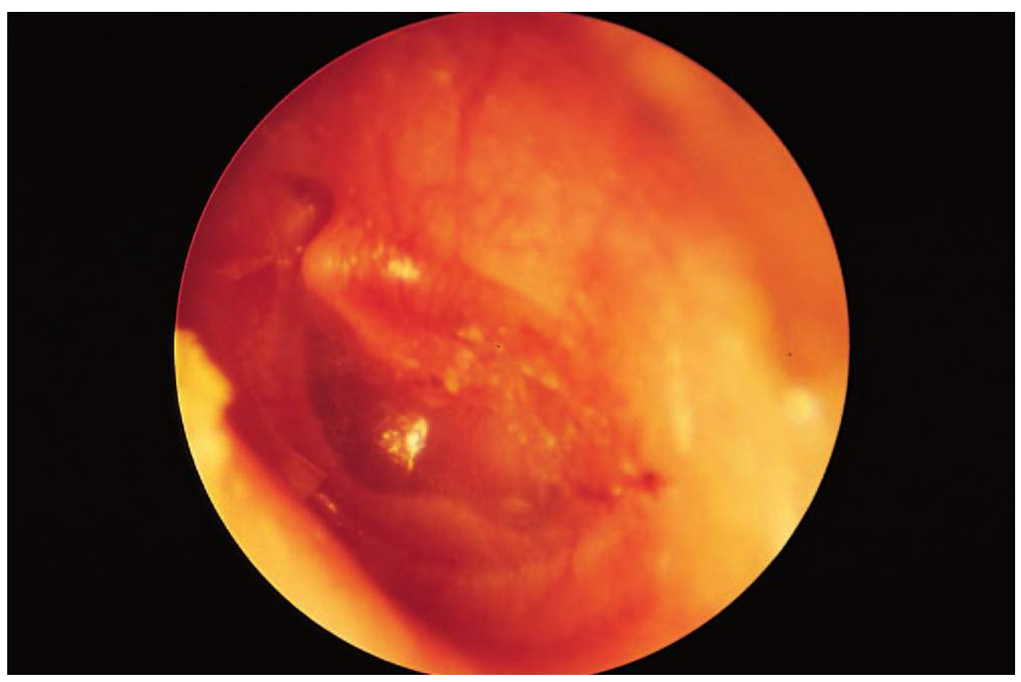


FIGURE 5.2 ■ **Early Acute Otitis Media.** A mildly erythematous tympanic membrane is seen with a small purulent effusion in the middle ear. (Photo contributor: C. Bruce MacDonald, MD.)

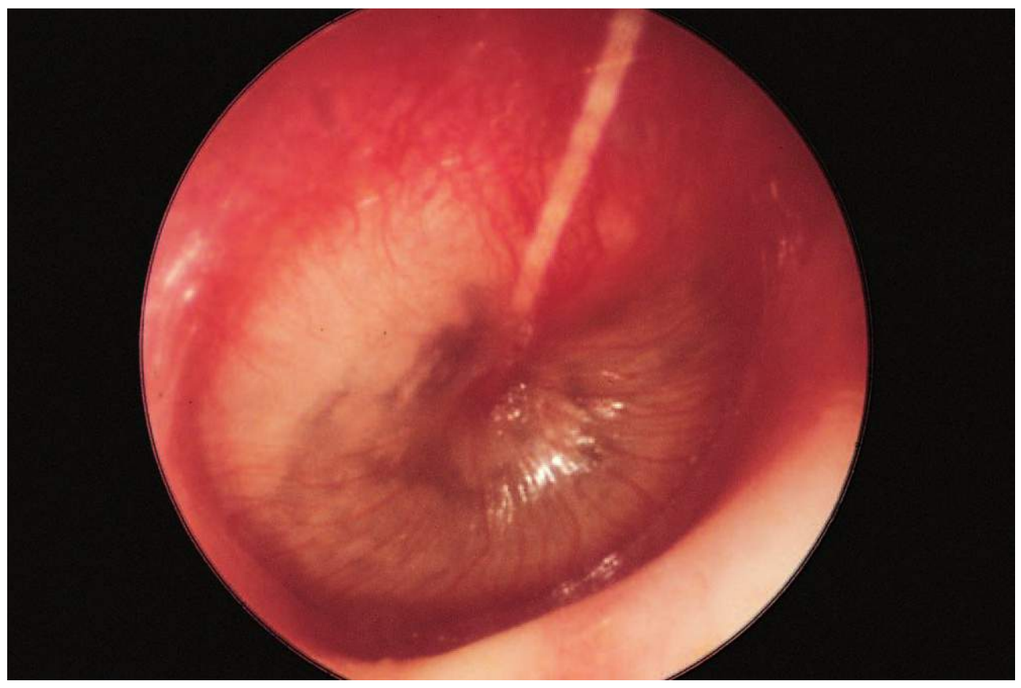


FIGURE 5.3 ■ **Acute Otitis Media.** The middle ear is filled with purulent material behind an erythematous, bulging tympanic membrane. (Photo contributor: Richard A. Chole, MD, PhD.)

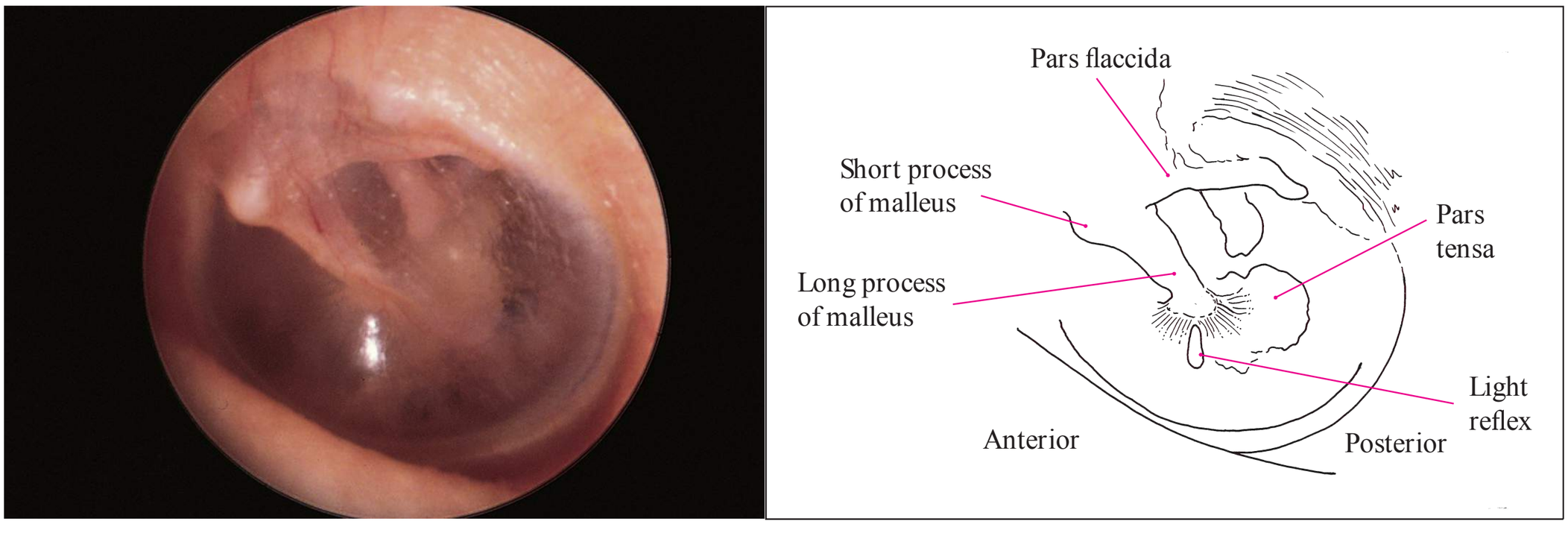


FIGURE 5.1 ■ **Normal Left Tympanic Membrane.** Normal tympanic membrane anatomy and landmarks. (Photo contributor: Richard A. Chole, MD, PhD.)

Management and Disposition

Although AOM generally resolves spontaneously, most patients are treated with antibiotics and analgesics. Steroids, decongestants, and antihistamines do not alter the course in AOM but may improve upper respiratory tract symptoms.

Follow-up in 10 to 14 days or return if symptoms persist or worsen after 48 hours is appropriate and patients should be referred to an otolaryngologist for further evaluation, an audiogram, and possible tympanostomy tubes if they have significant hearing loss, failure of two complete courses of outpatient antibiotics during a single event, chronic otitis media (OM) with or without acute exacerbations, or failure of prophylactic antibiotics.

Pearls

1. AOM is a clinical diagnosis based on the presence of distinct fullness or bulging of the TM.
2. In children, recurrent OM may be due to food allergies.
3. Only 4% of children less than 2 years with AOM develop temperatures greater than 104°F. Those with fever higher than 104°F or with signs of systemic toxicity should be closely evaluated for other causes before attributing the fever to AOM.
4. Most children over 2 years qualify for observation for 48 hours prior to initiation of antibiotics.
5. Management of AOM may require narcotic analgesia; topical Auralgan may decrease pain.

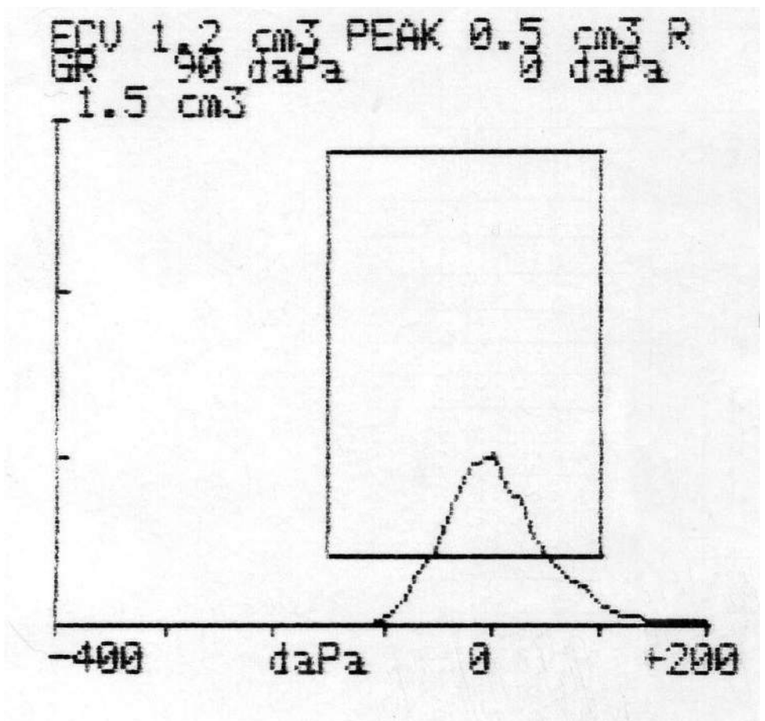


FIGURE 5.4 ■ **Tympanogram of Normal Ear.** The compliance tracing of a normal ear. (Photo contributor: Lawrence B. Stack, MD.)

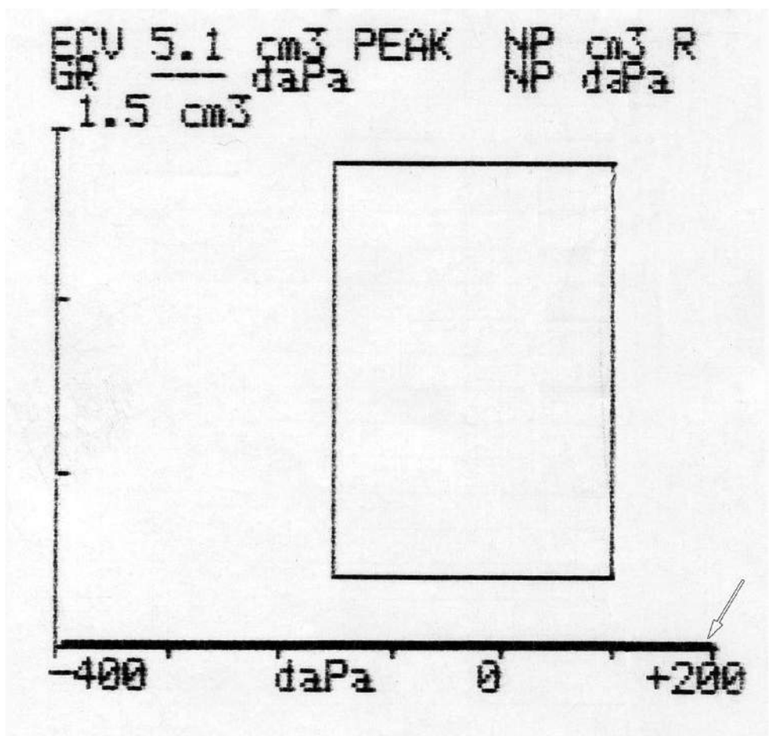


FIGURE 5.5 ■ **Tympanogram of Otitis Media.** The compliance tracing of an ear with otitis media. The line is flat (arrow). (Photo contributor: Lawrence B. Stack, MD.)

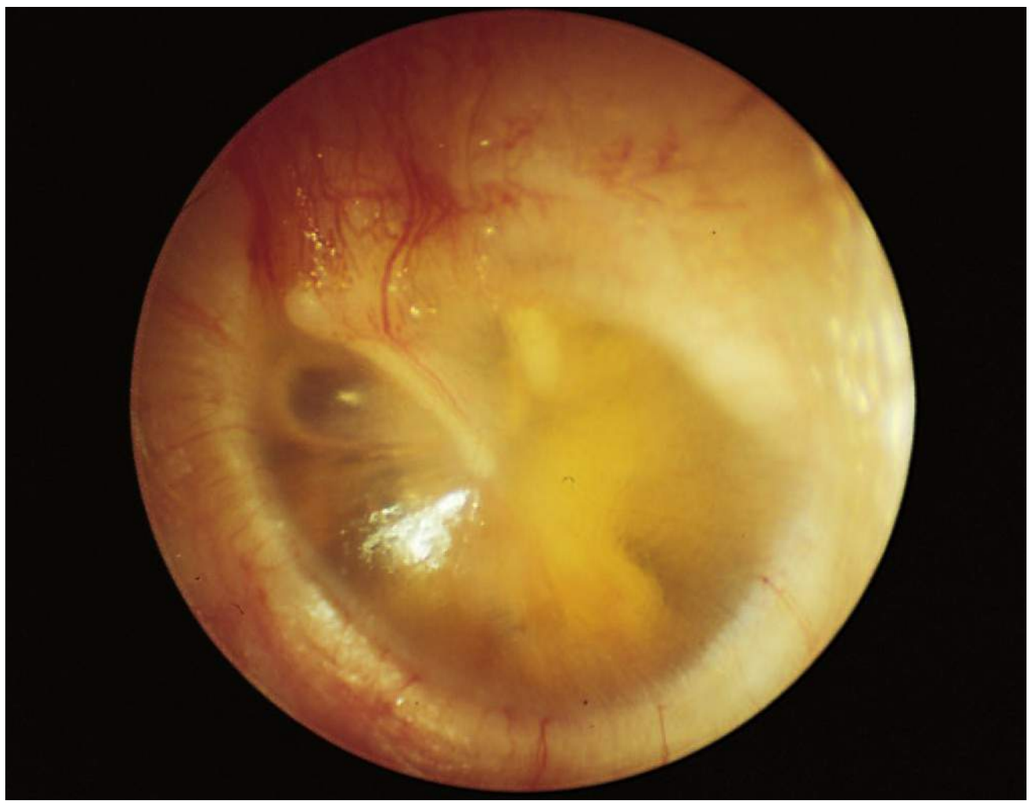


FIGURE 5.6 ■ **Serous Otitis Media with Effusion (OME).** Serous OME is commonly seen after AOM, but it is also common without this history. Any process that leads to obstruction of a eustachian tube may cause OME. A clear, amber-colored effusion with a single air-fluid level is seen in the middle ear behind a normal tympanic membrane. (Photo contributor: C. Bruce MacDonald, MD.)

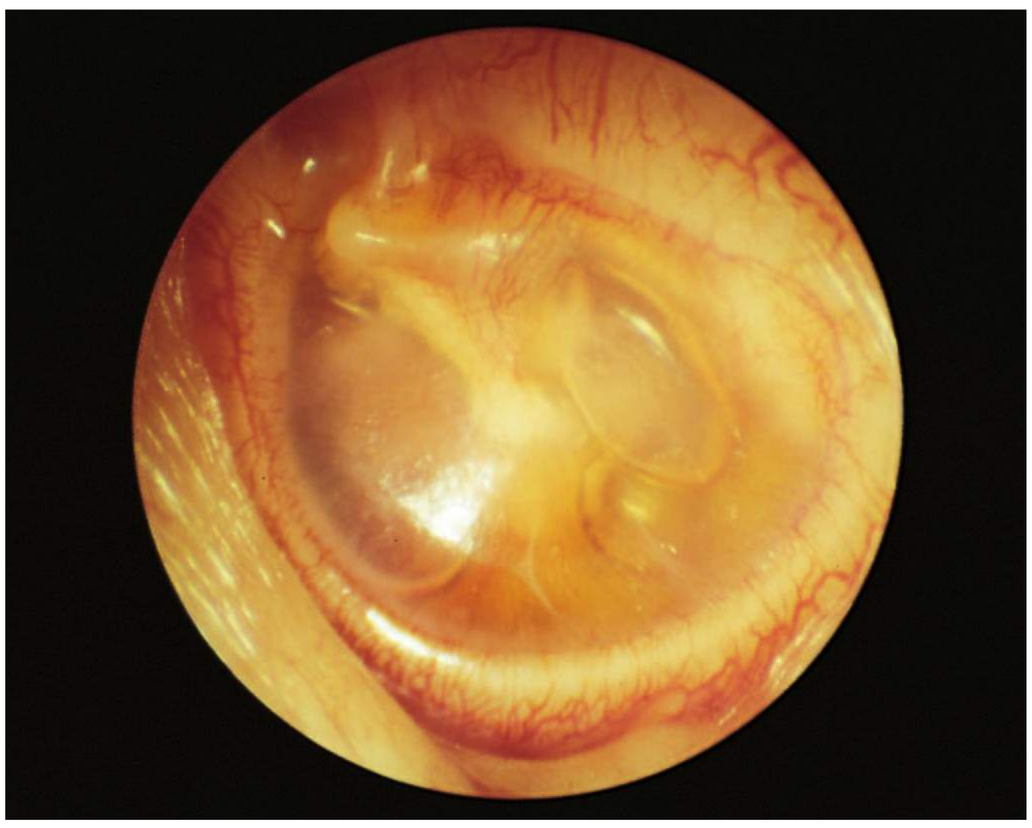


FIGURE 5.7 ■ **Serous OME.** A clear, amber-colored effusion with multiple air-fluid levels is seen in the middle ear behind a normal tympanic membrane. (Photo contributor: C. Bruce MacDonald, MD.)

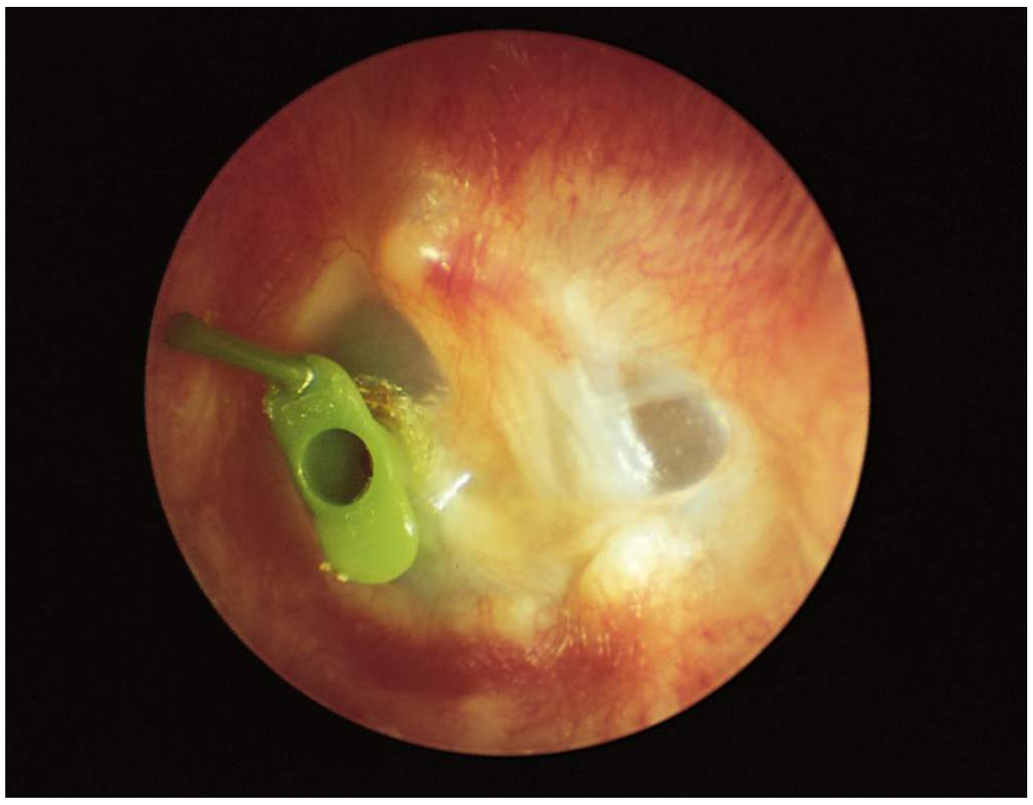


FIGURE 5.8 ■ **Tympanostomy Tube.** Typical appearance of a tympanostomy tube in the tympanic membrane. These tubes will migrate to the periphery and eventually drop out. Occasionally, they will be found in the external ear canal. (Photo contributor: C. Bruce MacDonald, MD.)

Clinical Summary

Bullous myringitis is a direct inflammation and infection of the TM secondary to a viral or bacterial agent. Vesicles or bullae filled with blood or serosanguinous fluid on an erythematous TM are the hallmarks. Frequently, a concomitant OM with effusion is noted. Typical pathogens are the same as seen in AOM.

The onset of bullous myringitis is preceded by an upper respiratory tract infection and is heralded by sudden onset of severe ear pain, scant serosanguinous drainage from the ear canal, and frequently some degree of hearing loss. Otoscopy reveals bullae on either the inner or outer surface of the TM. Patients presenting with fever, hearing loss, and purulent drainage are more likely to have concomitant infections, such as OM and otitis externa (OE).

Management and Disposition

Differentiation between viral and bacterial etiologies for TM bullae is not necessary. Although most episodes resolve spontaneously, many physicians prescribe antibiotics. Warm compresses, topical or strong analgesics, and oral decongestants provide symptomatic relief. Referral is not necessary in most cases unless rupture of the bullae is required for pain relief.

Pearls

1. Instruct parents that TM rupture may occur with sudden resolution of the pain and drainage from the ear canal.
2. Carefully differentiate TM bullae from cholesteatomas or herpetic vesicles.
3. Facial nerve paralysis associated with clear, fluid-filled TM vesicles is characteristic of herpes zoster oticus (HZO).

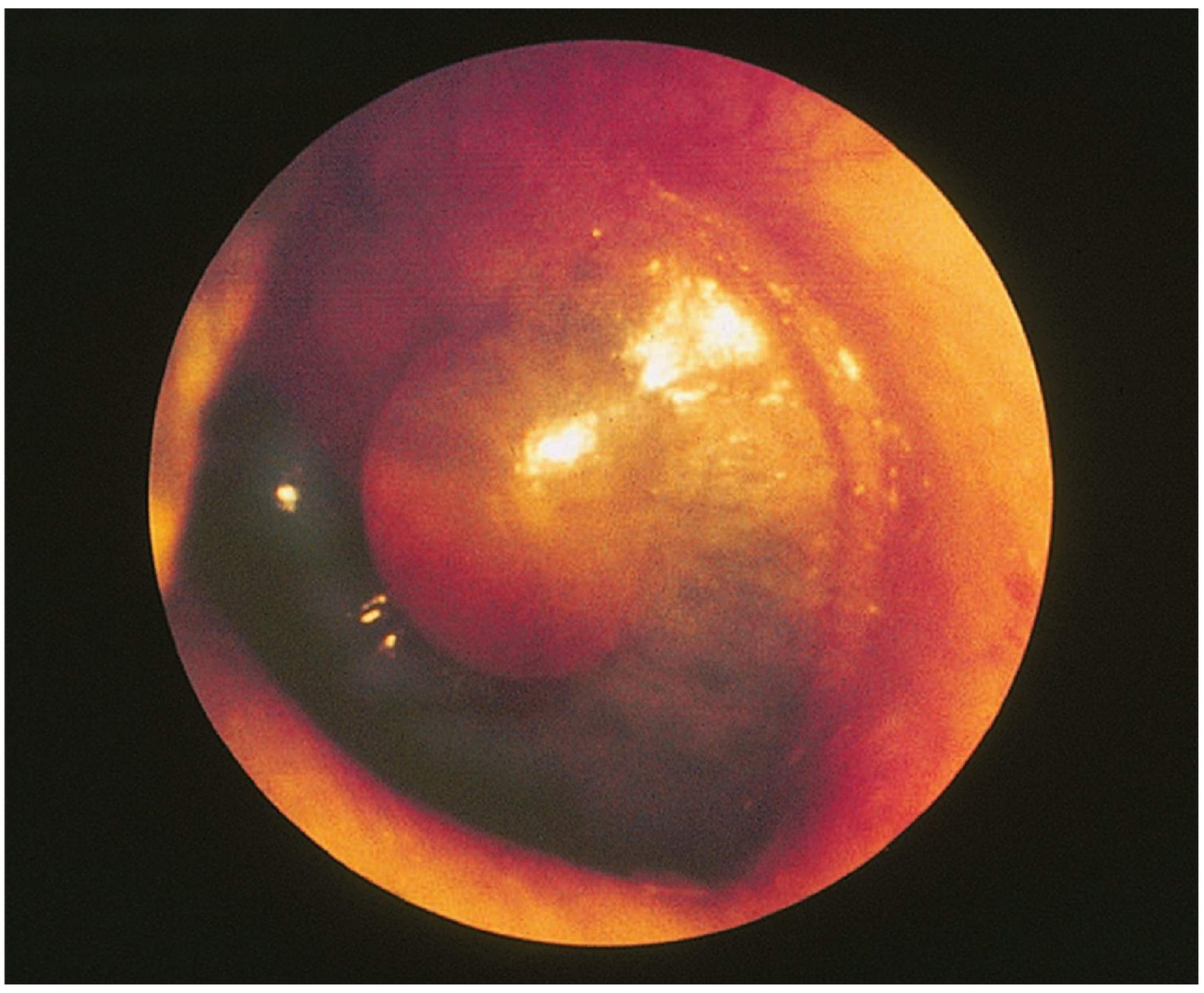


FIGURE 5.9 ■ **Bullous Myringitis.** A large fluid-filled bulla is seen distorting the surface of the tympanic membrane. (Photo contributor: Richard A. Chole, MD, PhD.)

Clinical Summary

Cholesteatomas are collections of desquamating stratified squamous epithelium found in the middle ear or mastoid air cells. Congenital cholesteatomas occur most frequently in children and young adults. Acquired cholesteatomas originate from perforation or retraction of the TM allowing migration of epithelium into the middle ear.

Injury may occur to middle ear ossicles through the production of collagenases, and may erode into the temporal bone, inner ear structures, mastoid sinus, or posterior fossa dura. Treatment delays can lead to permanent conductive hearing loss or infectious complications.

Many cholesteatomas have an insidious progression without associated pain or symptoms. Computed tomography (CT) scans may reveal bony destruction.

Management and Disposition

Refer initial diagnosis. No medical or surgical management is required unless they become symptomatic.

Pearls

1. Persistent pain associated with headache, facial motor weakness, nystagmus, or vertigo suggests inner ear or intracranial involvement.
2. Polyps found on the TM can indicate the presence of a cholesteatoma and require further evaluation to exclude its presence.

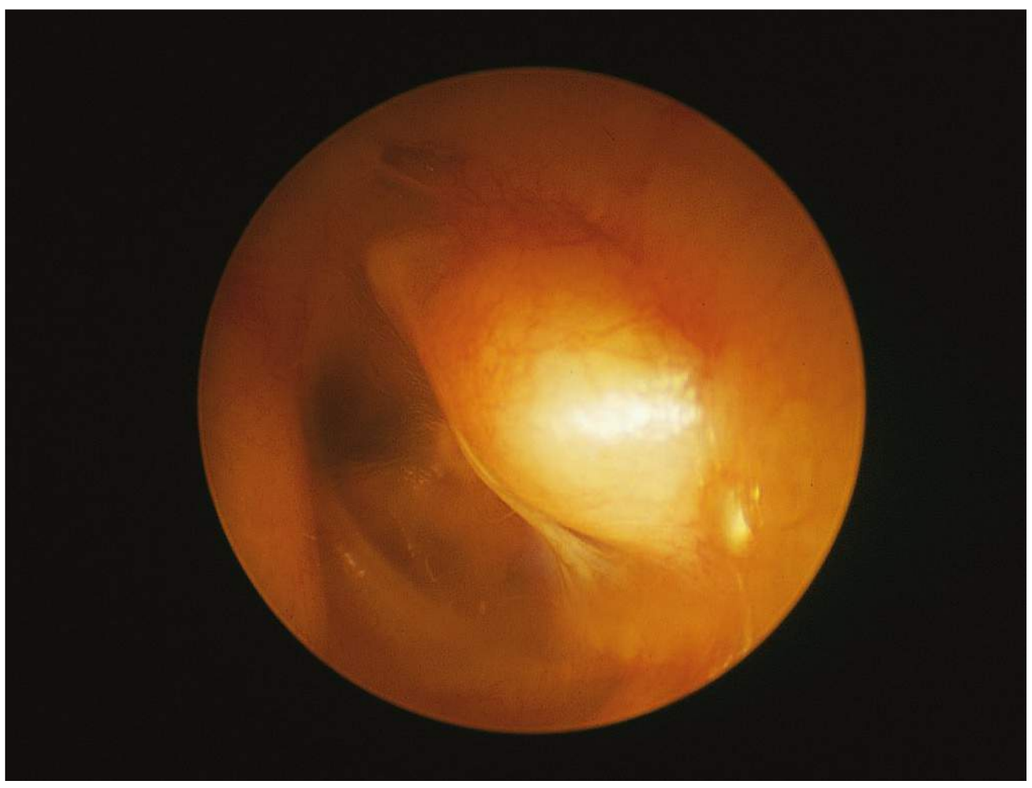


FIGURE 5.10 ■ **Congenital Cholesteatoma.** A congenital cholesteatoma is seen behind an intact tympanic membrane. (Photo contributor: C. Bruce MacDonald, MD.)

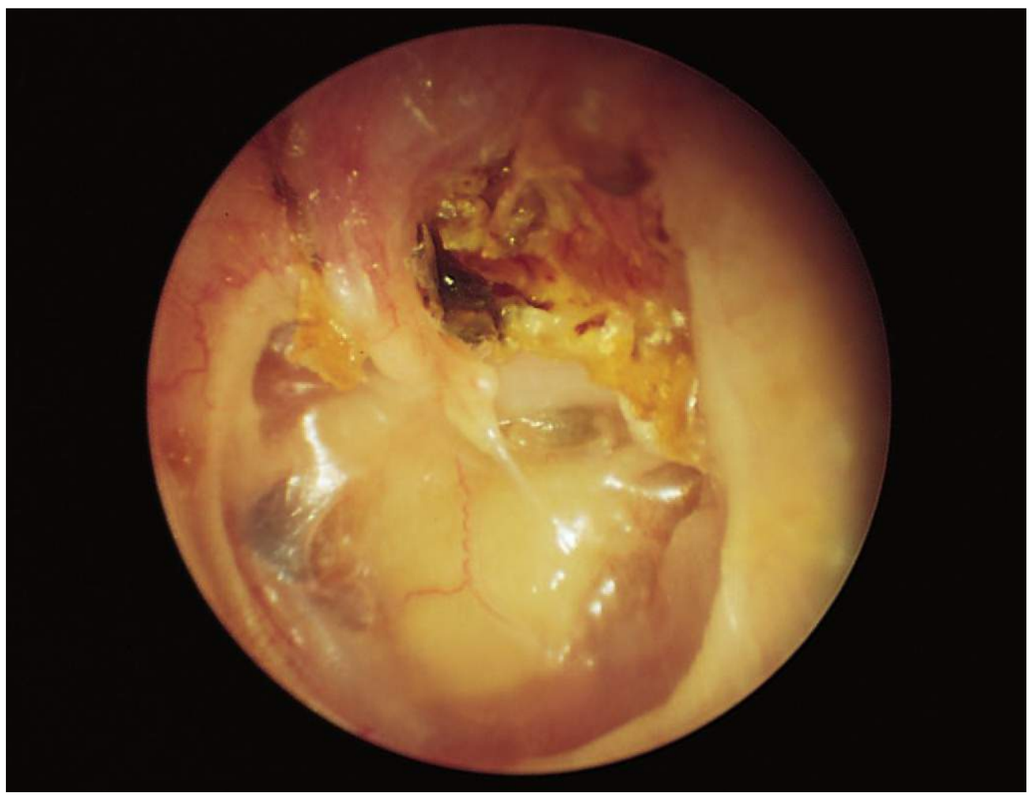


FIGURE 5.11 ■ **Acquired Cholesteatoma.** A cholesteatoma is seen. Note the yellow epithelial debris from the cholesteatoma in the area of the pars flaccida. Often there is an effusion and debris, which can distort the anatomy on otoscopy. (Photo contributor: C. Bruce MacDonald, MD.)

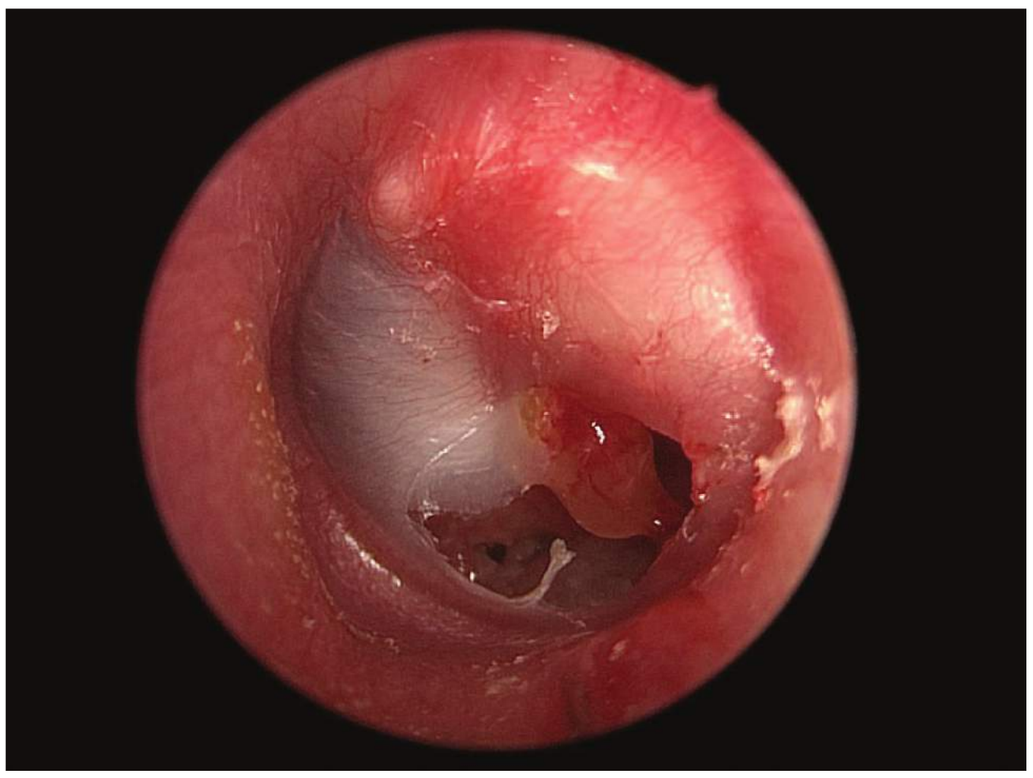


FIGURE 5.12 ■ **Acquired Cholesteatoma.** This is a left ear with an inferior perforation and granulation tissue present just below the tip of the malleus. A white mass is seen behind the eardrum in the posterosuperior quadrant, roughly 12-o'clock to 3-o'clock position. This patient had a history of PE tubes placed several years ago with a resultant perforation allowing squamous epithelium access to the middle ear. (Photo contributor: David R. White, MD)

Clinical Summary

Acute tympanic membrane (TM) perforations are caused by direct penetrating trauma, barotrauma, OM, corrosives, thermal injuries, and iatrogenic causes (foreign-body removal, tympanostomy tubes). TM perforations are occasionally accompanied by injuries to the ossicular chain and temporal bone.

Patients with an acute TM perforation complain of a sudden onset of ear pain, vertigo, tinnitus, and altered hearing after a specific event. Physical examination of the TM reveals a slit-shaped tear or a larger perforation with an irregular border, often associated with blood along the margins. Subacute or chronic perforations have smooth margins and a round or ovoid shape.

Management and Disposition

Treatment of acute TM perforations is tailored to the mechanism of injury. All easily removable foreign bodies should be extracted. Corrosive exposures require face, eye, and ear decontamination. Antibiotics and irrigation do not improve the rate or completeness of healing unless the injury is associated with OM. Systemic antibiotics should be reserved for perforations associated with OM, penetrating injury, and possibly water-sport injuries (see "Otitis Media" above). Topical steroids impede perforation healing.

Patients are instructed to avoid water in the ear while the perforation is healing and to return if symptoms of infection appear. While nearly 80% of all TM perforations heal spontaneously, all TM perforations require referral to an otolaryngologist for follow-up for possible myringoplasty.

Pearls

1. Corticosteroid eardrops of any formulation retard spontaneous healing and should be avoided.
2. Traumatic TM perforation associated with cranial nerve (CN) deficits or persistent vertigo requires immediate otolaryngology consultation due to possible temporal bone fractures or injury to the round or oval window.

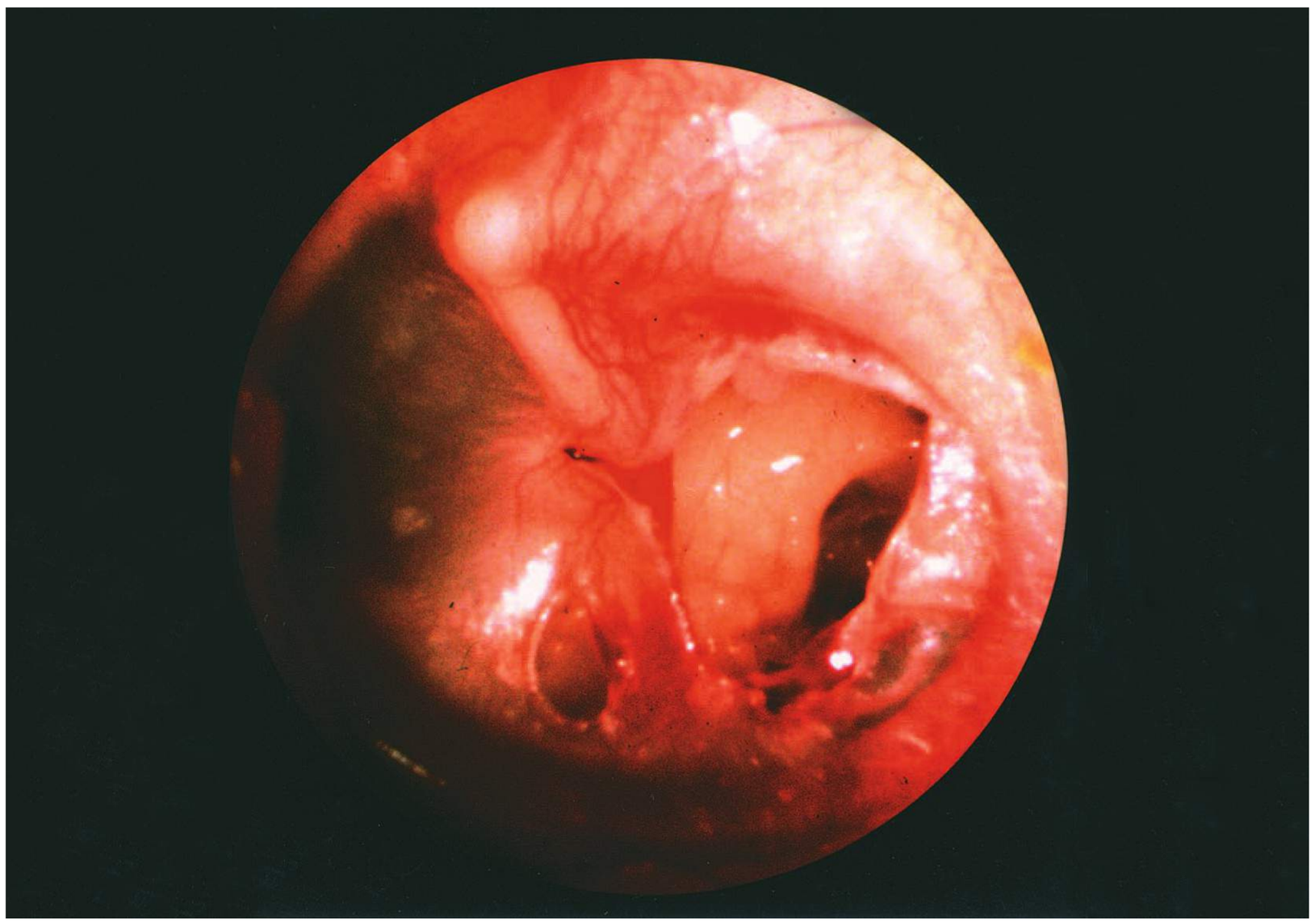


FIGURE 5.13 ■ **Acute Tympanic Membrane Perforation.** An acute tympanic membrane perforation is seen. Note the sharp edges of the ruptured tympanic membrane. (Photo contributor: Richard A. Chole, MD, PhD.)

OTITIS EXTERNA

Clinical Summary

Otitis externa (OE), or "swimmer's ear," is an inflammation and infection (bacterial or fungal) of the auricle and external auditory canal (EAC). Typical symptoms include otalgia, pruritus, otorrhea, and hearing loss. Physical examination reveals EAC hyperemia and edema, otorrhea, occlusion from debris and swelling, pain with manipulation of the tragus, and periauricular lymphadenopathy.

Several factors predispose the EAC to infection: increased humidity and heat, repeated water immersion, foreign bodies, trauma, hearing aids, and cerumen impaction. Bacterial OE is primarily an infection due to Pseudomonas species or Staphylococcus aureus. Diabetics are particularly prone to infections by Pseudomonas, Candida albicans, and, less commonly, Aspergillus niger.

Management and Disposition

EAC irrigation and suctioning is recommended to thoroughly evaluate the EAC. Topical antibiotic suspensions containing polymyxin, neomycin, and hydrocortisone or ciprofloxacin with ear wicks are effective. Topical solutions are not pH-balanced and thus are irritating and may cause inflammation in the middle ear if a perforation is present. Topical fluoroquinolones may be less irritating and are only given twice a day. Systemic antibiotics are not indicated unless extension into the periauricular tissues is noted. Patients should avoid swimming and prevent water from entering the ear while bathing. Dry heat aids in resolution, and analgesics provide symptomatic relief. Follow-up should be arranged in 10 days for routine cases.

Pearls

1. Resistant cases may have an allergic or eczematous component. These typically present with a dry, scaly, itchy EAC and are chronic in nature.
2. Drying the EAC after water exposure with a 50:50 mixture of isopropyl alcohol and water or with acetic acid (white vinegar) minimizes recurrence. If the TM is possibly perforated, isopropyl alcohol should be avoided.
3. If a TM perforation is suspected and antibiotic drops are indicated, a suspension is recommended.
4. Consider malignant OE, typically caused by Pseudomonas aeruginosa, in elderly, diabetics, or immunocompromised patients. Exposed bone, ulceration of the EAC, and facial nerve weakness are hallmarks.



FIGURE 5.14 ■ **Otitis Externa.** A discharge is seen coming from the external auditory canal, which is swollen and almost completely occluded. An ear wick placed in the EAC facilitates delivery of topical antibiotic suspension and drainage of debris. (Photo contributor: Frank Birinyi, MD.)

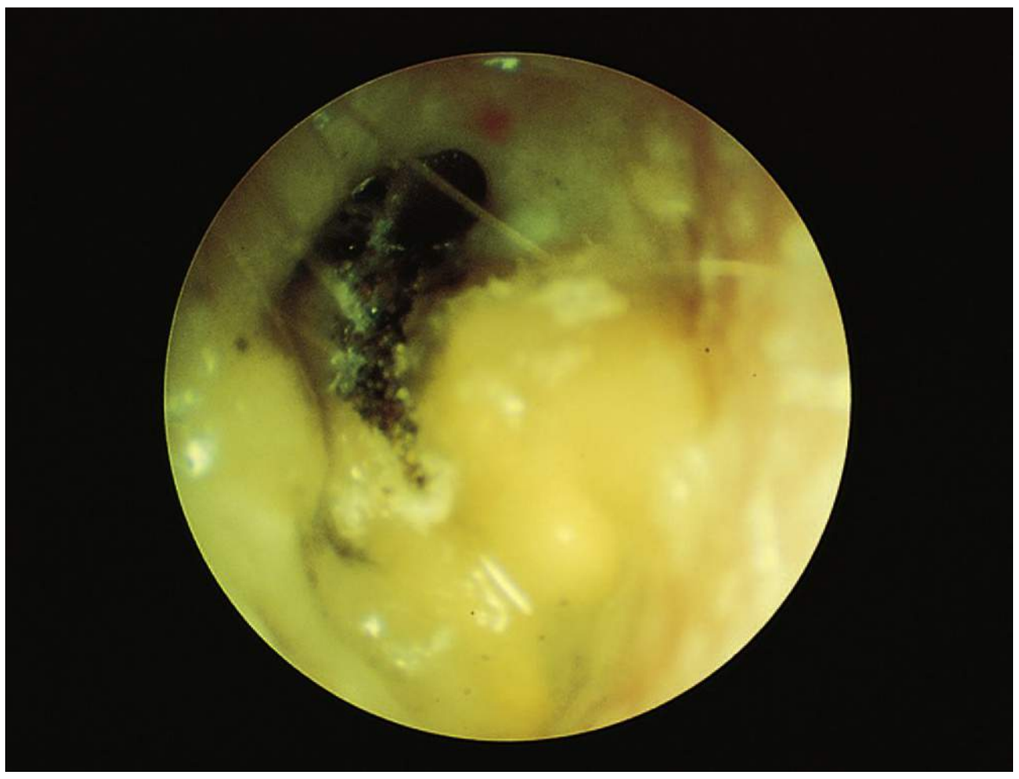


FIGURE 5.15 ■ **Aspergillus Otitis Externa.** Chronic otitis externa with copious debris, including black spores from A niger, cottony fungal elements, and wet debris. This patient had been treated with topical and systemic antibiotics. (Photo contributor: C. Bruce MacDonald, MD.)

PREAURICULAR SINUS ABSCESS

Clinical Summary

Preauricular sinus abscesses arise from pits or fissures originating from congenital malformations of the preauricular soft tissues. They are located near the front of the ear and mark the entrance to a sinus tract. These are lined with squamous epithelium and thus may produce epithelial-lined subcutaneous cysts, which may become infected, leading to cellulitis or abscess. Patients may have other congenital anomalies such as hearing loss or renal disease. Patients may present with recurrent ear discharge, pain, swelling, redness, itching, headache, and fever. The diagnosis is often overlooked in early stages.

Management and Disposition

Initiate oral antibiotics to cover for S aureus (most common). Incision and drainage should be avoided in the emergency department (ED). Needle aspiration may be attempted and may give some relief. Arrange follow-up with an otolaryngologist for complete excision of the sinus track.

Pearls

1. Preauricular sinus abscesses may be confused with first branchial cleft cysts.
2. Because of their close proximity to the facial nerve, referral to a specialist who is familiar with the underlying anatomy is recommended.



FIGURE 5.16 ■ **Preauricular Sinus Abscess.** Recurrent preauricular swelling and redness in the area of a "pit" suggests sinus abscess and was confirmed on contrast-enhanced CT scan. (Photo contributor: Lawrence B. Stack, MD.)

MASTOIDITIS

Clinical Summary

Mastoiditis is an infection or inflammation of the mastoid air cells often resulting from extension of purulent OM with progressive destruction and coalescence of air cells. Medial sinus wall erosion can cause cavernous sinus thrombosis, facial nerve palsy, meningitis, brain abscess, and sepsis. With the use of antibiotics for AOM, the incidence of mastoiditis has fallen sharply.

Patients present with fever, chills, postauricular ear pain, and otorrhea. Patients may have tenderness, erythema, swelling, and fluctuance over the mastoid process; proptosis of the pinna; erythema of the posterior-superior EAC wall; and otorrhea.

Management and Disposition

Initial evaluation includes a thorough head, neck, and CN examination. Contrasted CT of the head or mastoid sinus may reveal bone erosion and intracranial involvement.

While oral penicillinase-resistant penicillins, amoxicillin-clavulanic acid, third-generation cephalosporins, and the newer macrolides are effective in mild cases of mastoiditis, severe cases require parenteral antibiotics. Mastoiditis requires prompt consultation and close follow-up.

Pearls

1. Most patients require admission for parenteral antibiotics to cover H influenzae, M catarrhalis, streptococcal species, and S aureus.
2. Surgical incision and debridement, and possibly mastoidectomy, are reserved for refractory cases.
3. Chronic mastoiditis describes chronic otorrhea of at least 2-month duration. It is often associated with craniofacial anomalies.



FIGURE 5.17 ■ **Acute Mastoiditis.** Postauricular swelling, redness, and proptosis in a young girl with acute mastoiditis and sinusitis. (Photo contributor: Lawrence B. Stack, MD.)

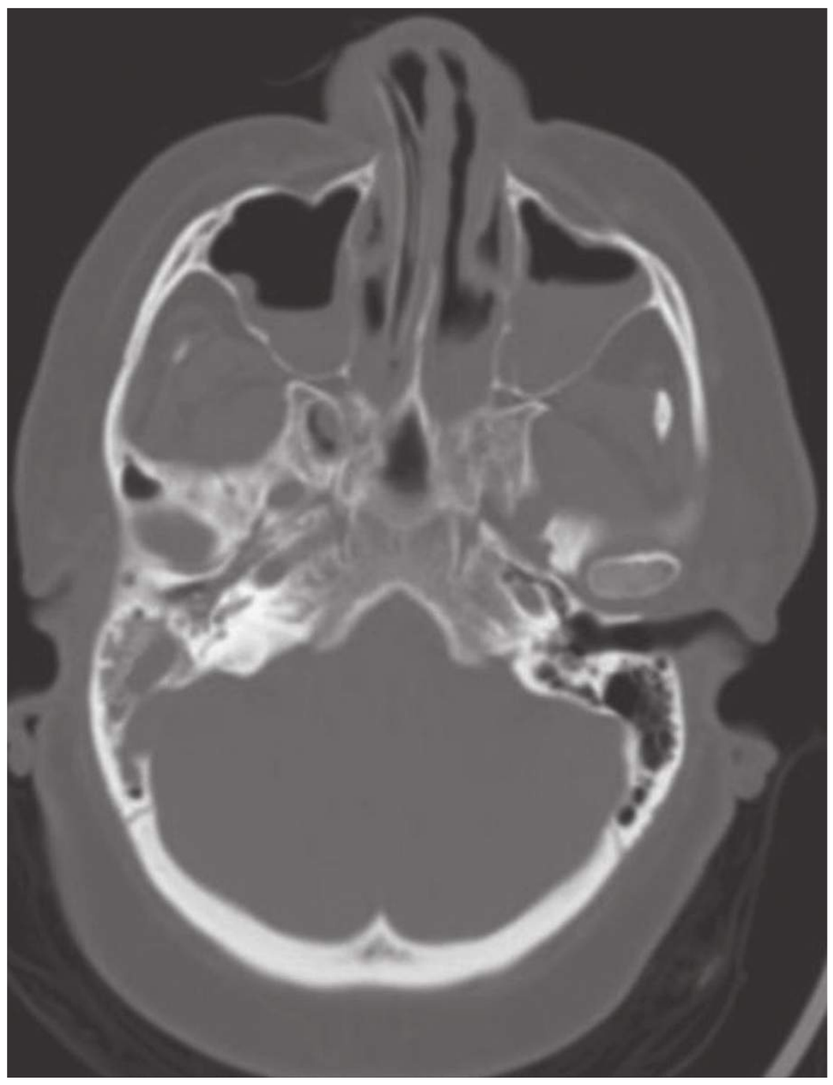


FIGURE 5.18 ■ **Acute Mastoiditis.** Bone window of a head CT scan which demonstrates fluid in the right mastoid air cells. Maxillary sinus disease is also present. (Photo contributor: Lawrence B. Stack, MD.)

AURICULAR PERICHONDRITIS

Clinical Summary

Auricular perichondritis is a bacterial infection of the overlying skin and perichondrium of the ear, by definition sparing the auricular cartilage. Causative organisms include P aeruginosa, S aureus, and S pyogenes. Predisposing factors for auricular perichondritis include surgery, ear piercing, burns, frostbite, insect bites, and contact sports. Auricular perichondritis may be an extension of OE. Clinical findings include a swollen, tender, erythematous, and warm auricle, which may involve the ear lobule, and often a fever; the TM is unaffected. Infectious perichondritis may be confused with relapsing polychondritis, an autoimmune condition involving the cartilage of the ears, nose, and trachea.

Management and Disposition

Outpatient management of the healthy patient includes appropriate oral antibiotics and follow-up in 48 hours. Hospitalization for parenteral antibiotics in the immunocompromised or diabetic patient is advised. Topical antibiotic otic drops should be used if the external canal is involved. Relapsing polychondritis is treated as an outpatient with prednisone if involvement is confined to the ear.

Pearls

1. P aeruginosa is the most common bacteria causing auricular perichondritis.
2. Ear piercing is the most common activity resulting in auricular perichondritis.
3. Findings of fluctuance and auricular deformity suggest auricular chondritis, a complication of perichondritis.



FIGURE 5.19 ■ **Perichondritis.** Erythema, swelling, and tenderness seen in the pinna with multiple ear piercings. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.20 ■ **Perichondritis.** Erythema is seen surrounding the piercing sites of the ear of this patient in DKA. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.21 ■ **Auricular Chondritis.** Deformity of the auricular cartilage is seen in this patient with chondritis. Ear piercing caused the initial insult to this pinna. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.22 ■ **Relapsing Polychondritis.** Erythema of the right pinna that spares the lobe. A month prior, the left pinna was involved and was treated as infectious perichondritis. (Photo contributor: Lawrence B. Stack, MD.)

HERPES ZOSTER OTICUS (RAMSAY HUNT SYNDROME)

Clinical Summary

Herpes zoster oticus (HZO), or Ramsay Hunt syndrome, is the second most common cause of facial paralysis, representing 3% to 12% of cases. The syndrome consists of facial and neck pain, auditory symptoms, and facial palsy associated with the reactivation of latent varicella zoster virus in the facial nerve and geniculate ganglion. Patients first note pruritus, followed by pain out of proportion to the physical examination over the face and ear. Patients may also experience vertigo, hearing loss from involvement of the eighth CN, tinnitus, rapid onset of facial paralysis, decrease in salivation, loss of taste sensation over the posterolateral tongue, and vesicles on the ear, EAC, and face.

Management and Disposition

The diagnosis of HZO is based largely on history and physical examination. Tzanck preparations may be difficult because of the vesicles' location. Magnetic resonance imaging (MRI) with contrast, if performed, may show enhancement of the geniculate ganglion and facial nerve.

Oral antivirals and steroids are mainstays of treatment. It is important to protect the involved eye from corneal abrasions and ulcerations by using lubricating drops. Referral to a specialist should be made for follow-up care.

Pearls

1. The prognosis for facial paralysis due to HZO is worse than that for Bell palsy. Approximately 10% and 66% of patients with full and partial facial paralysis, respectively, recover fully. The prognosis improves if the symptoms of HZO are preceded by the vesicular eruption.
2. The combination of antivirals with steroids produces better outcomes than with either agent alone.

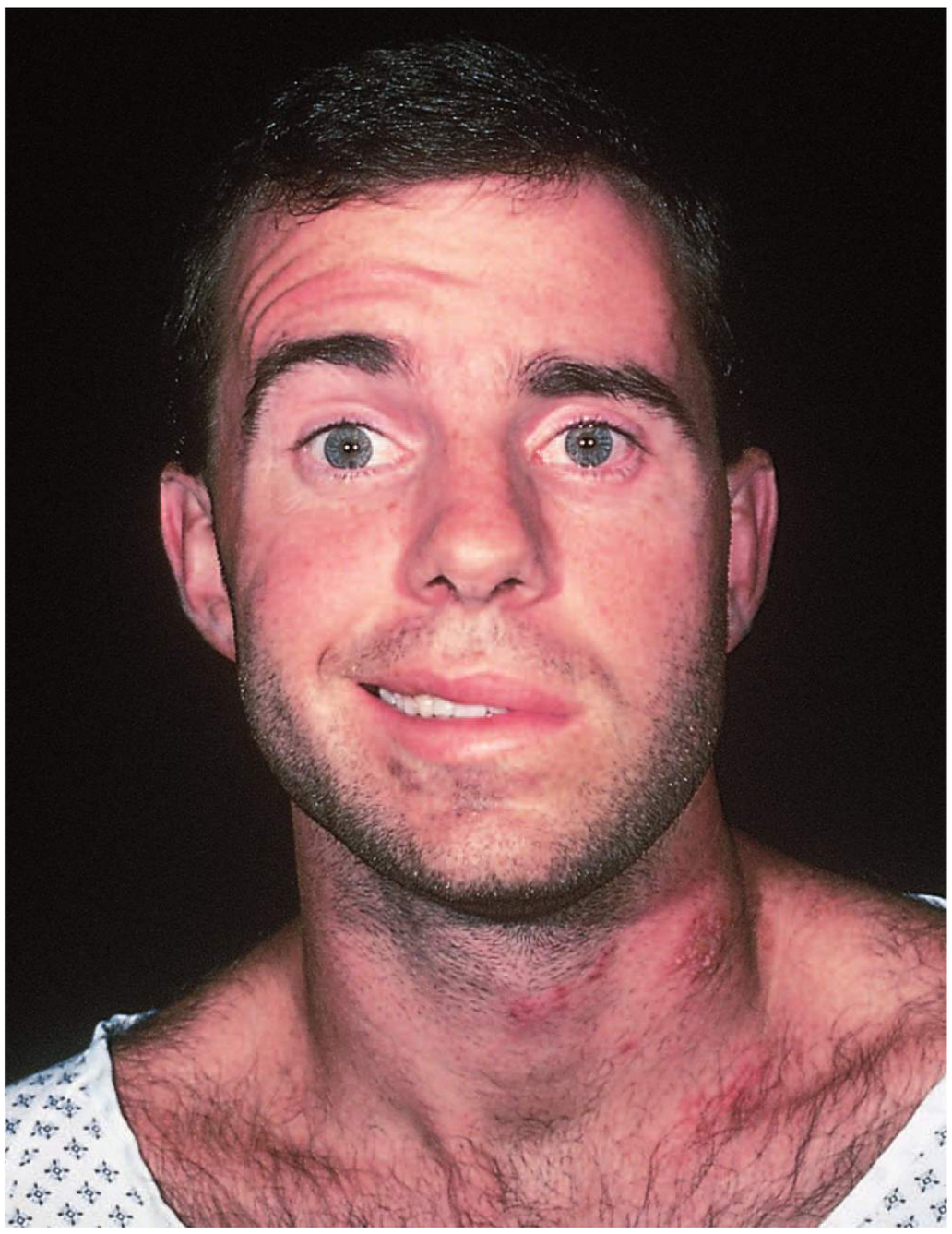


FIGURE 5.23 ■ **Herpes Zoster Oticus.** Facial palsy in a young adult. Note the vesicular eruptions on the neck. (Photo contributor: Frank Birinyi, MD.)



FIGURE 5.24 ■ **Herpes Zoster Oticus.** On closer examination, the vesicles extend up the neck to the external auditory canal. (Photo contributor: Frank Birinyi, MD.)



FIGURE 5.25 ■ **Herpes Zoster Oticus.** Erythema and drainage coming from the EAC is seen in this patient with herpes zoster oticus. Otitis externa can have a similar appearance but does not have vesicles, as seen in this patient. (Photo contributor: David Effron, MD.)



FIGURE 5.26 ■ **Herpes Zoster Oticus.** Vesicles of zoster are clearly seen in this patient. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

CN VII innervates the facial muscles via the five branches of the motor root, the submandibular, sublingual, and lacrimal glands, and the taste organs on the anterior two-thirds of the tongue; and it provides sensation to the pinna of the ear. Facial palsies are either central or peripheral. Central lesions occur proximal to the CN VII nucleus in the pons. Lesions distal to the nucleus are classified as peripheral lesions. The ipsilateral frontalis muscle is functional or "spared" in central lesions, since it receives innervation in the nucleus from both ipsilateral and contralateral motor cortices. Peripheral injuries involve the entire side of the face, including the forehead; thus, the forehead is not "spared."

Most commonly, seventh-nerve dysfunction is idiopathic (Bell palsy). One percent of patients have bilateral involvement; 60% have a viral prodrome. There is no age, sex, or racial predilection. Patients note an acute onset of facial weakness and may have numbness or pain on the ipsilateral face, ear, tongue, and neck as well as a decrease or loss of ipsilateral tearing and saliva flow. Hearing is preserved.

Prognosis is variable. Facial weakness has a better prognosis for full recovery than complete paralysis. Palsies due to herpes zoster have a protracted course, and many do not fully resolve. In comparison, 80% of patients with Bell palsy completely recover within 3 months.

Management and Disposition

Initial examination should include a thorough examination of the ear (including sensorineural or conductive hearing loss), the eye (including lacrimation), and the CNs—especially extraocular muscles (EOMs). Motor function of the seventh CN is evaluated by having the patient raise their eyebrows, smile, pucker, and frown. No single laboratory test is diagnostic. Screening CT or MRI of the head is of little value in the absence of additional findings on physical examination.

Initiate steroids within 72 hours of symptom onset for age >16 years and antivirals with steroids if viral etiology suspected. Eye lubricants and taping the eye shut at night help prevent keratitis and ulceration. Referral to a neurologist should be made for follow-up care.

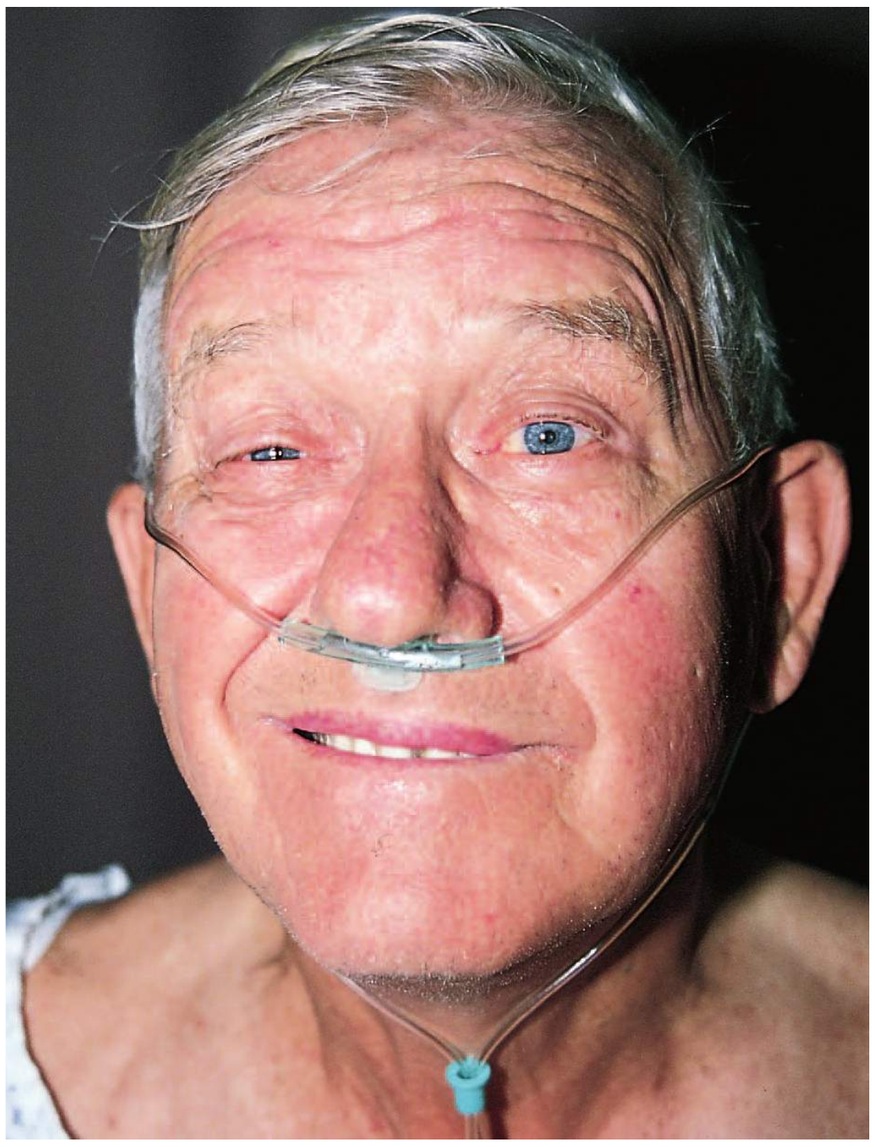


FIGURE 5.27 ■ **Central Seventh-Nerve Palsy.** Central facial nerve paralysis with forehead sparing. (Photo contributor: Frank Birinyi, MD.)

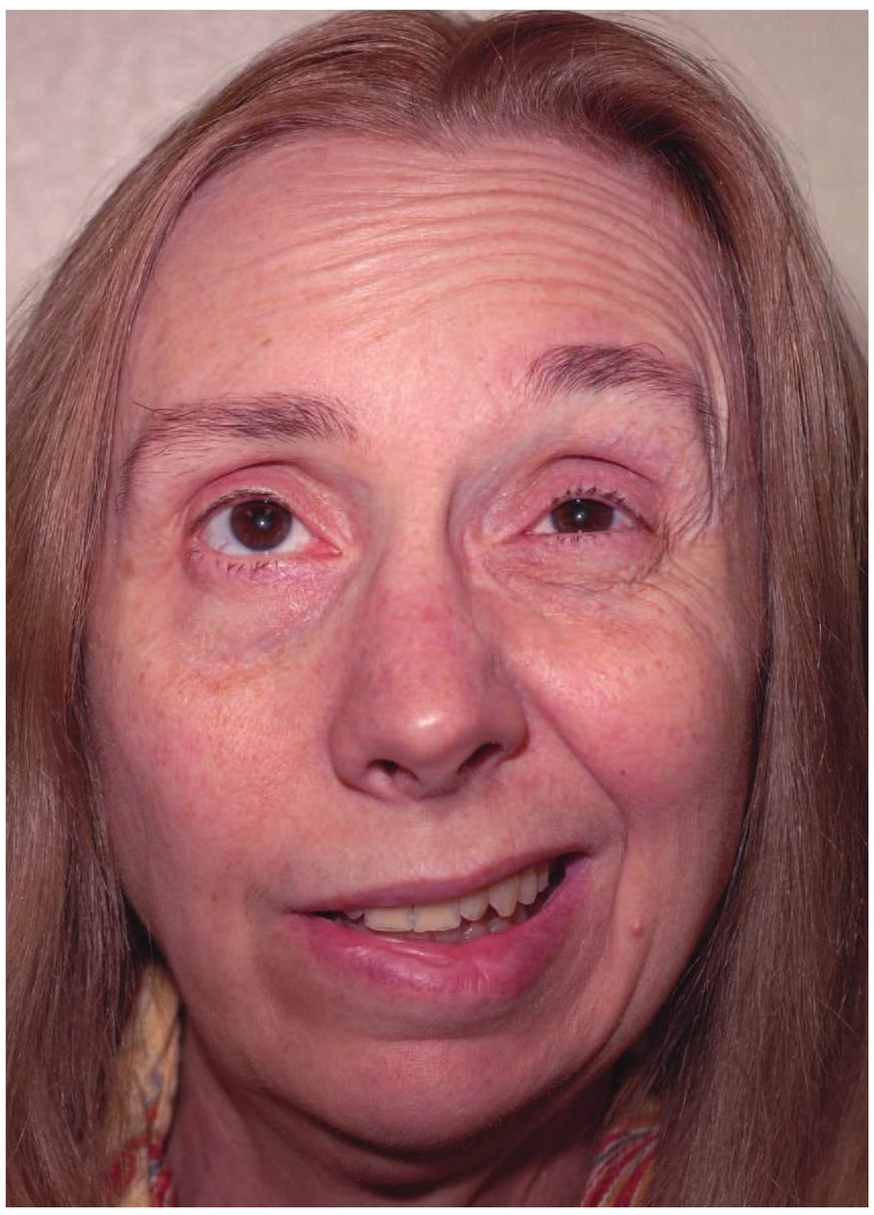


FIGURE 5.28 ■ **Peripheral Seventh-Nerve Palsy.** A peripheral nerve paralysis involving the entire ipsilateral face, including the forehead, is seen in this patient with Bell palsy. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.29 ■ **Peripheral Seventh-Nerve Palsy.** Forehead muscle paralysis is clearly apparent in this patient with Bell palsy. (Photo contributor: Suzanne Dooley-Hash, MD.)

Pearls

1. Facial nerve paralysis is a symptom, not a diagnosis.
2. If a provisional diagnosis of Bell palsy is made and no resolution of symptoms occurs, the diagnosis must be reconsidered. In patients misdiagnosed with Bell palsy, tumors are the most common missed etiology. MRI of the brainstem and internal auditory canal is indicated in these circumstances.
3. The finding of CN VI (lateral rectus) palsy along with CN VII palsy is diagnostic of a CN VI stroke, which involves the ipsilateral CN VII as it partially surrounds the CN VI nucleus. Hence, always evaluate for CN VI palsy when evaluating CN VII palsy.
4. Further ED evaluation is required for facial palsy presenting with sparing of the ipsilateral frontalis muscle as this represents a central lesion that may be caused by neoplasm or stroke.

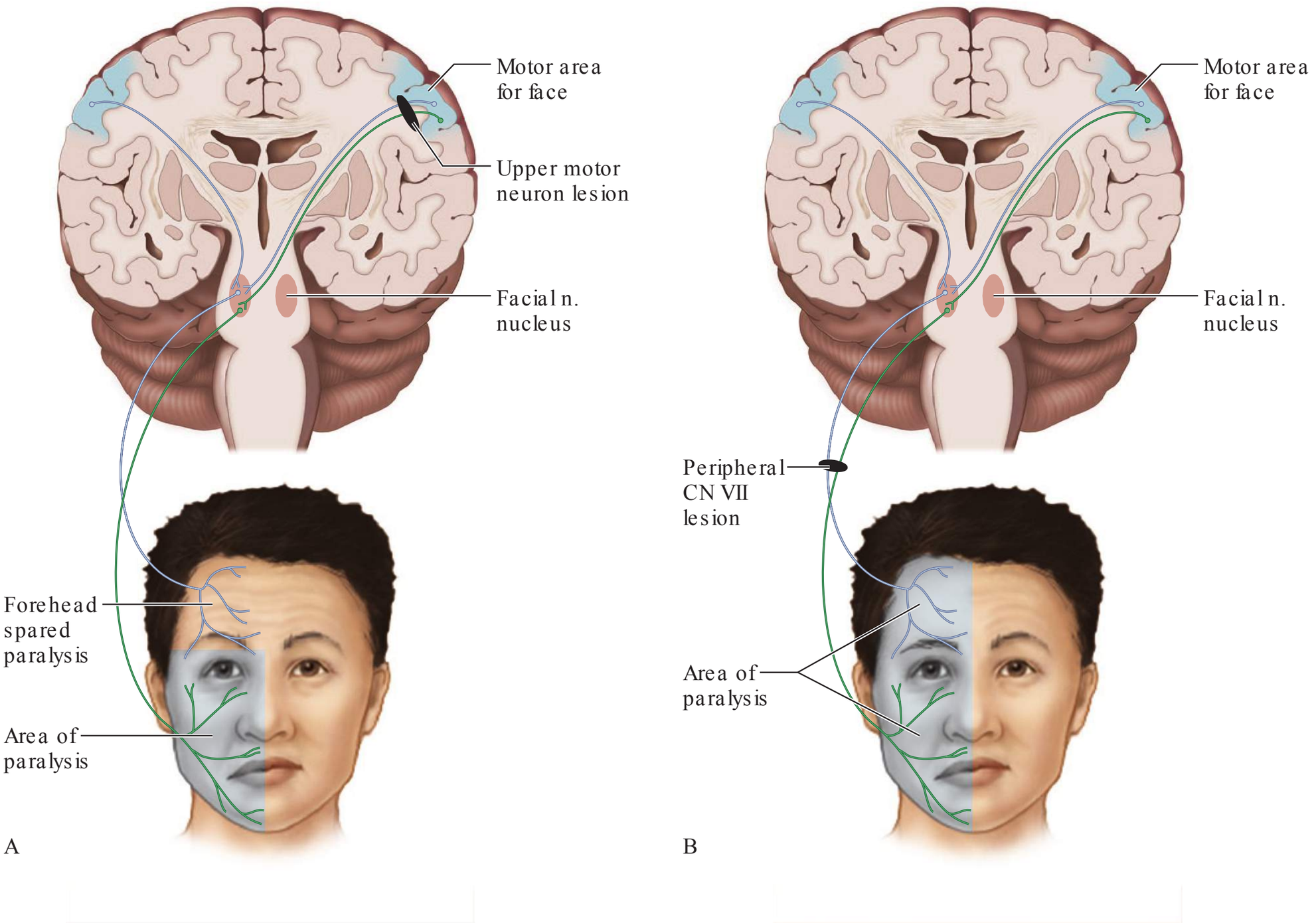


FIGURE 5.30 ■ **Cranial Nerve VII Neuropathways.** (**A**) Central CN VII Lesion. Crossed fibers allow forehead sparing in an upper motor neuron lesion identifying this presentation as a CVA vs a Bell palsy. (**B**) Peripheral CN VII nerve lesion causes paresis of both forehead and lower facial muscles.

Clinical Summary

Angioedema is clinically characterized by acute onset of well-demarcated cutaneous swelling of the face, lips, and tongue; edema of the mucous membranes of the mouth, throat, or abdominal viscera; or nonpitting edema of the hands and feet (often asymmetric). It is hereditary, allergic, or idiopathic. Hereditary angioedema is an autosomal dominant trait associated with a deficiency of serum inhibitor of the activated first component of complement (C1). Allergic angioedema can result from medications or contrast agents, environmental antigens, or local trauma. Complications range from dysphagia and dysphonia to respiratory distress, airway obstruction, and death. Angiotensin-converting enzyme (ACE) inhibitor–induced angioedema has a predilection for involvement of the lips, face, tongue, and glottis, and like hereditary angioedema, is often refractory to medical therapy.



FIGURE 5.31 ■ **Angioedema.** Severe angioedema of the face and tongue requiring emergent cricothyroidotomy. (Photo contributor: W. Brian Gibler, MD.)

Management and Disposition

Airway protection remains the primary focus of emergency treatment. Frequent reassessment and early airway management is mandatory as deterioration due to edema formation can be rapid.

Medical therapy includes steroids, H_1 and H_2 histamine blockers, and subcutaneous or intramuscular epinephrine. Chronic angioedema responds better to corticosteroids and H_2 blockers. Hereditary angioedema may be treated with C-1 esterase inhibitors (eg, Cinryze).

Disposition depends on the severity and resolution of symptoms. Patients with symptomatic improvement or showing no worsening after 4 hours of observation may be discharged home. Discontinue suspect medications. Airway involvement requires admission to a monitored environment with surgical airway equipment at the bedside.

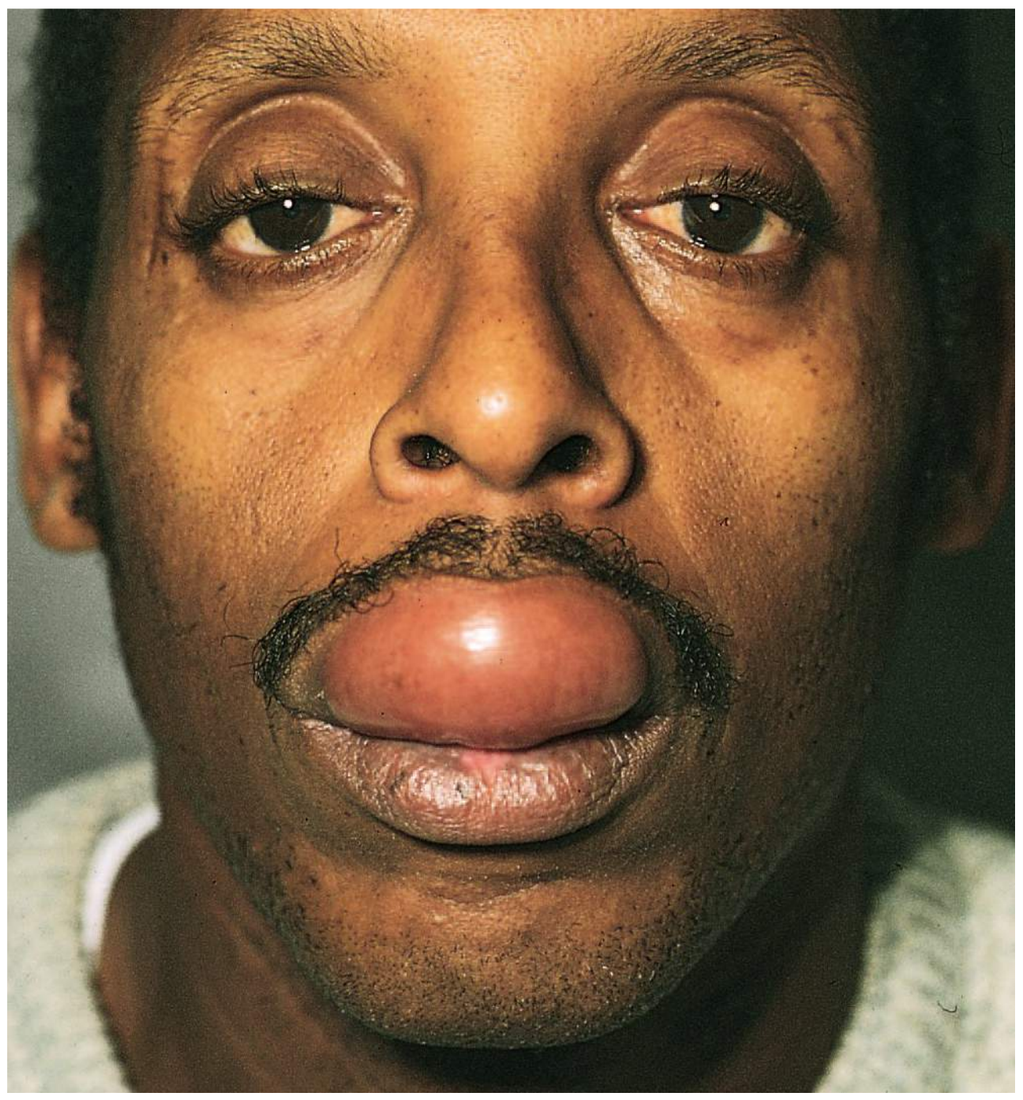


FIGURE 5.32 ■ **ACE Inhibitor–Induced Angioedema.** Angioedema of the upper lip in a man who had been taking an ACE inhibitor for 2 years. The patient had no previous episodes. (Photo contributor: Kevin J. Knoop, MD, MS.)

Pearls

1. Do not underestimate the degree of airway involvement; act early to preserve airway patency.
2. Angioedema can also cause gastrointestinal and neurologic involvement.
3. Early response to medical intervention does not preclude rebound of symptoms to a greater extent than at presentation.
4. Patients who have been using ACE inhibitors for months or years can still develop angioedema from these agents.



FIGURE 5.33 ■ **ACE Inhibitor–Induced Angioedema.** Unilateral tongue swelling in a man who had been taking ACE inhibitors for 5 years. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.34 ■ **Angioedema.** Severe lip swelling in a patient with hereditary angioedema. (Photo contributor: Clay B. Smith, MD.)

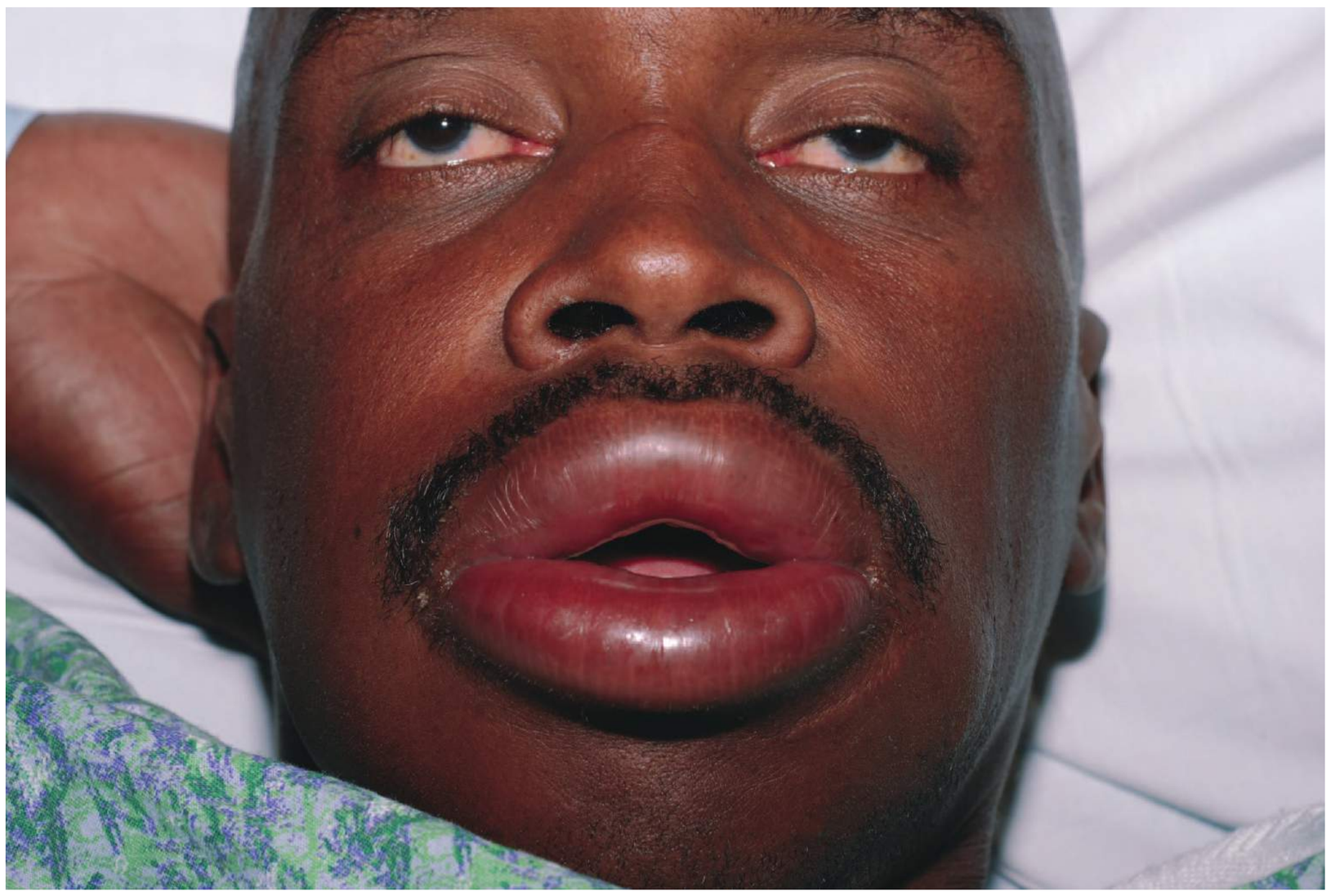


FIGURE 5.35 ■ **Angioedema.** Severe lip swelling from an unknown cause. (Photo contributor: Clay B. Smith, MD.)

Clinical Summary

Pharyngitis is an inflammation and commonly an infection of the pharynx and its lymphoid tissues. Viral causes account for 90% of all cases. Group A β-hemolytic streptococci (GABHS) is responsible for up to 50% of bacterial infections. Other bacterial causes include other streptococci, Mycoplasma pneumoniae, Neisseria gonorrhea, and Corynebacterium diphtheriae. In immunocompromised patients and patients on antibiotics, Candida species can cause thrush. Sore throats that last longer than 2 weeks should increase suspicion for either a deep-space neck infection or a neoplastic cause.

Patients with bacterial (especially GABHS) pharyngitis present with an acute onset of sore throat and fever, frequently accompanied by nausea, vomiting, headache, and abdominal cramping. They may have a mild to moderate fever, an erythematous posterior pharynx and palatine tonsils, tender cervical lymphadenopathy, and palatal petechiae. Classically, the tonsils have a white or yellow exudate with debris in the crypts; however, many patients do not have exudate on examination. Viral pharyngitis is typically more benign, with a gradual onset, less fever, and less impressive erythema and swelling of the pharynx. Except for infectious mononucleosis, which can take weeks to resolve, most cases of viral pharyngitis are self-limited, with spontaneous resolution in a matter of days. Lingual and adenoid tonsillitis may also be present.



FIGURE 5.36 ■ **Palatal Petechiae.** Palatal petechiae and erythema of the tonsillar pillars in a patient with streptococcal pharyngitis. (Photo contributor: Kevin J. Knoop, MD, MS.)

Management and Disposition

Treatment is largely supportive except for antibiotics and rehydration. Analgesics, antipyretics, and throat sprays or gargles can provide symptomatic relief. Patients with known or suspected GABHS require antibiotics primarily to prevent rheumatic fever and suppurative complications. The Centor criteria are clinical decision rules developed to guide physicians in testing and prescribing of antibiotics. Criteria include: (1) tonsillar exudates, (2) tender anterior cervical adenopathy, (3) fever by history, and (4) absence of cough. Patients with fewer than two criteria should not receive either antibiotic treatment or diagnostic testing. Most authorities now favor evaluation using a sensitive rapid streptococcal antigen test (RSAT) for identification of GABHS, without throat culture for negative results, in adult patients with two or more Centor criteria. However, in children it is recommended that all negative RSAT be followed up with a throat culture. Current first-line antibiotic therapies remain a single dose of intramuscular benzathine penicillin or oral penicillin for 10 days.

Pearls

1. Other manifestations of pharyngitis should be sought in patients with sore throat. Sandpaper rash, Pastia lines, and desquamation are suggestive of scarlet fever. Hepatomegaly and/or splenomegaly should raise suspicion for infectious mononucleosis.
2. Sore throat or pharyngitis that lasts more than 2 weeks must be referred for further evaluation to rule out possible neoplastic or neurologic causes, especially in patients over 50 years old who have a smoking or chewing tobacco history.
3. Amoxicillin should be avoided if infectious mononucleosis is a possibility, as a morbilliform eruption will occur in up to 80% of patients.
4. Pharyngitis itself may be a prodrome for other pathologic conditions, such as measles, scarlet fever, and influenza.



FIGURE 5.37 ■ **Exudative Pharyngitis.** Intense erythema with scant exudates is seen in this early (<24 hours) case of GABH pharyngitis. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 5.38 ■ **Tonsillar Exudate.** White and yellow cryptic exudates are seen in this patient with rapid strep test proven streptococcal pharyngitis. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.39 ■ **Infectious Mononucleosis.** Nearly touching exudative tonsils in a patient with elevated liver transaminases and hepatosplenomegaly. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.40 ■ **Viral Pharyngitis.** Intense erythema of the uvula and tonsillar pillars and ulcerations are typical of viral pharyngitis. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.41 ■ **Inflammatory Pharyngitis.** Inflammation and ulcerations of the uvula and soft palate seen in this adolescent patient with a Crohn disease flare-up. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.42 ■ **Hemorrhagic Pharyngitis.** Hemorrhagic tonsils, palatal petechiae, and pinch purpura of the buccal mucosa are seen in this patient with infectious mononucleosis-induced immunologic thrombocytopenic purpura. (Photo contributor: Sheila McMorrow-Jones, MD.)

Clinical Summary

Septal hematoma, with or without abscess formation, is an uncommon complication of septal surgery or direct nasal trauma. Bleeding from submucosal blood vessels leads to an accumulation of blood between the mucoperichondrium and the septal cartilage, which may lead to ischemic avascular necrosis of the underlying cartilage, destruction of the cartilage, and saddle nose deformity (see Fig. 1.13). The hematoma and any necrotic cartilage may then serve as a nidus for infection, resulting in a septal abscess. Chronic cocaine use results in vasoconstriction and eventual necrosis of the septal cartilage leading to perforation.

Septal injuries may lead to cosmetic nasal deformity, chronic sinus infections, recurrent epistaxis, and sleep disturbances. Rarely, it can result in more serious complications such as cavernous sinus thrombosis and meningitis. Since the original trauma is often minor, patients may present days to weeks after the injury. Young children and infants may present with poor feeding, fever, and rhinorrhea. Older children and adults may note bleeding, headache, and more focal pain.

Examination reveals a large, red, round swelling originating off the septum and occluding most of the nasal cavity. The mass is tender to palpation and may cause the outer aspects of the nose to be tender as well. Septal abscesses tend to be more painful and larger than uncomplicated hematomas. Fever is frequently present. S aureus, group A β-hemolytic streptococcus, H influenzae, and S pneumoniae are the organisms most commonly isolated in septal abscesses. A septal perforation is easily seen with penlight examination.



FIGURE 5.43 ■ **Septal Hematoma.** Fluctuant grapelike structure in the right naris after blunt facial trauma consistent with a septal hematoma. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.44 ■ **Septal Abscess.** A septal abscess is seen in both nares 1 week after blunt nasal trauma. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Suspicion and recognition are essential in diagnosing septal injuries. Prompt referral to an otolaryngologist is mandatory for incision and drainage of the hematoma or abscess.

Pearls

1. Intranasal examination for septal hematoma in all patients with a history of nasal trauma regardless of severity is crucial.
2. Antibiotics are required in septal hematomas with a clinical suspicion for a secondary infection or abscess.
3. The physician must explore the possibility of child abuse in young children and infants with a septal hematoma or abscess.

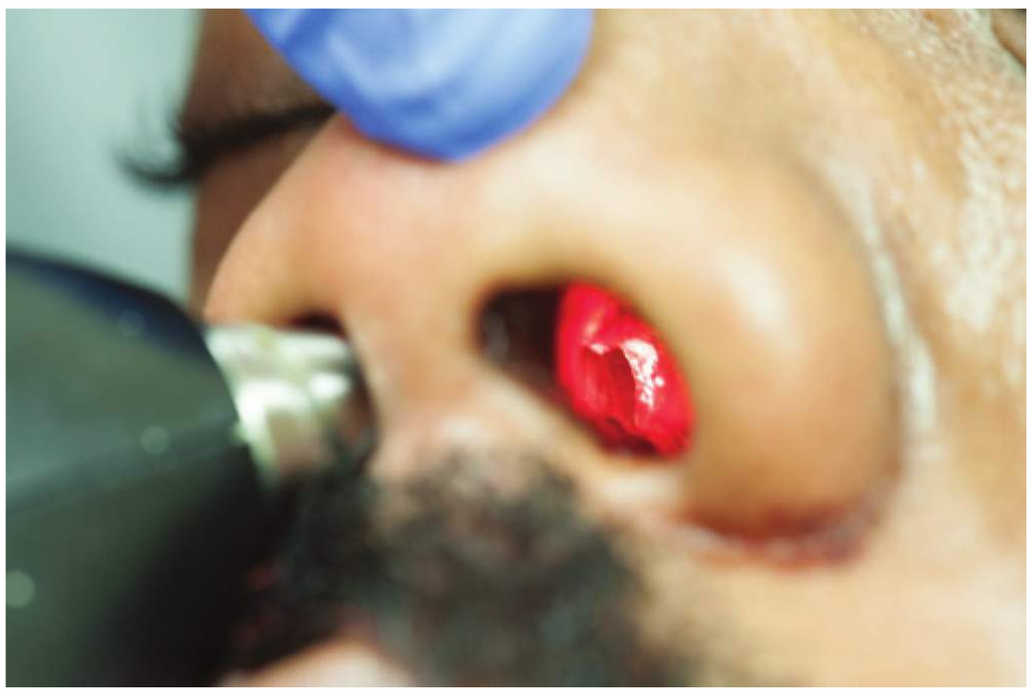


FIGURE 5.45 ■ **Septal Perforation**. A septal perforation is illuminated with a light in a chronic cocaine user. (Photo contributor: Lawrence B. Stack, MD.)

NASAL CELLULITIS

Clinical Summary

Nasal cellulitis is an infection of the skin and subcutaneous tissues, and does not include nasal cartilage. It is most common at the extremes of age. Bacterial invasion due to disruption of the skin is the usual cause. S pyogenes and S aureus cause most infections. Risk factors include nasal surgery, instrumentation, diabetes, immunodeficiency, and nasal piercing. Clinical features include pain, redness, and swelling of the nasal tissues. Headache, fever, and malaise suggest complicated disease. Complications of nasal cellulitis include abscess, cavernous sinus thrombosis, chondritis of the nasal cartilage, bacteremia, and sepsis.

Management and Disposition

The diagnosis of nasal cellulitis is clinical. However, evaluation may include complete blood count, blood and tissue cultures, contrasted CT if abscess is suspected, and CT-V if a cavernous sinus thrombosis is suspected. Remove any foreign body that might be a nidus of infection. Amoxicillin-clavulanate or amoxicillin-sulbactam is first-line antibiotics. Clindamycin, vancomycin, trimethoprim-sulfamethoxazole, and first-generation cephalosporins are other options. Patients should be hospitalized if they have systemic symptoms, are immunocompromised, have diabetes or a suspected abscess, or have retained foreign bodies. Patients at extremes of age should be strongly considered for admission.

Pearls

1. Threshold for hospital admission for patients with nasal cellulitis should be low.
2. In children, consider H influenza and S pneumoniae as causative organisms of facial cellulitis.
3. Patients with pain at the nasal entrance but no overt physical findings of cellulitis may have vestibulitis, responsive to topical antibiotics (mupirocin).



FIGURE 5.46 ■ **Nasal Cellulitis.** Swelling and erythema of the nose in a middle-aged man. A contrasted CT was done to exclude abscess because of the marked swelling of the nasal septum. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.47 ■ **Nasal Cellulitis.** Swelling and erythema of the nose of a 2-year-old child, suggesting nasal cellulitis. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.48 ■ **Nasal Cellulitis.** Swelling and erythema of soft tissue surrounding the internal and external sides of nasal cartilage seen in early nasal cellulitis. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.49 ■ **Nasal Cellulitis.** Intense erythema, swelling, and drainage consistent with MRSA abscess and cellulitis of the nose. (Photo contributor: Lawrence B. Stack, MD.)

DIPHTHERIA

Clinical Summary

Diphtheria is a highly contagious disease caused by the exotoxin-producing bacterium C diphtheriae. It is transmitted either by direct contact or through respiratory aerosolization. Many adults are now susceptible to diphtheria because their vaccine-induced immunity decreased over time or owing to decreased opportunity for naturally acquired immunity.

Diphtheria may involve any mucous membrane; but most commonly, it affects the mucosa of the upper respiratory tract. It typically produces an ulcerated pharyngeal mucosa with a white-to-gray inflammatory pseudomembrane, classically with a "wet mouse" odor. Patients present with symptoms, in order of frequency, of fever, sore throat, weakness, pain with swallowing, change in voice, loss of appetite, neck swelling, difficulty breathing, and nasal discharge.

While the organism remains localized to the mucosa, hematogenous spread of the exotoxin typically produces myocarditis or peripheral neuropathies. Deaths from diphtheria occur from either tracheobronchial obstruction by the pseudomembrane acutely or cardiac complications several weeks after the primary infection.

Management and Disposition

The diagnosis is initially made clinically and confirmed by successful isolation and toxigenicity testing of C diphtheriae.

Antitoxin, available from the Centers for Disease Control and Prevention (CDC), is the mainstay of therapy and must be given even before laboratory confirmation. Erythromycin or penicillin given promptly when diphtheria is suspected has been shown to decrease both exotoxin production and spread of the bacterium.

Patients require hospital admission for observation of airway obstruction, pulmonary support, and intravenous hydration and antibiotics. Strict isolation is essential.

Pearls

1. Outcome is improved with early treatment; thus, the diagnosis of diphtheria must be made clinically and treatment begun empirically before bacteriologic confirmation.
2. Patients with a membranous pharyngitis need to be questioned regarding immunization, exposures, and recent travel.
3. All contacts should have a booster dose of vaccine (TD or Td, depending on age), while nonimmune contacts should also be given prophylactic antibiotics after a throat swab.



FIGURE 5.50 ■ **Diphtheria Pharyngitis.** An exudative pharyngitis with a gray pseudomembrane is seen in this patient with diphtheria. (Photo contributors: Dileep C. Unnikrishnan, MD, and Nadeem Kocheri, MD.)

Clinical Summary

Peritonsillar abscess (PTA), or quinsy, is the most common deep neck infection. Although most occur in young adults, immune compromised and diabetic patients are at increased risk. Most abscesses develop as a complication of tonsillitis or pharyngitis, but may also result from odontogenic spread, recent dental procedures, and local mucosal trauma.

The pathogens involved are similar to those causing tonsillitis, especially streptococcal species, but many infections are polymicrobial and involve anaerobic bacteria. Patients present with a fever, severe sore throat that is often out of proportion to physical findings, localization of symptoms to one side of the throat, trismus, drooling, dysphagia, dysphonia, fetid breath, and ipsilateral ear pain.

During the early stages, the tonsil and anterior pillar are erythematous, appear full, and may be shifted medially. Later, the uvula and soft palate are shifted to the contralateral side. The tonsillar pillar may feel fluctuant and tender.

Management and Disposition

Most patients can have needle aspiration performed as the sole surgical drainage procedure and expect a satisfactory outcome. Alternative surgical drainage procedures—including incision and drainage and abscess tonsillectomy—can be performed by an otolaryngologist or oral surgeon. Most are managed as outpatients on oral antibiotics following drainage. Patients who are immunocompromised, have airway involvement, appear toxic, or cannot tolerate oral intake require admission for rehydration, parenteral antibiotics, and specialty consultation.

Pearls

1. The value of culturing aspirates is questionable, with a review of several studies showing no clinical benefit from the cultures unless the patient is immunocompromised.
2. ED ultrasound along with palpation of the mass is a valuable adjunct in confirming diagnosis and verifying landmarks prior to aspiration.
3. A contrasted CT scan of the neck will confirm the presence of a PTA if the diagnosis is uncertain.



FIGURE 5.52 ■ **Peritonsillar Abscess.** Acute peritonsillar abscess showing medial displacement of the uvula, palatine tonsil, and anterior pillar. (Photo contributor: Lawrence B. Stack, MD)

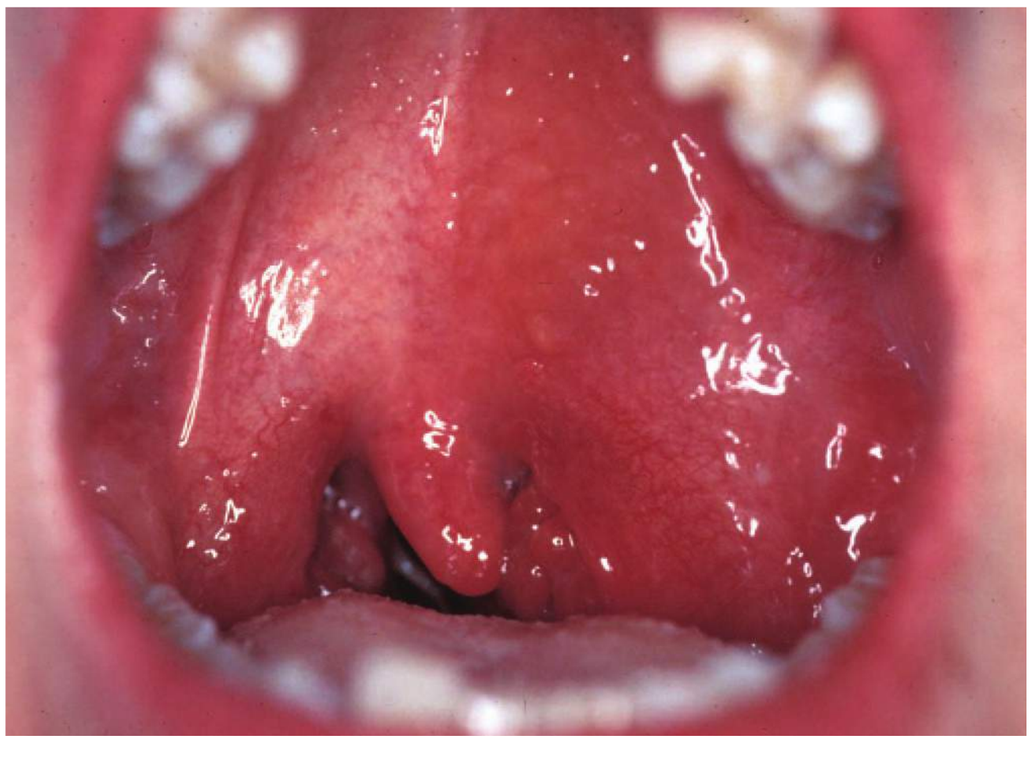


FIGURE 5.51 ■ **Early Peritonsillar Abscess.** Edema and marked erythema of the left tonsillar pillar in early peritonsillar abscess. (Photo contributor: Kevin J. Knoop, MD, MS.)

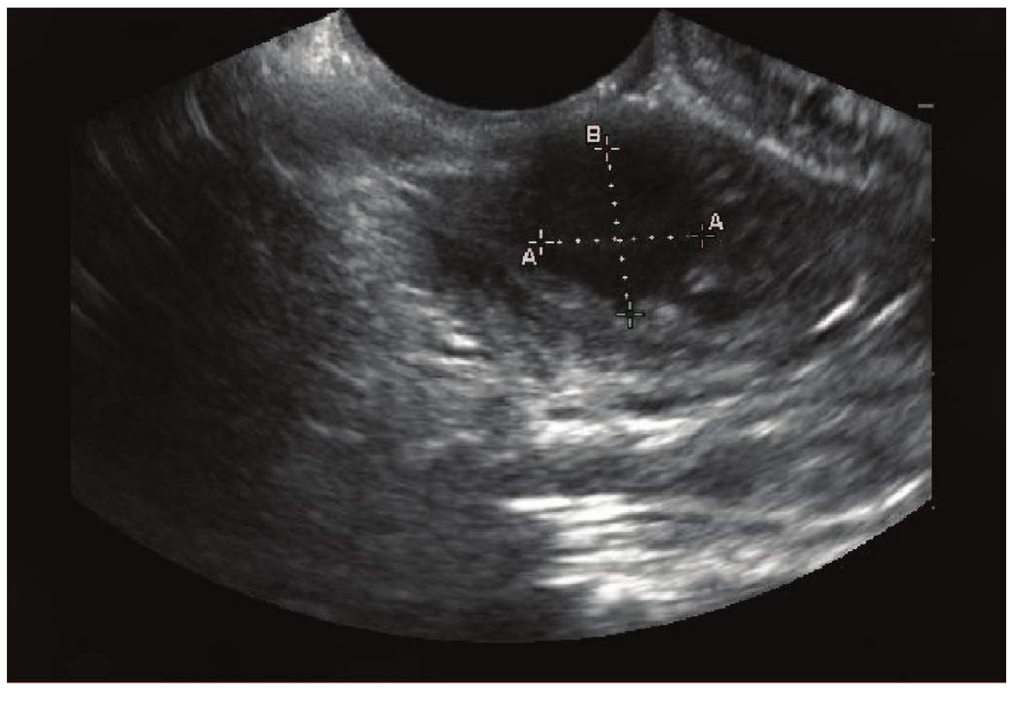


FIGURE 5.53 ■ **Peritonsillar Abscess—Ultrasound.** Ultrasound demonstrates size and location of abscess, in addition to confirming relationship to vascular structures. Over 2 cc of pus was aspirated from this abscess. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 5.54 ■ **Peritonsillar Phlegmon.** Marked erythema of the tonsillar pillars is seen in this patient currently on oral penicillin. No swelling or fluctuance is present. (Photo contributor: Lawrence B. Stack, MD.)

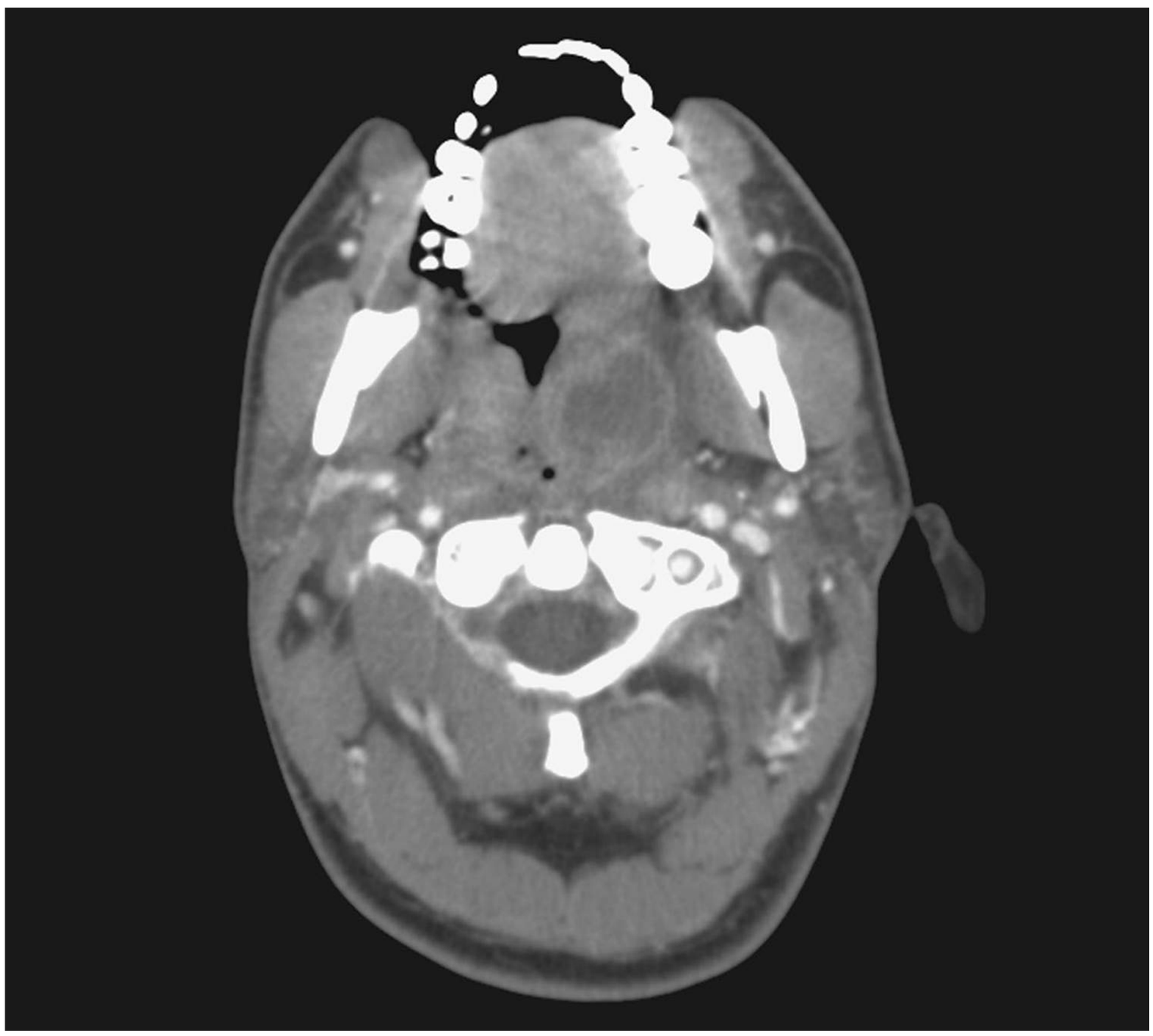


FIGURE 5.55 ■ **Peritonsillar Abscess—CT.** Ring-enhancing lesion with a hypodense core consistent with a left peritonsillar abscess. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Epiglottitis or supraglottitis is an infection of the epiglottis and adjacent tissues. Bacterial epiglottitis, a rare but potentially fatal infection, is caused primarily by H influenzae, but S pneumoniae, S aureus, and β-hemolytic streptococcus have also been isolated. The advent of the H influenzae B vaccination for infants has changed what used to be a disease primarily of children, with a peak age range from 2 to 6 years, to one occurring predominantly in adults. Bacterial epiglottitis occurs most commonly in the winter and spring.

Patients, especially children, with acute epiglottitis appear quite ill. They present with sore throat, fever, drooling, severe dysphagia, dyspnea, muffled or hoarse voice, and occasionally inspiratory stridor. Patients with severe respiratory distress assume the "tripod" position: sitting upright with the neck extended, arms supporting the trunk, and the jaw thrust forward. This position maximizes airway patency and caliber. Adults typically have an indolent course with a prodromal viral illness, but many children have a sudden onset and rapid progression to respiratory distress.

Management and Disposition

Airway management is paramount. Even prior to diagnosis, children should be calmed, comforted by a parent, and allowed to assume whatever position they feel is most comfortable. Anesthesiology and ENT should be consulted immediately. Indications for intubation are clinical, but severe stridor and respiratory distress are clear reasons to intervene. Nasotracheal intubation over a flexible endoscope is preferred. Needle cricothyrotomy can provide temporary oxygenation until a surgical airway is provided.

Radiographs of the neck may reveal the classic "thumb" sign, a thickened epiglottis on the lateral soft-tissue neck radiograph. Visualization of the epiglottis is possible in the stable adult patient via direct and indirect laryngoscopy and fiberoptic nasopharyngoscopy. In children, the top of the swollen epiglottis may be visualized on careful oral examination, whereas pharyngoscopy is typically reserved for an experienced anesthesiologist or otolaryngologist in a controlled setting.

The mainstay of epiglottitis treatment is antibiotic therapy. Third-generation parenteral cephalosporins, ampicillin with sulbactam or trimethoprim-sulfamethoxazole, have proven efficacy in treating epiglottitis. Steroids or epinephrine, either nebulized or subcutaneous, may provide some improvement in edema.

In addition to airway compromise, complications of epiglottitis include epiglottic abscess, meningitis, pulmonary edema, pneumonia, and empyema (associated with H influenzae).

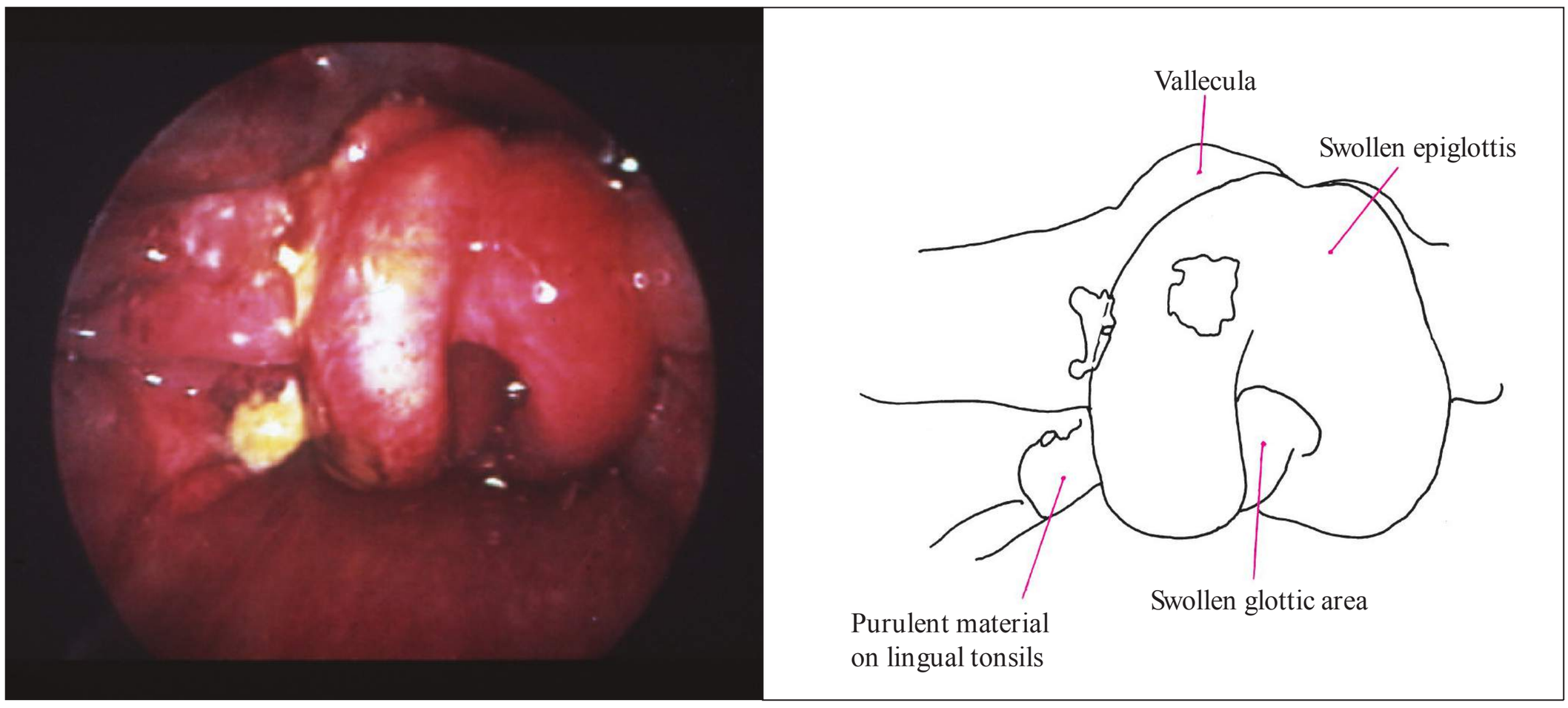


FIGURE 5.56 ■ **Adult Epiglottitis.** Fiberoptic laryngoscopy showing an edematous epiglottis and glottic area with marked airway compromise in an adult with epiglottitis. (Photo contributor: Timothy L. Smith, MD.)

Pearls

1. Transport of patients with suspected epiglottitis must be done by an experienced transport team. The airway must be secured before transport of all but the most stable patients.
2. During intubation, pushing on the patient's chest may cause a bubble to form at the airway orifice, guiding placement of the tube.
3. In areas with a significant prevalence of infection with community-associated methicillin-resistant S aureus (MRSA), clindamycin should be considered as part of the empiric choice for gram-positive coverage.
4. Failure to intervene prior to loss of the airway carries a six-fold increase in mortality.



FIGURE 5.57 ■ **Adult Epiglottitis.** Soft-tissue lateral neck radiograph of an adult with epiglottitis demonstrating the classic "thumb" sign of a swollen epiglottis. (Photo contributor: Kevin J. Knoop, MD, MS.)

Clinical Summary

Except for idiopathic, the two most common causes of uvular enlargement are infections and angioedema. Most patients complain of a sore throat, a gagging sensation, or a foreign-body sensation in the back of the mouth.

The infectious etiologies of uvulitis are bacterial, including H influenzae and streptococci; fungal, such as C albicans; and viral. Infections are typically extensions from adjacent infections, such as epiglottitis, tonsillitis, PTAs, and pharyngitis. Patients note fever, odynophagia, trismus, facial pain, hoarseness, neck pain, and headache. On examination, the uvula is red, firm, swollen, and very tender to palpation.

Angioedema of the uvula, known as the Quincke disease, can be hereditary, acquired, or idiopathic. Medications, allergens, thermal stimuli, pressure, and iatrogenic or accidental trauma can initiate angioedema. In addition to the swollen uvula, patients may note pruritus, urticaria, and wheezing. With uvular edema, the angioedema may also involve the face, tongue, and oropharynx. Airway compromise is more common in angioedema of the uvula which appears pale, boggy, and edematous, resembling a large white grape (uvular hydrops).

Management and Disposition

Most cases of uvulitis are benign and self-limited. Angioedematous uvulitis is treated with steroids, antihistamines, and epinephrine in severe cases, either subcutaneously or nebulized. For infectious uvulitis, antibiotic coverage is dictated by the primary source of infection. Admission is based on severity of airway compromise and accompanying infections.

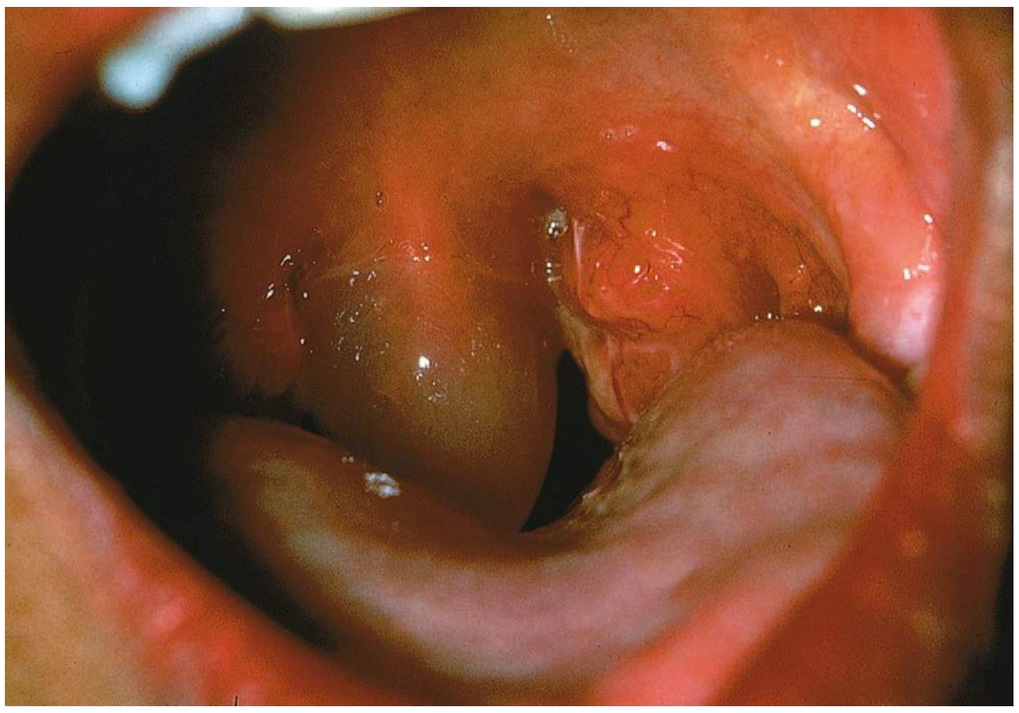


FIGURE 5.59 ■ **Uvulitis.** Angioedema of the uvula, known as the Quincke disease. (Photo contributor: Robin T. Cotton, MD.)

Pearls

1. Any airway symptom prompts an evaluation of the hypopharynx, by either radiographic imaging, or fiber-optic nasopharyngoscope, or direct laryngoscopy.

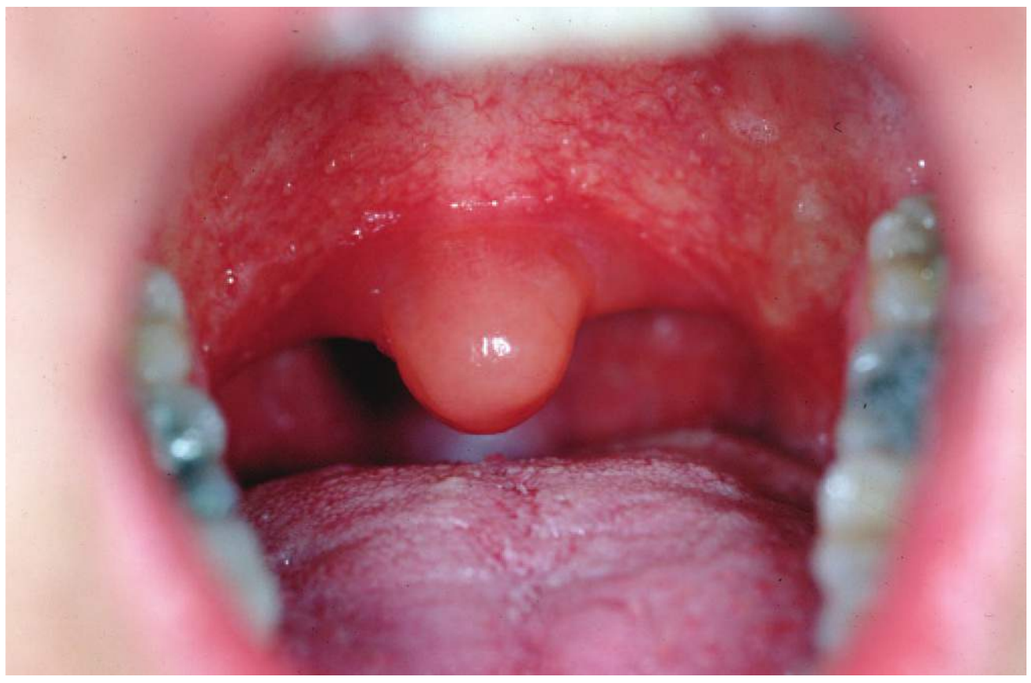


FIGURE 5.58 ■ **Uvulitis.** Isolated edema of the uvula in a patient who presents with a foreign-body sensation of the throat. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.60 ■ **Uvulitis.** Isolated edema of the uvula showing a globular structure that presents with foreign-body sensation. (Photo contributor: Lawrence B. Stack, MD.)

MUCOCELE (RANULA)

Clinical Summary

Ranulas are mucoceles (mucous retention cysts) that develop in the floor of the mouth, arising from obstructed sublingual or submandibular ducts or smaller minor salivary glands. Initially, the cysts are small and barely noticeable, but over time they can expand outward or deeper into the neck (plunging ranula). Large cysts can displace the tongue forward and upward, making the patient uncomfortable. Unlike those with sialolithiasis, patients with ranulas may not always notice an increase in swelling associated with eating. Physical examination reveals a soft, minimally tender, translucent cyst with dilated veins running over its surface. Unlike carcinomas, no ulceration is noted with ranulas, and they are generally softer.



FIGURE 5.61 ■ **Ranula.** Sublingual ranula, or mucocele, lateral to Wharton duct. The patient was asymptomatic except for being aware of the lesion. (Photo contributor: Kevin J. Knoop, MD, MS.)

Management and Disposition

Recognition by the physician is essential for proper referral. Definitive treatment is excision or marsupialization, although needle aspiration of the cyst can provide temporary relief. Unless there is a secondary infection, no antibiotic coverage is required.

Pearls

1. Most ranulas are painless and are incidental findings on routine examinations.
2. Ranulas often recur, requiring total excision of the offending salivary gland.



FIGURE 5.62 ■ **Mucocele.** Focal swelling of the upper lip that developed months after local trauma consistent with a mucocele. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Sialoadenitis is a general term describing inflammation of any salivary gland. The three major salivary gland pairs are the parotid, submandibular, and sublingual. There are also numerous smaller salivary glands that empty into the oral cavity and all are capable of becoming inflamed. Salivary gland disorders have a broad spectrum of causes, including acute and chronic infections; metabolic, systemic, and endocrine disorders; infiltrative processes; obstructions; allergic inflammation; and neoplastic diseases. Key features in the history are the duration and course of the symptoms, complaints of pain, and unilateral or bilateral location.

Both viral and bacterial infections of the salivary gland can lead to enlarged, swollen, painful masses. Suppurative sialoadenitis is most commonly caused by S aureus and is found in patients who are elderly, diabetic, or have poor oral hygiene. It may also follow episodes of dehydration, such as those due to surgery or debilitation. Viral sialoadenitis, such as mumps parotitis, is the most common cause. It occurs with a concomitant viral illness and is usually bilateral, whereas bacterial infections are primarily unilateral.

Obstructive sialoadenitis occurs from a stone or calculus in the salivary gland or duct, most commonly in the submandibular gland. The flow of saliva is obstructed, causing swelling, pain, and firmness. Patients with sialolithiasis note general xerostomia and recurrent worsening of swelling and pain during eating.

A thorough head and neck examination is essential, especially a bimanual examination of the major salivary glands. In suppurative sialoadenitis, purulent drainage may be expressed from the submandibular duct (Wharton) or parotid duct (Stensen) and the glands are very tender and painful to examination. Sialolithiasis can manifest as enlargement of the ducts with minimal saliva expressed on stripping and, rarely, a palpable or visible stone or duct thickening. Facial radiographs are of limited utility. Ultrasound or CT may be useful to detect abscesses.

Management and Disposition

Treatment of suppurative sialoadenitis requires antibiotics with coverage of Staphylococcus and oral flora, rehydration, proper oral hygiene, sialogogues, local heat, and occasionally surgical irrigation and drainage of abscesses. Obstructive sialoadenitis is rarely an emergency. Most salivary stones pass spontaneously without complication, and patients can be discharged home on lozenges to stimulate salivary secretions. Prompt follow-up of sialoadenitis is essential to prevent possible morbidity and mortality associated with infections or neoplasms.

Pearls

1. Examine secretions of both mouth and eyes, and elicit any history of dry eyes, keratoconjunctivitis, cutaneous lesions, or rheumatoid arthritis to establish the diagnosis of a systemic disorder.
2. Medications such as antihistamines, psychotropic drugs, and those possessing atropine-like side effects can cause xerostomia, exacerbating or predisposing to sialoadenitis.
3. Lack of improvement on antibiotics suggests an abscess or multiple loculated abscesses that require drainage.

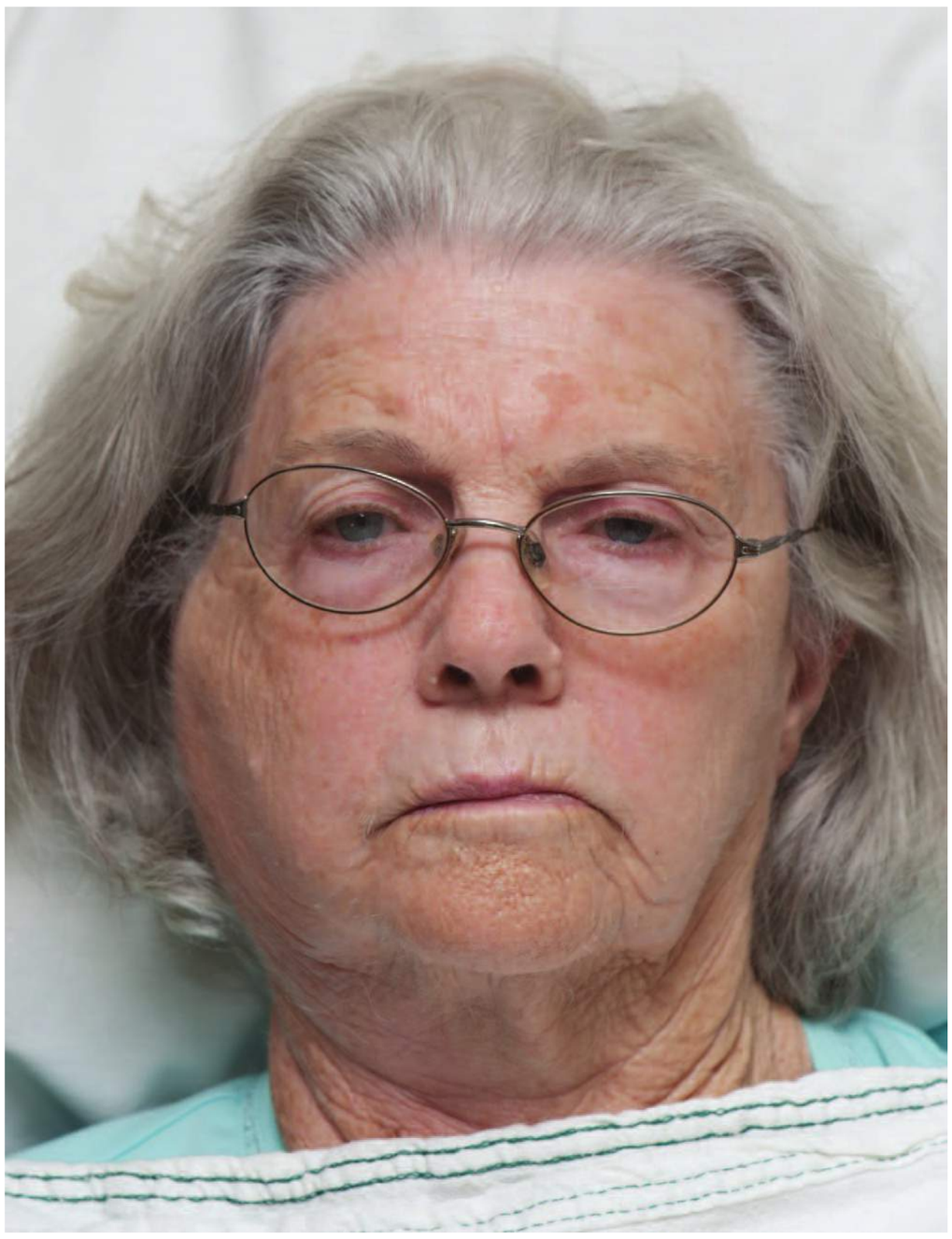


FIGURE 5.63 ■ **Suppurative Parotid Sialoadenitis**. Fever, swelling, and tenderness over the parotid gland along with purulent discharge expressed from Stensen duct suggest suppurative parotid sialoadenitis. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.64 ■ **Suppurative Parotid Sialoadenitis.** Pus from Stensen duct confirms suppurative parotid sialoadenitis. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.65 ■ **Suppurative Submandibular Sialoadenitis**. Unilateral submandibular swelling. (Photo contributor: Jeffery D. Bondesson, MD.)



FIGURE 5.66 ■ **Sialolithiasis.** A stone is seen at the orifice of Wharton duct. Small ranula are also seen at the tongue base. (Photo contributor: David Effron, MD.)



FIGURE 5.67 ■ **Sialolithiasis.** A stone is seen at the orifice of Wharton duct. (Photo contributor: Larry B. Mellick, MD



FIGURE 5.68 ■ **Suppurative Submandibular Sialoadenitis.** After applying firm pressure, purulent discharge is seen coming from Wharton duct. (Photo contributor: Jeffery D. Bondesson, MD.)



FIGURE 5.69 ■ **Sialolithiasis.** Rapid swelling and pain of the right parotid gland suggest an obstructed parotid duct. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.70 ■ **Sialolithiasis.** This stone was subsequently delivered through Stensen duct. (Photo contributor: Lawrence B. Stack, MD.)

SINUSITIS

Clinical Summary

Sinusitis results from an impairment of mucociliary clearance and subsequent inflammation of the paranasal sinuses. Most cases of bacterial sinusitis are associated with antecedent viral upper respiratory tract infection.

Maxillary sinusitis (most common) is associated with paranasal facial pain, maxillary dental pain, purulent rhinorrhea, retroocular pain, and conjunctivitis. Ethmoid sinusitis, more common in children, produces a low-grade fever and periorbital pain. Frontal sinusitis can cause a severe supraorbital headache, which is exacerbated by leaning forward; a low-grade fever; upper lid edema; and rhinorrhea. Sphenoid sinusitis is rare, and patients typically complain of a vertex headache and retroocular pain. It can involve CNs, the pituitary gland, and the cavernous sinus. Involvement of all sinus cavities is referred to as pansinusitis. Important complications of sinusitis include periorbital and orbital cellulitis, cavernous sinus thrombosis, and intracranial abscess.

Pott's puffy tumor (a rare osteomyelitis of the cranium from direct extension of a frontal sinusitis) presents as a boggy, tender swelling overlying the frontal sinus.

H influenzae and S pneumoniae together represent 60% to 70% of all bacterial causes. Others include S pyogenes, S aureus, and M catarrhalis. Immunocompromised patients are susceptible to fungal infections, including Aspergillus and Mucor species.

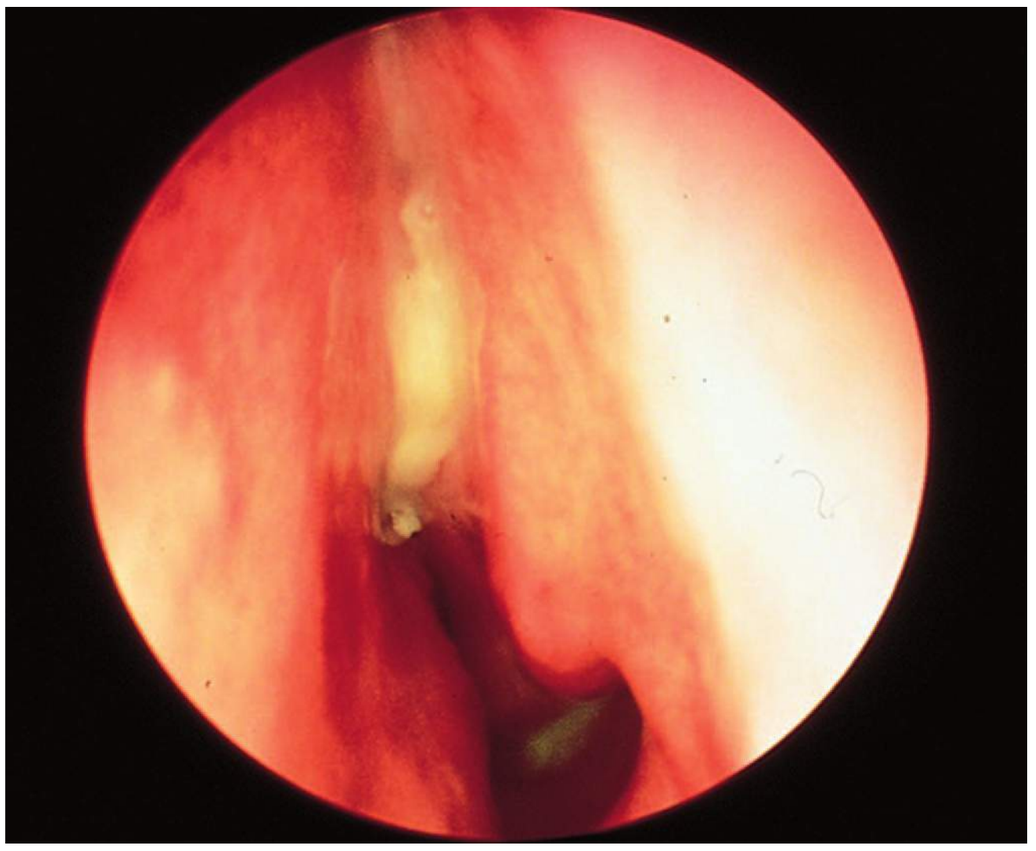


FIGURE 5.71 ■ **Sinusitis.** Purulent drainage from the maxillary sinus ostium in a patient with maxillary sinusitis. Drainage may not always be apparent, since the ostium may be occluded from swelling and inflammation. (Photo contributor: Robin T. Cotton, MD.)

Management and Disposition

Treat most patients with decongestants and analgesics. Decongestants reduce local edema, increase air movement within the sinuses, and decrease local secretions. Humidified air, steam, or saline nasal sprays also facilitate drainage. Patients should be strongly encouraged to stop smoking. If symptoms persist beyond 7 days, consider antibiotics.

Parenteral steroids are not used in acute or recurrent sinusitis. Intranasal steroids may have a role in allergic and chronic sinusitis. CT is the most sensitive and specific imaging modality and allows for better delineation of the sphenoid and ethmoid sinuses.

Otolaryngologist or primary care referral or follow-up should be made for all patients within 3 weeks for routine cases. Patients with comorbid illnesses or more complicated sinusitis should be admitted for parenteral antibiotic therapy and supportive care.

Pearls

1. Chronic sinusitis may be due to mucoid retention cysts, deviated septum, or polyps, which are often visible on plain radiographs. Refer these patients for possible surgery.
2. Physicians must consider fungal etiologies in immunocompromised patients or those with comorbid illnesses.

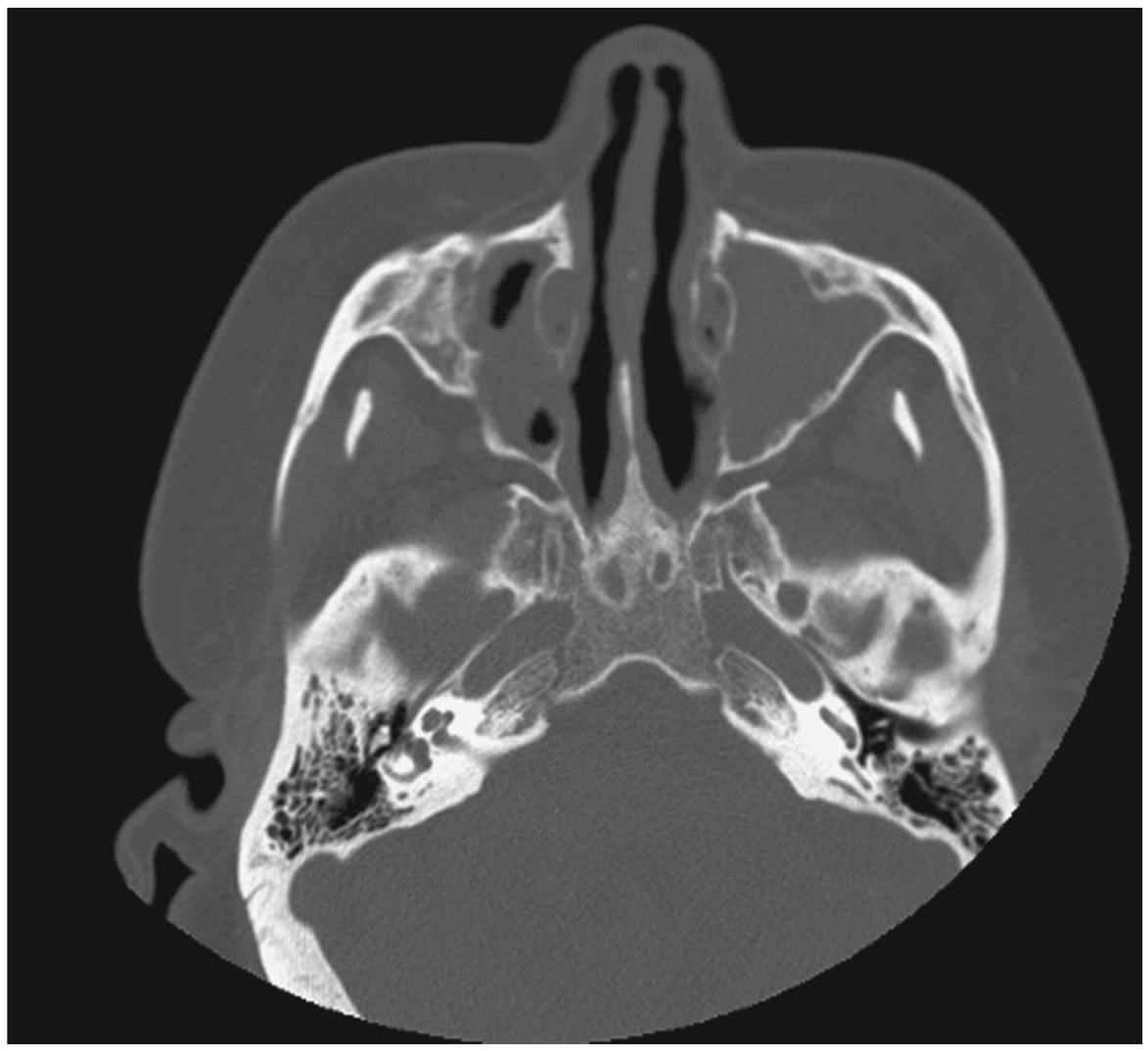


FIGURE 5.72 ■ **Sinusitis.** Bone windows of a sinus CT demonstrating bilateral maxillary sinus disease. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.73 ■ **Sinusitis—Pott's Puffy Tumor.** Forehead and right periorbital edema in a patient with pansinusitis suggests osteomyelitis of the anterior table of the frontal sinus. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 5.74 ■ **Sinusitis—Pott's Puffy Tumor.** Forehead and periorbital edema in a child with pansinusitis suggests osteomyelitis of the anterior table of the frontal sinus. (Photo contributor: David Effron, MD.)

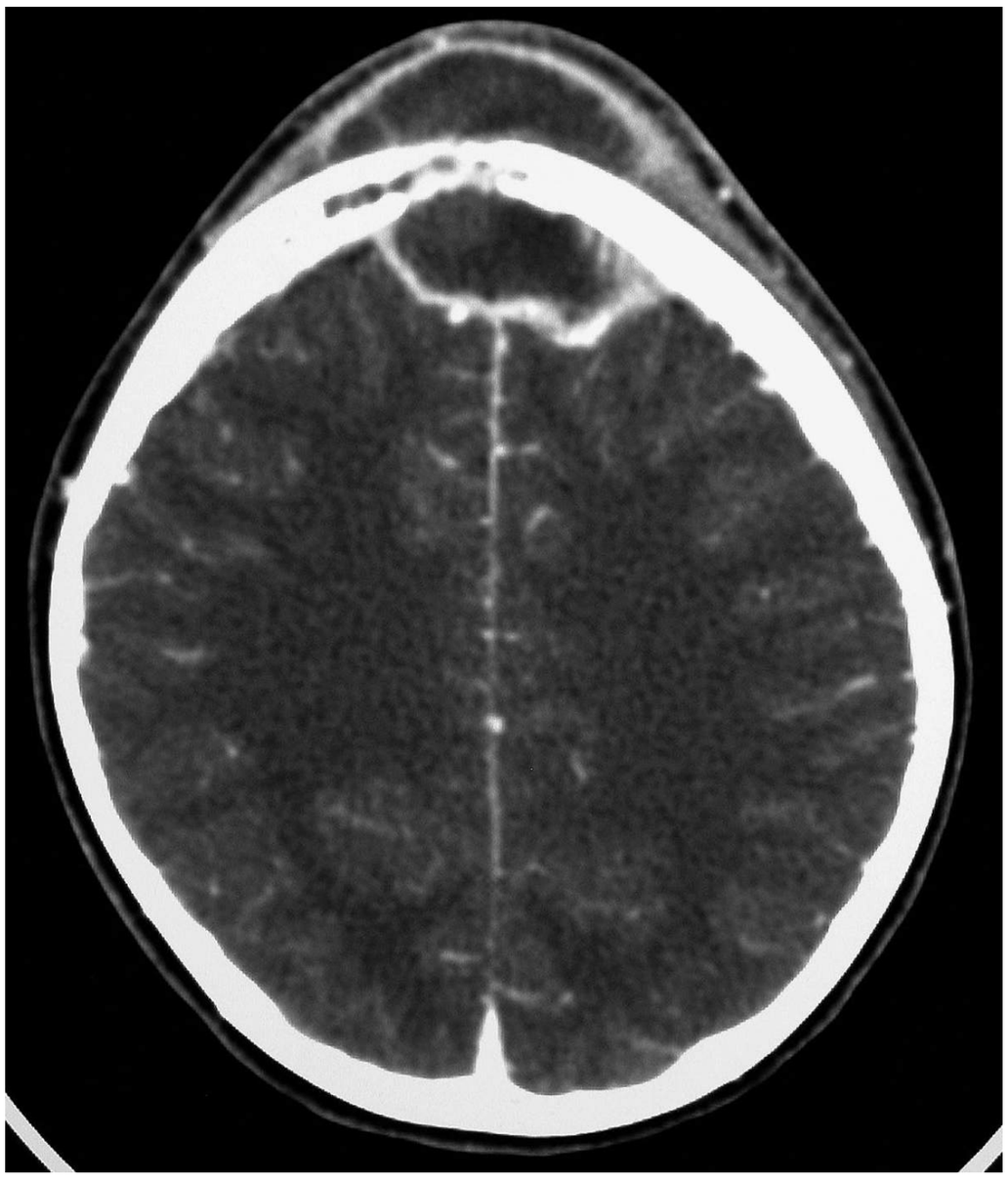


FIGURE 5.75 ■ **Sinusitis—Pott's Puffy Tumor.** CT (parenchymal view) demonstrating intracranial abscess formation and bony destruction of frontal bone from sinusitis. (Photo contributor: David Effron, MD.)

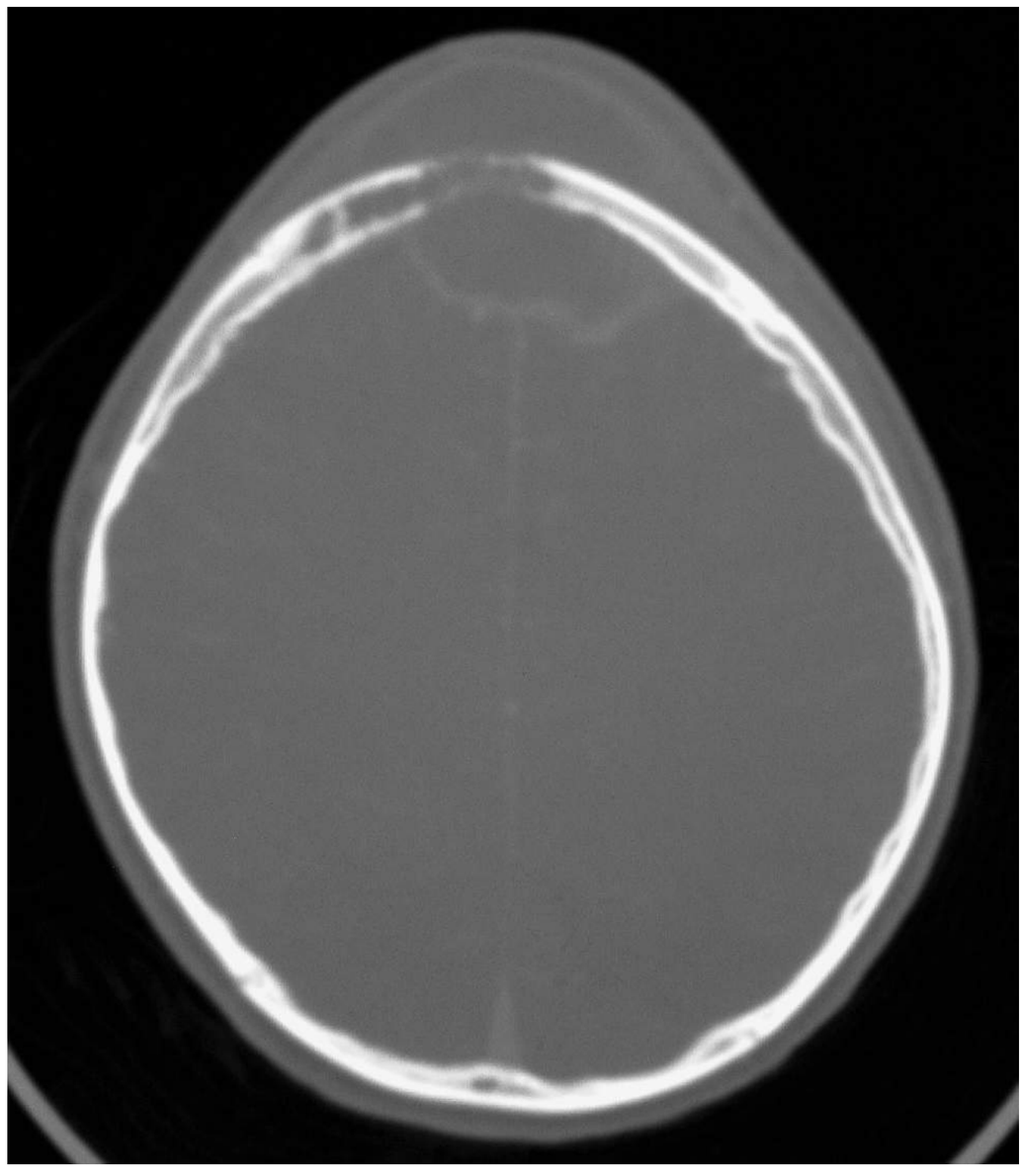


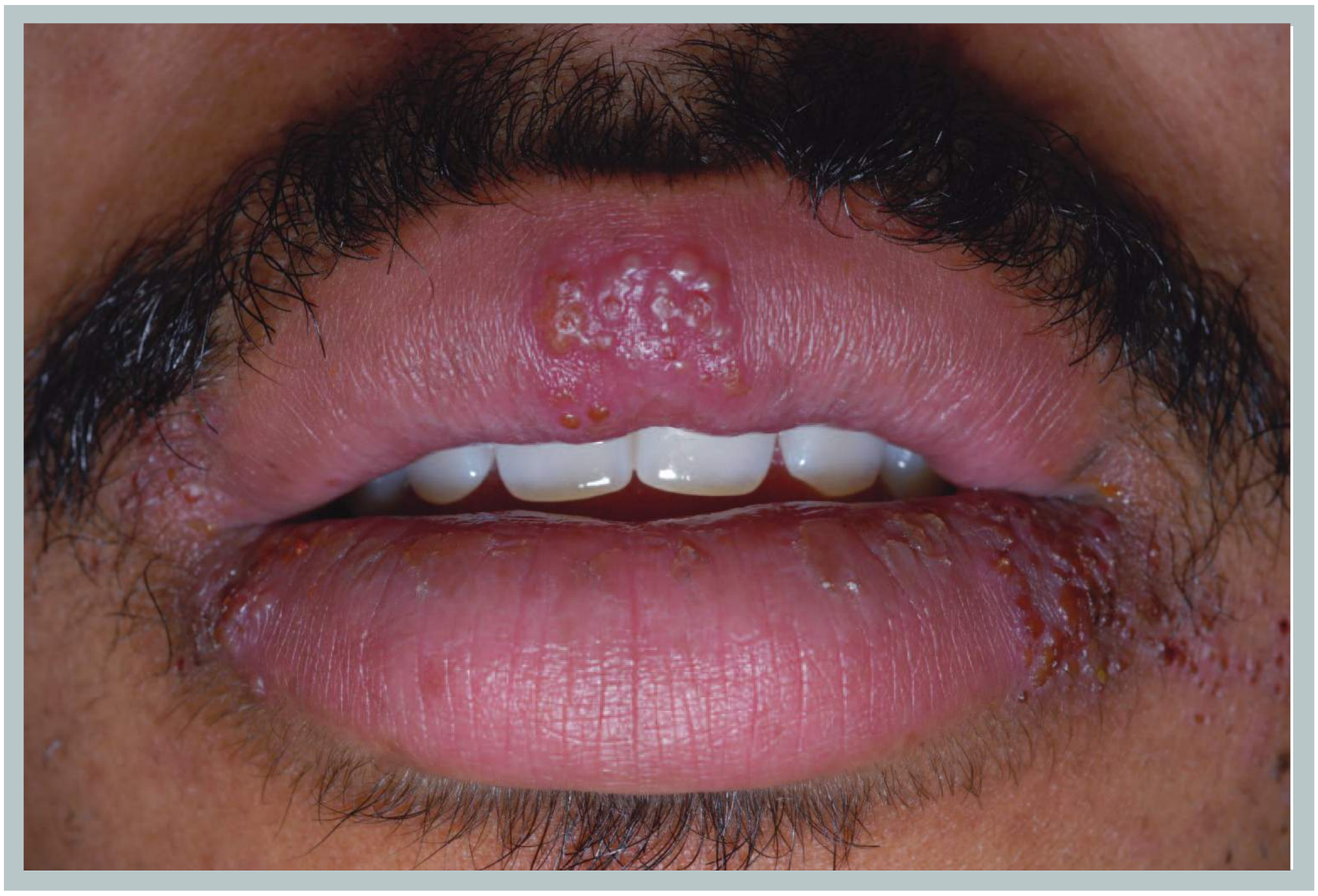
FIGURE 5.76 ■ **Sinusitis—Pott's Puffy Tumor.** CT (bone window) highlighting bony destruction of inner and outer table of frontal bone from sinusitis. (Photo contributor: David Effron, MD.)

Chapter 6

ORAL CONDITIONS

Edward C. Jauch
James W. Howell

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Herpes Labialis. Note the extensive painful ulcerations on the patient's upper lip and corners of the mouth. A prodromal period of fever, malaise, and cervical adenopathy may herald the onset of these painful ulcerations. (Photo contributor: R. Jason Thurman, MD.)

The authors acknowledge Brent E. Gottesman, MD, Edwin D. Turner, MD, and Sara-Jo Gahm, MD, for portions of this chapter written for the previous editions of this book.

TOOTH SUBLUXATION

Clinical Summary

Tooth subluxation, the loosening of a tooth in its alveolar socket, is most commonly secondary to trauma; however, infection and periodontal disease may also produce subluxation. Gingival lacerations and alveolar fractures are associated with dental subluxations. Gentle pressure to the teeth with a tongue blade or fingertip may produce movement, mild displacement, or blood along the crevice of the gingiva, all signs of subluxation. Dental impaction and alveolar ridge fracture should be considered and ruled out clinically or radiographically.

Management and Disposition

1. Primary teeth: If the subluxated tooth is forced into close proximity to the underlying permanent tooth, follow-up for extraction is indicated. Otherwise, the patient should be instructed to follow a soft diet for 1 to 2 weeks, allowing the tooth to reimplant spontaneously.
2. Permanent teeth: Unstable teeth should be temporarily immobilized using gauze packing, a figure-eight suture around the tooth and an adjacent tooth, aluminum foil, or a special periodontal dressing, and the patient referred for dental follow-up.

Pearls

1. Any evidence of tooth mobility following trauma is a subluxation by definition.
2. Always consider an associated underlying alveolar or occult root fracture.



FIGURE 6.1 ■ **Tooth Subluxation.** Note the presence of blood along the crevice of the gingival margin of both central incisors—an indication of subluxation following trauma. Mild displacement of the subluxated teeth is noted. (Photo contributor: James F. Steiner, DDS.)



FIGURE 6.2 ■ **Tooth Subluxation.** Tooth subluxations can be quite subtle, as in this case of a slightly subluxed tooth #9 missed on initial examination. A careful dental examination is essential in patients with oral trauma. (Photo contributor: Kevin J. Knoop, MD, MS.)

TOOTH IMPACTION (INTRUSIVE LUXATION)

Clinical Summary

Impacted or intruded teeth result when a tooth is forced deeper into the alveolar socket or surrounding tissues as a result of trauma. The tooth appears shorter than its contralateral partner. An impacted tooth may be partially visible or completely hidden by the gingiva and buried in the alveolar process. Completely impacted teeth may erroneously be considered avulsed until a radiograph demonstrates the intruded position. The apex of a completely impacted permanent central incisor may be driven through the alveolar bone into the floor of the nares, causing epistaxis. Associated injuries may include alveolar fractures, dental crown or root fractures, and oral mucosal or gingival lacerations. Pulp necrosis occurs in 15% to 50% of cases.

Management and Disposition

Impacted primary teeth usually re-erupt and reposition spontaneously within 1 to 3 months. Any intruded primary tooth whose apex is displaced toward or impacts on the follicle of its permanent successor requires dental follow-up for extraction and monitoring clinically and radiographically for 1 year. Permanent teeth do not re-erupt. Surgical reduction is indicated to prevent complications such as external root resorption and loss of supporting bone. Orthodontic repositioning and splinting is generally carried out over 3 to 4 weeks.

Pearls

1. An undiagnosed impacted tooth is predisposed to infection and may have a poor cosmetic result.
2. The maxillary incisors are the most commonly impacted teeth.



FIGURE 6.3 ■ **Tooth Intrusion.** This impaction injury with multiple anterior maxillary tooth involvement shows various degrees of tooth impaction. Also note the complete absence of a central incisor. This may indicate a complete intrusion into the alveolar socket or an avulsion of the tooth. Radiographic studies are required when a tooth's location is in question. (Photo contributor: James F. Steiner, DDS.)

TOOTH AVULSION

Clinical Summary

Avulsion is the total displacement of a tooth from its socket. There is usually a history of trauma; however, infectious etiologies may result in complete disruption of the periodontal ligament from the affected tooth. Various degrees of bleeding from the socket and surrounding gingiva may be noted and there may be underlying alveolar fracture depending on the mechanism of injury. Prompt inquiry into the location of any unaccountable tooth is indicated. Radiographic evaluation to rule out aspiration, soft tissue entrapment, impaction, or dentoalveolar fracture is required when teeth are missing.

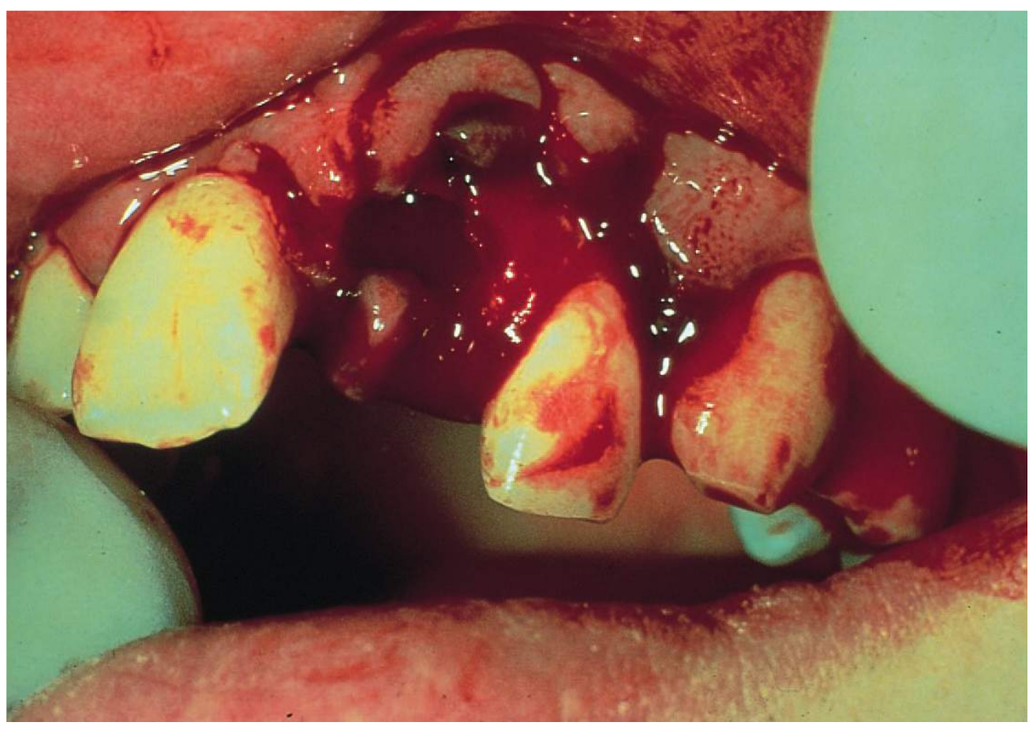


FIGURE 6.4 ■ **Tooth Avulsion.** Avulsion injury with angulation and displacement of teeth from the alveolar socket. (Photo contributor: James F. Steiner, DDS.)

Management and Disposition

Successful reimplantation decreases by 1% for every minute the tooth is out of its socket. Permanent teeth should be replaced in their sockets as soon as possible. Successful reimplantation depends on the survival of periodontal ligament fibers; the tooth should be rinsed with saline, but not scrubbed, with care not to handle the root while replacing it in the socket. Emergent dental consultation, tetanus prophylaxis, and antibiotics targeting mouth flora are indicated. If not replaced, the avulsed tooth should be stored in the mouth of the patient or parent, or in a container of milk. Normal saline and commercial preservatives are reasonable alternatives, but tap water should not be used. Primary teeth are not reimplanted; reimplanted teeth may interfere with eruptions of permanent teeth because of ankylosis and fusion to the bone. Follow-up should be obtained for possible orthodontia until the permanent tooth erupts.



FIGURE 6.5 ■ **Tooth Avulsion.** Significant avulsion injury of tooth #8 in a patient with direct oral trauma. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. Primary teeth should not be reimplanted.
2. Successful reimplantation occurs best within the first 30 minutes.
3. Storage and transport media in decreasing order for preserving tooth viability include balanced salt solution or a tissue culture medium, chilled low-fat milk, saline, and saliva.



FIGURE 6.6 ■ **Tooth Avulsion Reimplanted.** Reimplantation of the avulsed tooth in Fig. 6.5 was performed by an oral surgeon with the use of an arch bar to secure the tooth. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Anatomically, each tooth has a crown and root portion. Externally, the crown is covered with white enamel and the root portion with cementum. The cementoenamel junction (cervical line) is where the crown and root meet. The yellow-to-tan dentin is the second innermost layer and comprises the bulk of the tooth. The red-to-pink pulp tissue is located in the center of the tooth and includes the tooth's neurovascular supply. The Ellis classification system is commonly used to describe tooth fractures above the cervical line in anterior teeth:

Ellis class I: Involves only the enamel.

Ellis class II: Involves the enamel plus exposure of the dentin. The patient may complain of temperature sensitivity.

Ellis class III: Fracture extends into the pulp. A pink or bloody discoloration on the fracture surface is diagnostic. The patient may have severe pain but may also have no pain due to loss of tooth nerve function.

Tooth fractures of the dental root may also occur below the cementoenamel junction and are commonly missed on initial evaluation. Bleeding may be observed at the gingival crevice with associated tooth tenderness on percussion. Radiographic evaluation may aid in differentiating these conditions.

Management and Disposition

Ellis class I: Pain control and referral for rough tooth edge and cosmetic management are indicated.

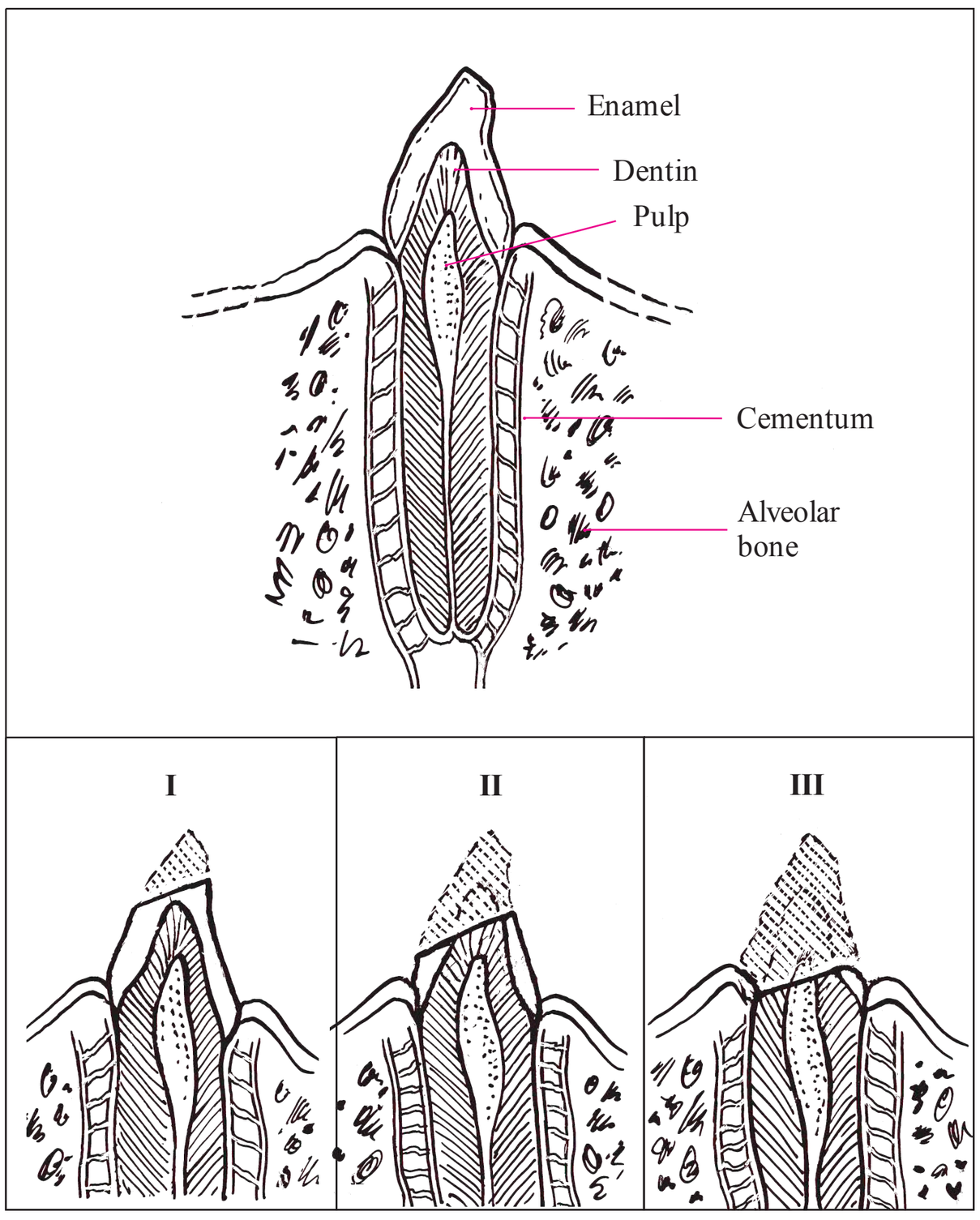


FIGURE 6.7 ■ **Tooth Fractures.** Enamel, dentin, and pulp are the anatomic landmarks used in the Ellis classification of tooth fractures.

Ellis class II: Patients under 12 years of age have less dentin than older patients and are at risk for pulp infection. They should have a calcium hydroxide dressing placed, covered with gauze or aluminum foil, and seen by a dentist within 24 hours. Older patients should see a dentist within 24 to 48 hours.

Ellis class III: This is considered a dental emergency, and dental consultation within 24 to 48 hours is indicated. Delay in treatment may result in abscess formation.

Root fractures: Early reduction, immobilization/splinting, and dental referral within 24 to 48 hours are indicated. Most teeth sustaining root fractures maintain pulp viability.

Pearls

1. Check for tooth mobility on initial examination to aid in differentiating mobility involving the entire tooth from involvement of only the fractured segment.



FIGURE 6.8 ■ **Ellis Class I Tooth Fracture.** Note the fracture of the left upper central incisor. The sole involvement of the enamel is consistent with an Ellis class I fracture. (Photo contributor: James F. Steiner, DDS.)



FIGURE 6.9 ■ **Ellis Class II Tooth Fractures.** Bilateral maxillary central incisor injuries with exposed enamel and dentin consistent with an Ellis class II fracture. (Photo contributor: James F. Steiner, DDS.)



FIGURE 6.10 ■ **Ellis Class III Tooth Fracture.** A fracture demonstrating blood at the exposed dental pulp. This sign is pathognomonic for an Ellis class III fracture. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 6.11 ■ **Multiple Ellis Fractures.** Multiple Ellis fractures resulting from blunt trauma: Ellis I fractures are seen in teeth 7 and 11, Ellis II in teeth 8 and 10, and Ellis III in tooth 9. (Photo contributor: Rosie Korman, MD.)

Clinical Summary

The alveolus is the tooth-bearing segment of the mandible and maxilla. Fracture of the alveolar process tends to occur most often in the thinner maxilla. The anterior alveolar processes are at greatest risk for fracture due to more direct exposure to trauma. Both subluxation and avulsion of teeth may be associated with underlying alveolar fractures. Various degrees of tooth mobility and gingival bleeding may occur.

Management and Disposition

Significant cosmetic deformity may result from alveolar bone loss; preservation of viable tissue is important. Gentle direct pressure over the alveolar segment with saline-soaked gauze, avulsed teeth preservation, and tetanus and antibiotic therapy administration are indicated. Oral surgery consultation should be obtained for possible wire stabilization or arch bar fixation.

Pearls

1. Always consider the possibility of an associated cervical spine injury when evaluating patients with facial trauma.
2. Consider aspiration of avulsed teeth.

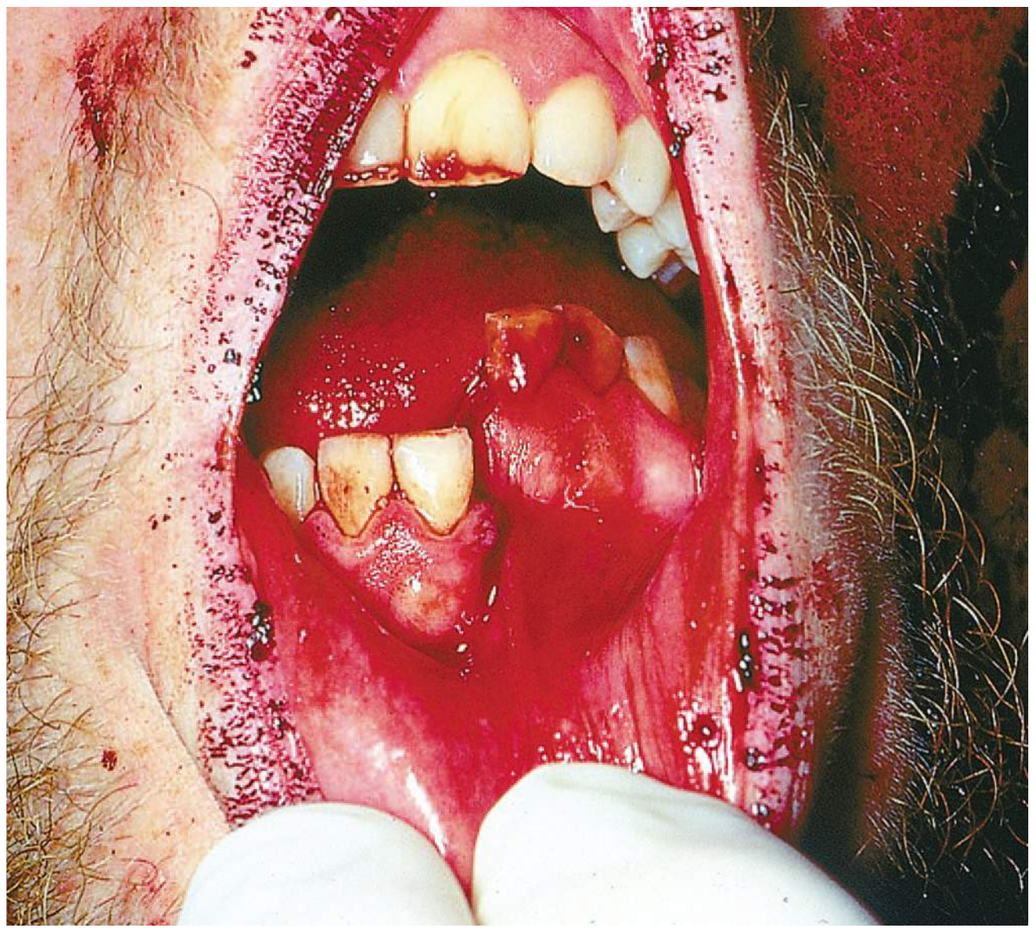


FIGURE 6.12 ■ **Alveolar Ridge Fracture.** Note the exposed alveolar bone segment and associated multiple teeth involvement. Attempts should be made to maximally preserve all viable tissue. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 6.13 ■ **Alveolar Ridge Fracture.** This alveolar ridge fracture was caused by blunt trauma from a steering wheel in a frontal impact motor vehicle collision. (Photo contributor: R. Jason Thurman, MD.)

TEMPORAL MANDIBULAR JOINT DISLOCATION

Clinical Summary

Temporal mandibular joint (TMJ) dislocation generally occurs in predisposed individuals after a vigorous yawn or seizure, or less commonly from direct trauma to the chin while the mouth is open. Dislocation occurs when the mandibular condyles displace forward and become locked anterior to the articular eminence. Masseter muscle spasm contributes to prevention of spontaneous relocation. Weakness of the temporomandibular ligament, an overstretched joint capsule, and a shallow articular eminence are predisposing factors. Patients usually present with an inability to close an open mouth. Other associated symptoms include pain, discomfort, facial swelling near the temporomandibular joint, and difficulty speaking and swallowing. Anterior dislocations are most common; however, posterior dislocation may occur with significant trauma, often in association with basilar skull fractures. Unilateral dislocation results in deviation of the mandible to the unaffected side. TMJ hemarthrosis and dystonic reactions may mimic TMJ dislocations. Mandibular fractures should be considered if there is a history of facial trauma.



FIGURE 6.15 ■ **Bilateral TMJ Dislocation Panorex.** This Panorex image demonstrates bilateral anterior displacement of the mandibular condyles. (Photo contributor: Jake Block, MD.)

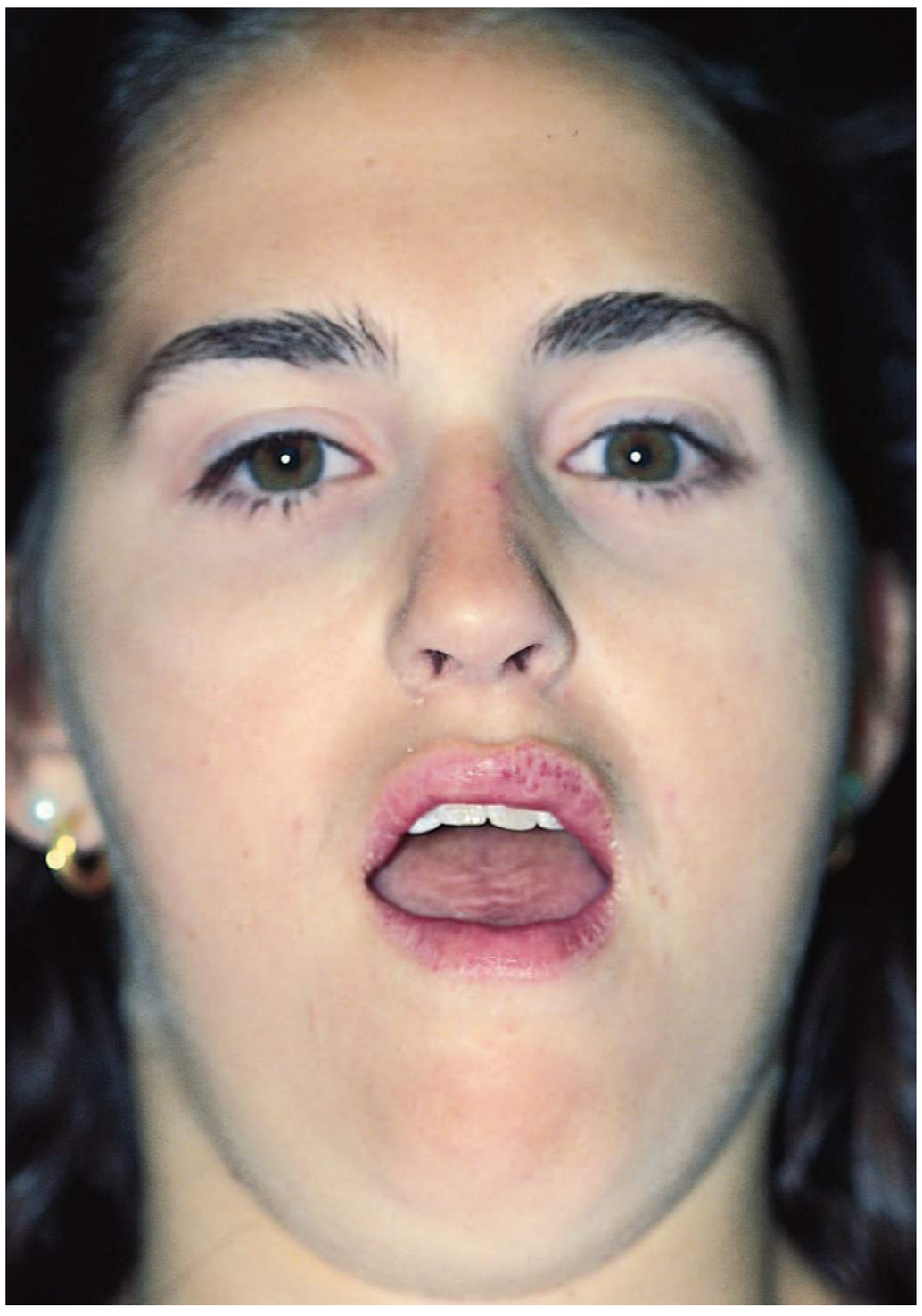


FIGURE 6.14 ■ **TMJ Dislocation (Bilateral).** This patient awoke from sleep with the inability to close her mouth. Note the dry lips and tongue secondary to prolonged exposure. (Photo contributor: Warren K. Russell, MD.)

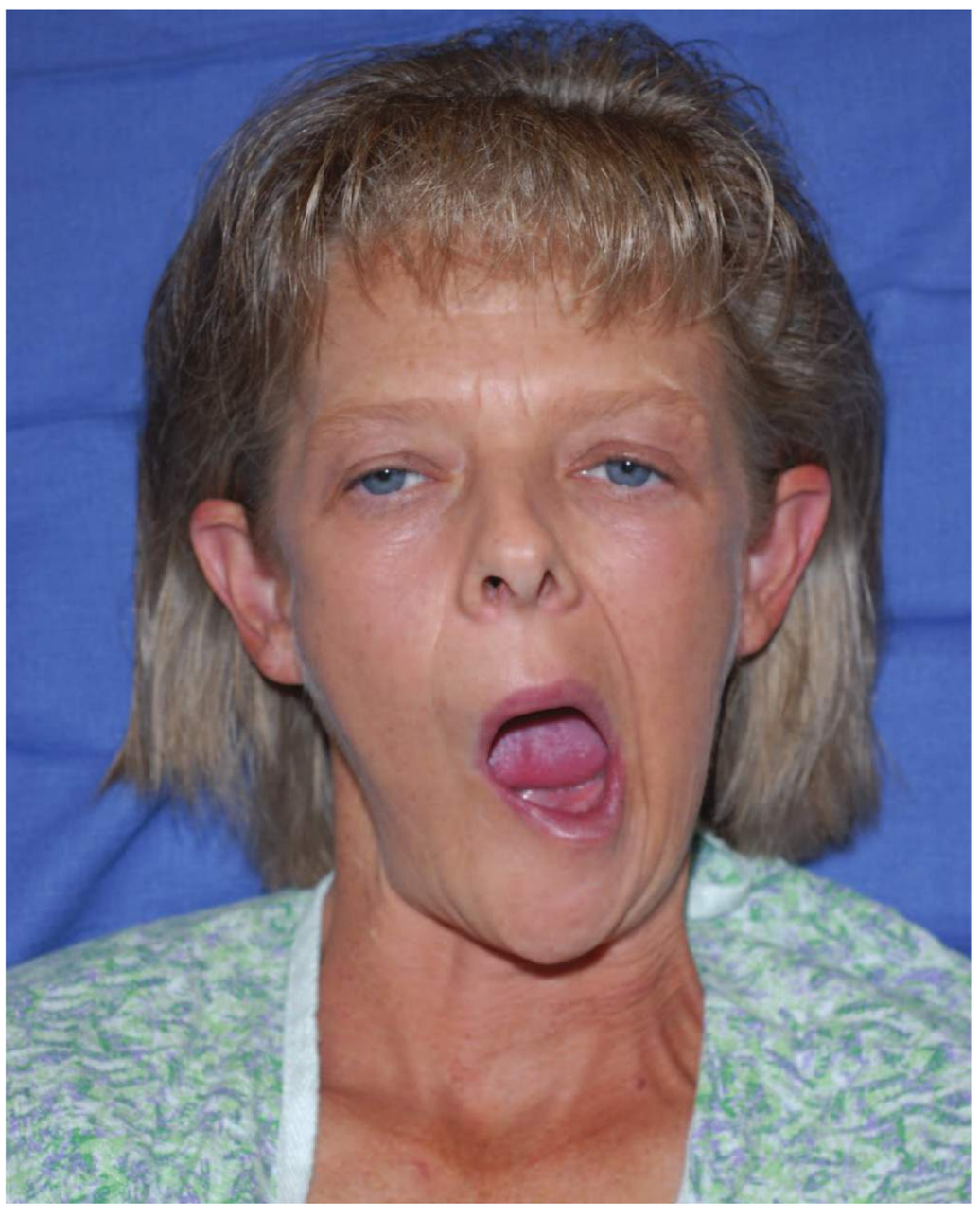


FIGURE 6.16 ■ **TMJ Dislocation, Edentulous Patient (Unilateral).** The mandible is deviated toward the unaffected side. (Photo contributor: R. Jason Thurman, MD.)

Management and Disposition

Acute reduction of pain, muscle spasm, and anxiety is achieved using reassurance, analgesics, and benzodiazepine muscle relaxants. Panorex or TMJ x-ray films (prereduction and postreduction) should be considered to exclude a fracture. A reduction maneuver is performed while facing the sitting patient and grasping the angles of the mandible with both hands. The thumbs are wrapped in gauze for protection and rest on the occlusive surfaces of the molars while downward and backward pressure is steadily applied until the condyle slides back into the articular eminence. Reduction may require some time and force to overcome muscle spasm. Following reduction, instruct the patient to avoid excessively wide mouth opening while eating and yawning for 3 to 4 weeks. Warm compresses to the TMJ, a soft diet for 1 week, and the use of nonsteroidal anti-inflammatory drugs (NSAIDs) are advised. Dental/oral surgery follow-up should be arranged.



FIGURE 6.17 ■ **TMJ Reduction Technique.** Under sedation, the patient in Fig. 6.16 undergoes reduction with inferior-posterior force applied by the physician. The thumbs are not wrapped with gauze in this case as the patient is edentulous. (Photo contributor: R. Jason Thurman, MD.)

Pearls

1. TMJ dysfunction secondary to a neuroleptic or antipsychotic medication–related dystonic reaction is treated with diphenhydramine or benztropine.
2. When trauma is the cause of TMJ dislocation, maintain a high index of suspicion for mandible fractures and cervical spine injuries.
3. A reportedly successful technique for reduction of TMJ dislocation without sedation involves placing a syringe between the posterior molars and having the patient roll the syringe back and forth while gently biting down until reduction is achieved.

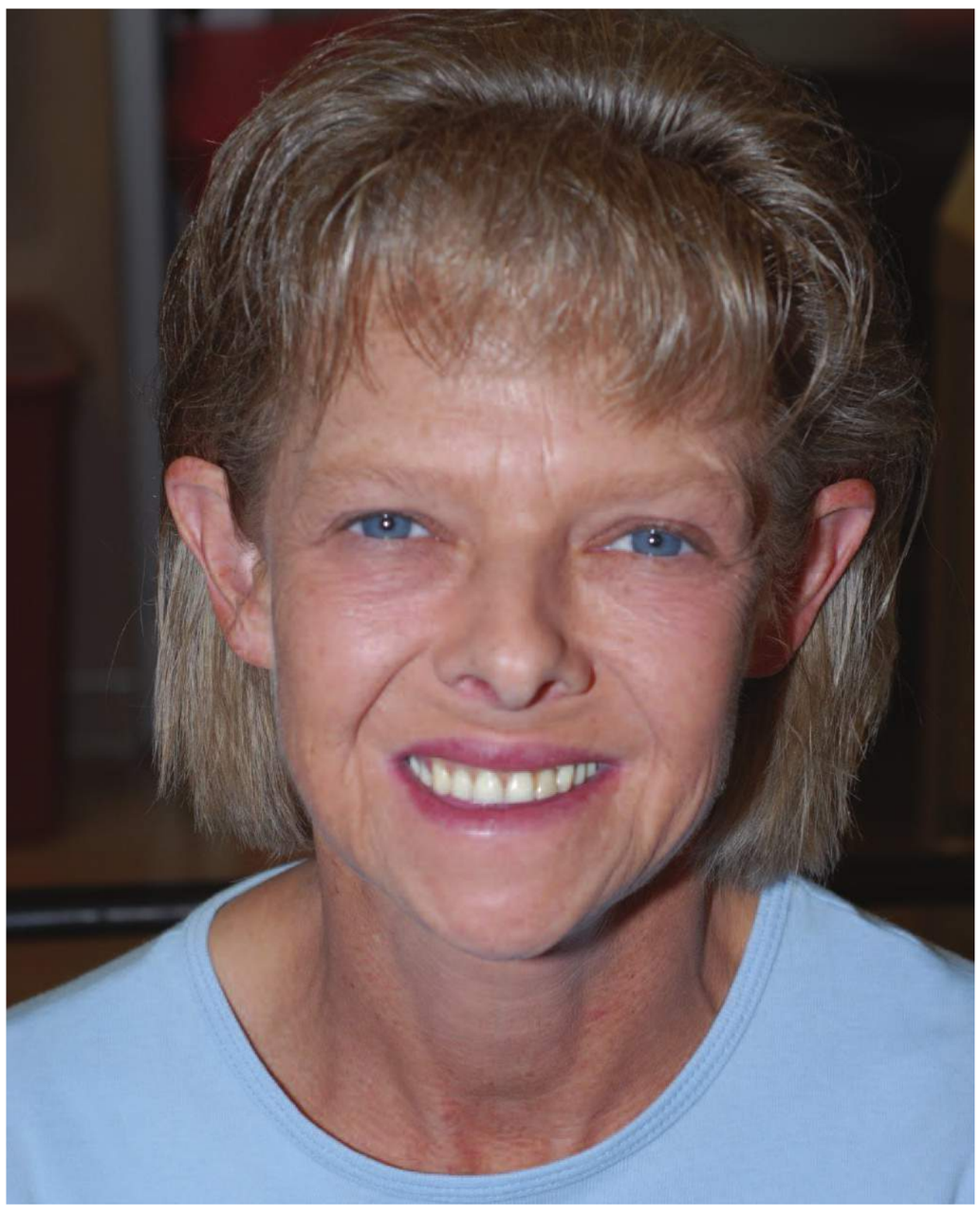


FIGURE 6.18 ■ **TMJ Reduced.** Improved anatomic alignment and a happy patient after reduction of her unilateral TMJ dislocation is accomplished. (Photo contributor: R. Jason Thurman, MD.)

TONGUE LACERATION

Clinical Summary

Injuries to the tongue or mouth floor can cause serious hemorrhage and potential airway compromise and injury to or absence of teeth should be ascertained by inspecting the wound for possibly entrapped dental elements. Dorsal surface tongue lacerations may be associated with a mandibular surface laceration.

Management and Disposition

Most tongue lacerations do not require repair. Lacerations involving the tip or lateral margins or lacerations greater than 1 cm in length that gape widely or actively bleed are best stabilized by a few rapidly absorbable sutures using large bites to include both mucosa and muscle. Anesthesia of the anterior two-thirds of the tongue is obtained by an inferior alveolar/lingual nerve block. Local anesthesia, infiltrated at the site of the wound, may also be used.

Pearls

1. Extensive complex tongue lacerations are at risk for infection and should be prophylactically treated with antibiotics covering oropharyngeal flora.
2. Regional anesthesia for tongue laceration repair avoids distortion of the anatomy prior to repair and is generally better tolerated than direct infiltration of local anesthesia into the tongue.

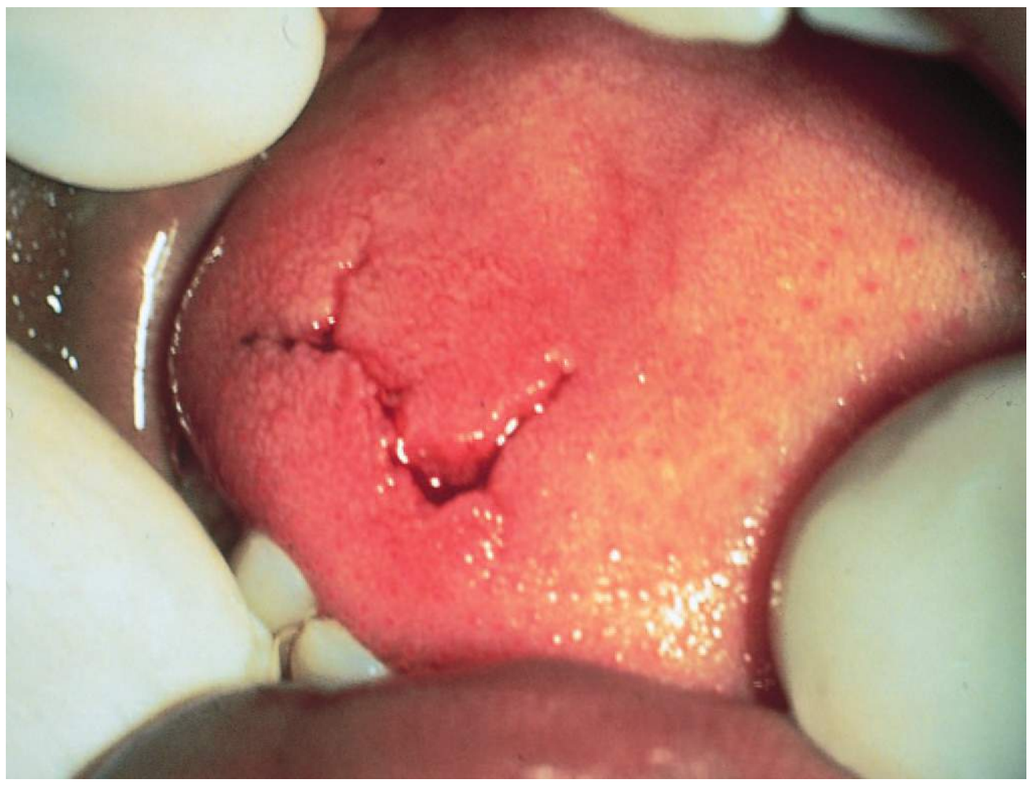


FIGURE 6.20 ■ **Tongue Laceration.** A stellate tongue laceration that does not require suturing is shown. The ventral aspect of the tongue should be examined for additional lacerations sustained from the mandibular teeth. (Photo contributor: James F. Steiner, DDS.)



FIGURE 6.19 ■ **Tongue Laceration.** Due to its length and gaping, this tongue laceration was repaired with absorbable sutures. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 6.21 ■ **Healing Tongue Edge Laceration.** Lacerations of the tip or edge of the tongue should be reapproximated as chronic defects or "forked tongue" may result. (Photo contributor: Lawrence B. Stack, MD.)

VERMILION BORDER LIP LACERATION

Clinical Summary

Anatomically, the vermilion border of the lips represents the transition area from mucosal tissue to skin. Lip lacerations involving the vermilion border present a unique clinical situation, since relatively minor malalignment may produce an unacceptable cosmetic result. An associated underlying gingival or dental injury is a common finding.

Management and Disposition

Accurate vermilion margin reapproximation is the first goal of lip repairs. An unapproximated vermilion margin of 2 mm or greater results in a cosmetic deformity. A regional block of the mental or infraorbital nerve is recommended for anesthesia to avoid additional tissue edema and anatomic distortion produced by local infiltration. Deep or through-and-through lacerations involving the vermilion border should be closed in layers. After closure of the deeper tissue, the first skin suture is always placed at the vermilion border to reestablish the anatomic margin. Using 5-0 or 6-0 nylon, sutures should be placed along the vermilion surface until the moist mucous membrane is encountered. The deep muscular and dermal layer may be closed with 4-0 chromic or Vicryl sutures. Mucosal layers are loosely reapproximated with absorbable suture. The patient should be given wound care instructions and follow-up for wound evaluation and possible suture removal within 3 to 7 days.

Pearls

1. A vermilion border with as little as 2 mm of malalignment may produce a cosmetic defect.
2. Always carefully place the first skin suture at the vermilion border in any lip laceration.

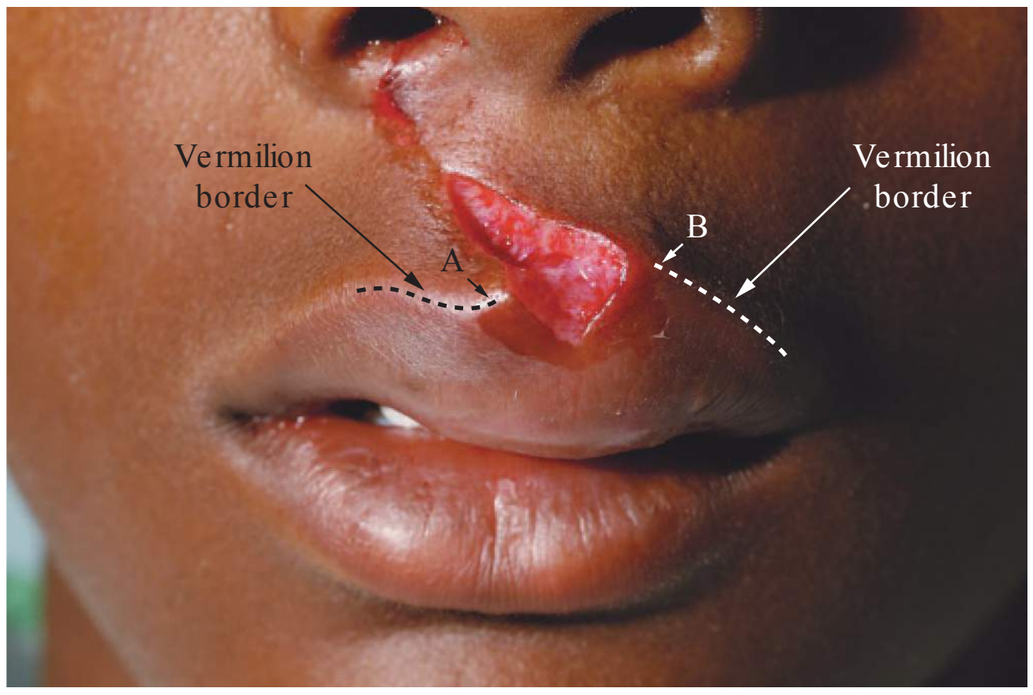


FIGURE 6.22 ■ **Vermilion Border Lip Laceration.** A lip laceration with disruption of the vermilion border. Wound repair begins at the vermilion-skin junction (precise approximation of A to B in this case) for a good cosmetic result. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 6.23 ■ **Complex Vermilion Border Lip Lacerations.** This patient suffered vermilion border lacerations in two areas from an assault with a broken bottle. Following shaving of the moustache, each vermilion-skin junction was repaired before remaining superficial wound closure was achieved. (Photo contributor: R. Jason Thurman, MD.)

GINGIVAL ABSCESS (PERIODONTAL ABSCESS)

Clinical Summary

Gingival abscesses tend to involve the marginal gingiva and result from entrapment of food and plaque debris and subsequent staphylococcal, streptococcal, anaerobic, or mixed bacterial overgrowth. Localized swelling, erythema, tenderness, and fluctuance in the space between the tooth and gingiva ensue. There may be spontaneous purulent drainage from the gingival margin, or an area of abscess pointing. When the gingival abscess involves the deeper supporting periodontal structures, referred to as a periodontal abscess, the patient may present with a fluctuant vestibular abscess or with a draining sinus that opens onto the gingival surface.

In contrast, periapical abscesses are deep and not obvious on inspection. They usually present as tenderness to percussion or pain with chewing over the involved tooth. A parulis may also simulate a gingival abscess; however, a parulis represents the cutaneous manifestation of a deeper periapical abscess. Unlike a parulis or periapical abscess, gingival abscesses are not usually associated with dental caries or fillings. Pericoronal abscesses tend to involve the gingiva overlying a partially erupted third molar.

Management and Disposition

The initial management is a small incision with drainage and warm saline irrigation. Oral antibiotic therapy, analgesics, and dental follow-up are indicated. The patient's tetanus status should be addressed.

Pearls

1. Patients with gingival abscesses are usually afebrile. Consider more extensive abscess formation and oral disease processes in the febrile toxic-appearing patient.
2. Patients with chronic, deep periodontal abscesses complain of dull, gnawing pain as well as a desire to bite down on and grind the tooth.

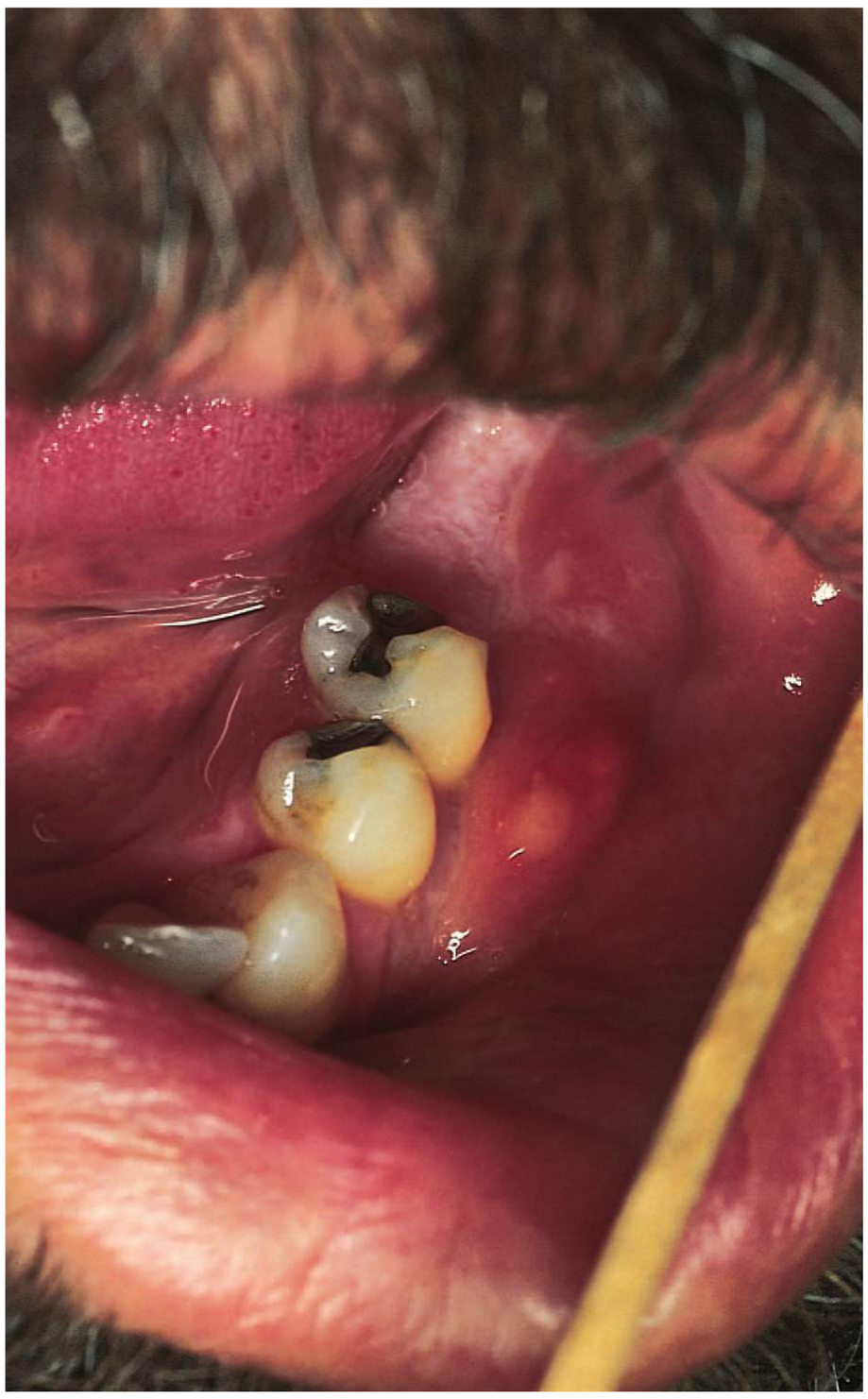


FIGURE 6.24 ■ **Periodontal Abscess.** Localized gingival swelling, erythema, and fluctuance are seen in this periodontal abscess with spontaneous purulent drainage. (Photo contributor: Kevin J. Knoop, MD, MS.)

Clinical Summary

Acute pain, swelling, and mild tooth elevation are characteristics of a periapical or dentoalveolar abscess. Exquisite sensitivity to percussion or chewing on the involved tooth is a common sign. The involved tooth may have had dental caries, a filling, or a root canal. Periapical abscesses can enlarge over time and "point," either internally on the lingual or buccal mucosal surfaces, or extraorally with swelling and redness of the overlying skin. Occasionally, these lesions may track up to the alveolar periosteum and gingival surface to form a parulis. Radiographically, these abscesses appear as well-circumscribed areas of radiolucency at the dental apex or along the lateral aspect of the root. Early acute periapical abscesses may not demonstrate any radiographic changes. Both deep periodontal and periapical abscesses may have sinuses draining purulent material onto the gingival surface. If the infection is allowed to progress, it can erode through cortical bone, manifesting itself in a variety of locations. Panorex films, dental radiographs, or a CT scan may aid in the diagnosis.



FIGURE 6.25 ■ **Periapical Abscess.** This periapical abscess points externally to the overlying skin. (Photo contributor: Robin Cotton, MD.)

Management and Disposition

NSAIDs or oral narcotics for pain, tetanus prophylaxis, and antibiotic therapy are indicated. A regional nerve block provides immediate temporary relief. Dental follow-up in 1 to 2 days is recommended for endodontic evaluation or possible extraction of the involved tooth. Incision and drainage along with saline irrigation and prompt referral are indicated for parulis management.



FIGURE 6.26 ■ **"Gumboil" (Parulis).** This lesion is a sinus tract extension of a periapical abscess. It is differentiated from a periodontal abscess by tenderness to tooth percussion. (Photo contributor: Alan B. Storrow, MD.)

Pearls

1. More than one tooth may be involved.
2. Exquisite tenderness and pain on tooth percussion are key features on physical examination and identify the involved tooth.

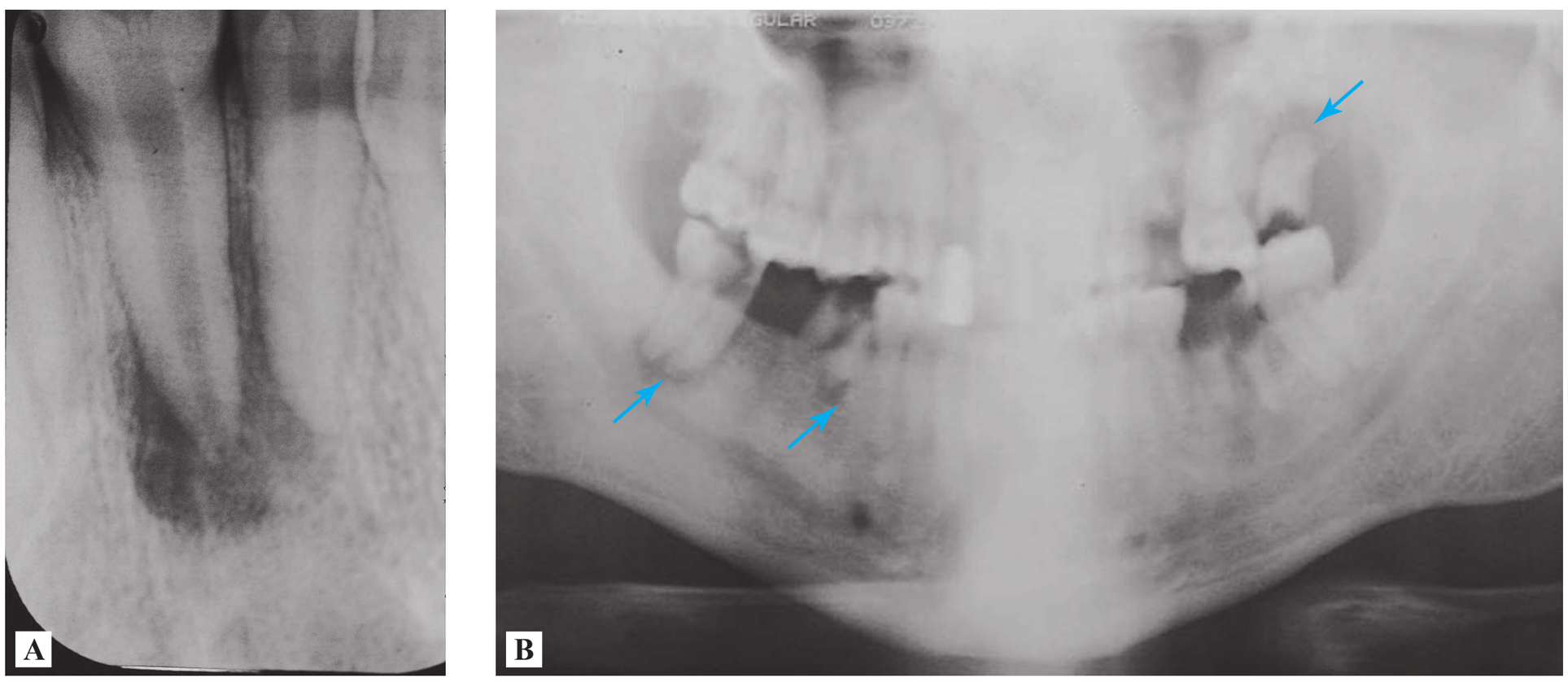


FIGURE 6.27 ■ **Periapical Abscess.** (**A**) Note the well-defined radiolucent area at the apex and lateral root of the tooth in this radiograph. (Photo contributor: James L. Kretzschmar, DDS, MBS.) (**B**) This Panorex film shows lucencies consistent with periapical abscesses, most notably of the right inferior posterior molar tooth (see arrows). (Photo contributor: David P. Kretzschmar, DDS, MS.)

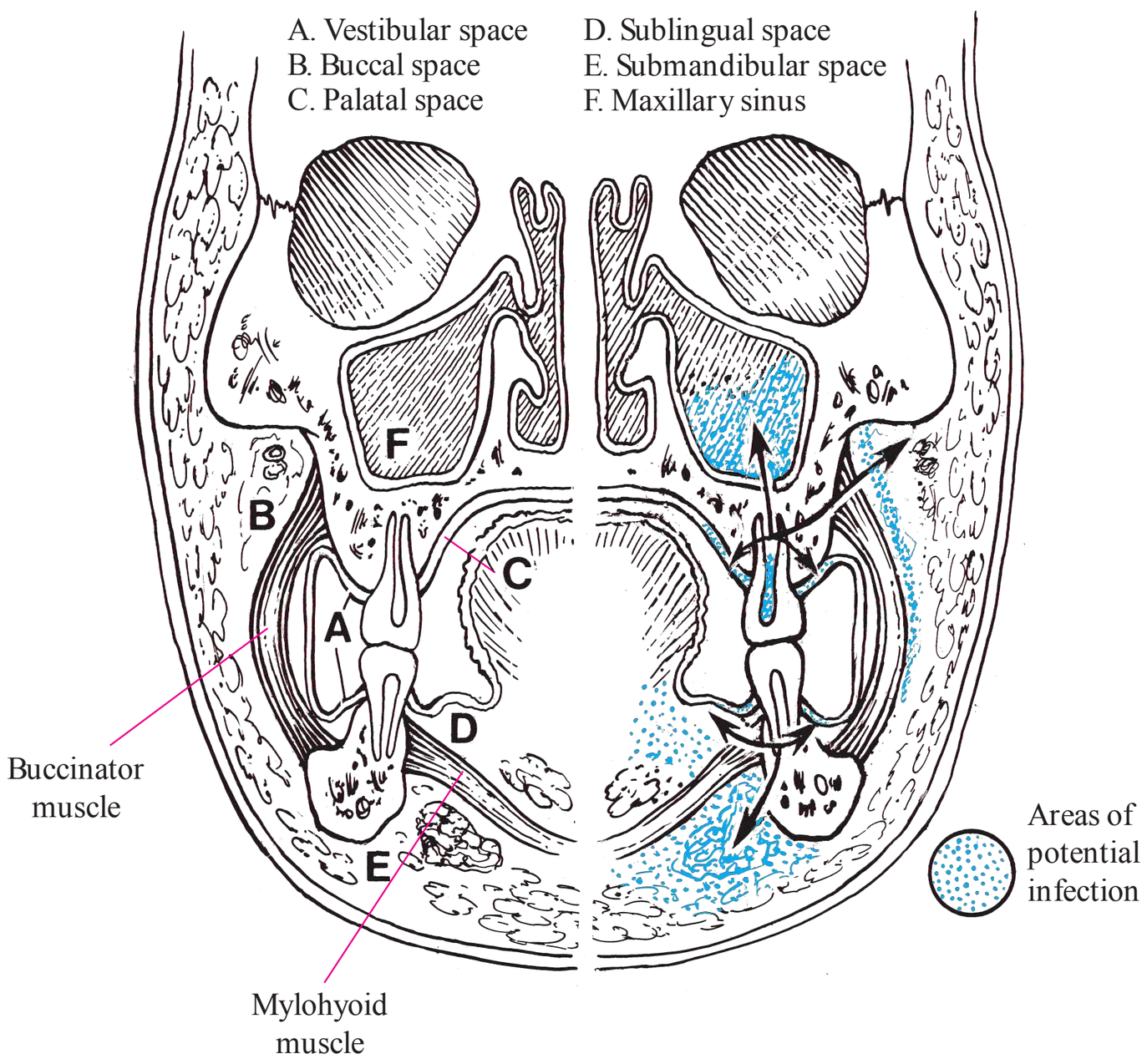


FIGURE 6.28 ■ **Odontogenic Abscesses.** As infection progresses from the pulp at the tooth apex, it erodes through the bone and can express itself in a variety of places. This illustration notes several possible locations or spaces. (Adapted with permission from Cummings C, Schuller D (eds). Otolaryngology Head and Neck Surgery. Chicago: Mosby-Year Book; 1986.)

Clinical Summary

A partially erupted or impacted third molar (wisdom tooth) is the most common site of pericoronitis and pericoronal abscesses. The accumulation of food and debris between the overlying gingival flap and crown of the tooth creates a focus for pericoronitis and subsequent abscess formation. The gingival flap becomes irritated and inflamed, and the tissue is repeatedly traumatized by the opposing molar tooth. The inflamed gingival process may eventually become infected and form an abscess. Foul taste, inability to close the jaw, and fever may occur. Swelling of the cheek and angle of the jaw as well as localized lymphadenopathy is also characteristic. More advanced disease may spread posteriorly to the base of the tongue, oropharyngeal area, and deep cervical spaces with resulting Ludwig angina and peritonsillar abscesses.

Management and Disposition

Superficial incision and drainage of the abscess with warm saline irrigation, analgesia, and antibiotic coverage and referral for possible extraction of the involved teeth are indicated.

Pearls

1. Pericoronitis and abscess formation rarely occur in the pediatric population and tend to be late-adolescent and adult processes; the mandibular third molar is the most commonly involved tooth.
2. Airway compromise is a potential complication with posterior extension of a pericoronal abscess.

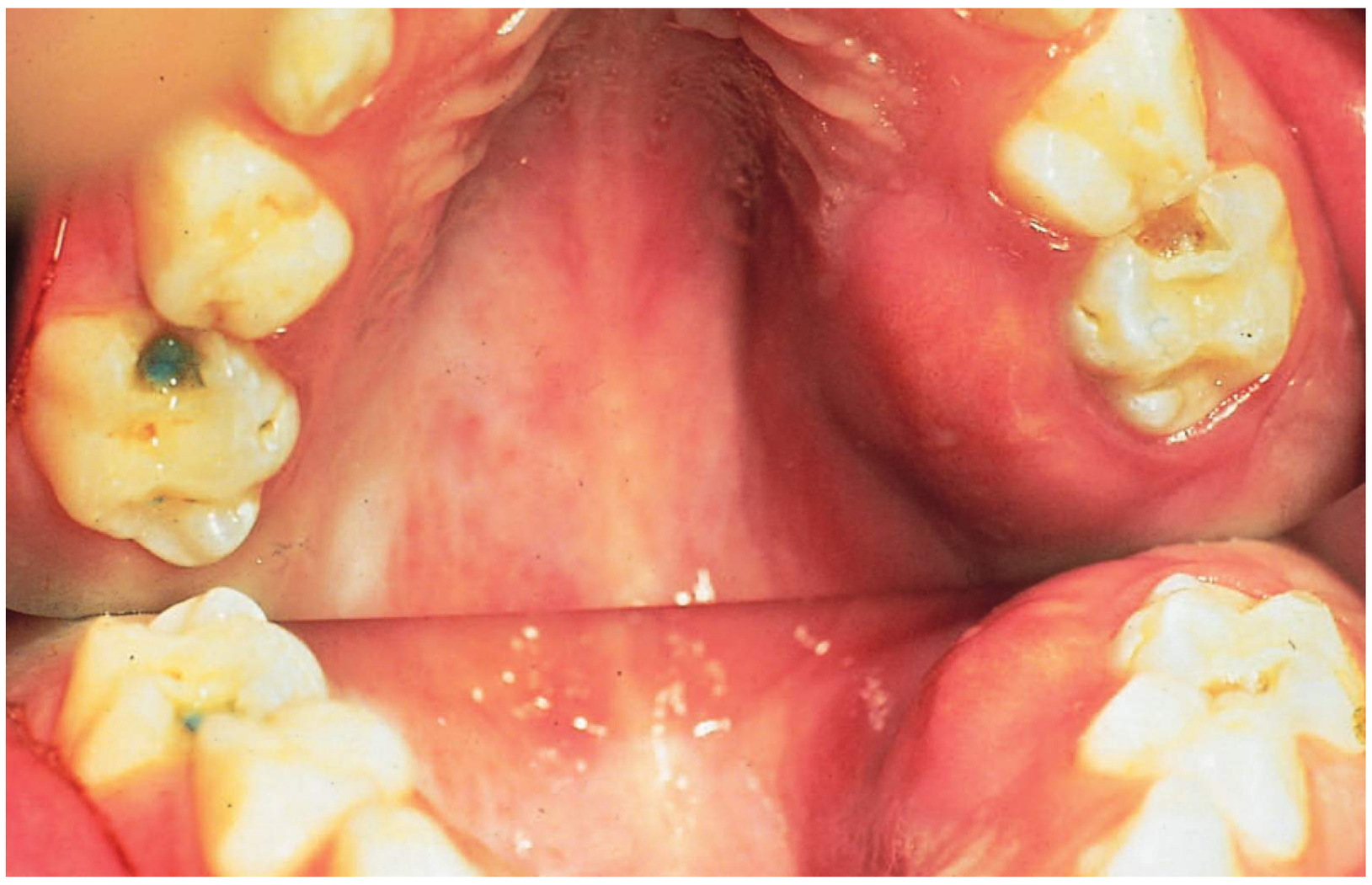


FIGURE 6.29 ■ **Pericoronal Abscess.** Note the inflamed fluctuant gingival tissue approximating the incompletely erupted third molar. (Photo contributor: James F. Steiner, DDS.)

BUCCAL SPACE ABSCESS

Clinical Summary

The buccal space lies between the buccinator muscle and the overlying superficial fascia and skin. The maxillary second and third molars are the usual nidus of buccal space infections, eroding either superiorly through the maxillary alveolar bone or, rarely, inferiorly from the third mandibular molar through the mandibular alveolar bone into the buccal space. Patients usually present with unilateral facial swelling, redness, and tenderness of the cheek. Trismus is generally not present. Parotid gland enlargement due to mumps and suppurative bacterial parotitis should also be considered. The former lacks erythema and warmth of the overlying skin, while the latter is accompanied by trismus and purulent drainage from Stensen duct. Inspection of all the maxillary and third mandibular molar teeth is essential to make the diagnosis. A CT scan may help localize the infection.

Management and Disposition

Broad-spectrum parenteral antibiotic therapy, NSAIDs or oral narcotic analgesics, and dental or oral surgical consultation for endodontic therapy, abscess drainage, and possibly dental extraction are indicated.

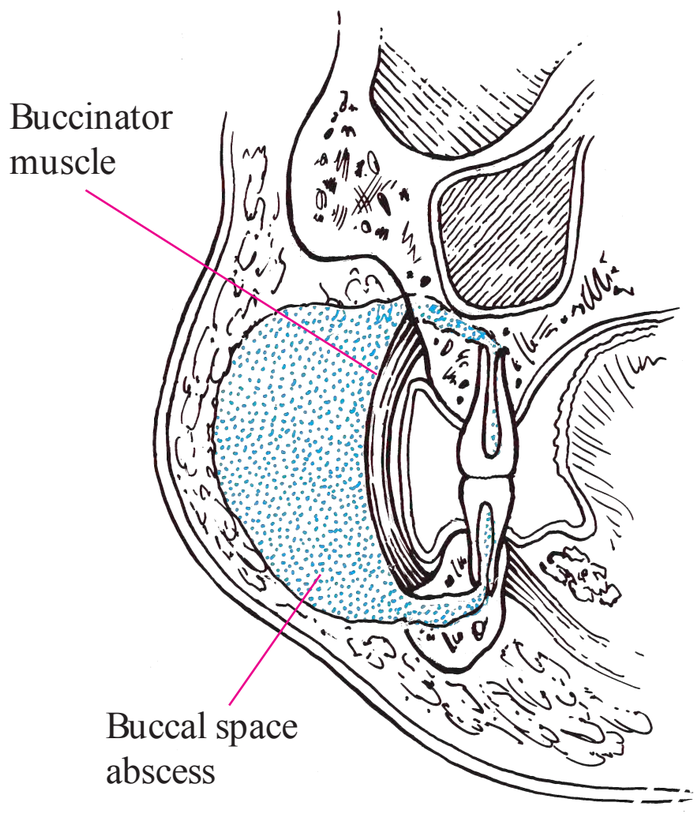


FIGURE 6.30 ■ **Buccal Space Anatomy.** The buccal space lies between the buccinator muscle and the overlying skin and superficial fascia. This potential space may become involved secondary to maxillary or mandibular molar infections. (Adapted with permission from Cummings C, Schuller D (eds). Otolaryngology Head and Neck Surgery. 2nd ed. Chicago: Mosby-Year Book; 1993.)

Pearls

1. Odontogenic infections of the second or third maxillary molars are the most common source for buccal space abscesses.
2. Ovoid cheek swelling with sparing of the nasolabial fold helps to differentiate this from a canine space abscess.



FIGURE 6.31 ■ **Buccal Space Abscess.** Note the ovoid cheek swelling with sparing of the nasolabial fold. This finding, along with accompanying redness and tenderness, helps to identify buccal space abscess formation. (Photo contributor: Michael J. Nowicki, MD.)



FIGURE 6.32 ■ **Buccal Space Abscess.** Note the marked facial asymmetry in this adult patient presenting with a buccal space abscess. (Photo contributor: David Effron, MD.)

Clinical Summary

The canine space lies between the anterior surface of the maxilla and levator labii superioris muscle of the face. Erosion of a maxillary tooth infection through the alveolar bone into the canine space leads to abscess formation, although cutaneous infections from the upper lip and nose are rare sources. Unilateral facial redness, pain, and swelling lateral to the nose with obliteration of the nasolabial fold are characteristic. Severe upper lip and lower eyelid swelling may cause drooling at the corner of the mouth or eye. Maxillofacial CT scan may aid in differentiating these lesions.

Management and Disposition

Parenteral antibiotic therapy including anaerobic coverage is indicated. Dental or oral surgical consultation for incision and drainage is the most definitive treatment for canine space abscesses. Extraction or endodontic treatment of the involved anterior maxillary teeth is usually necessary.

Pearls

1. Loss of the nasolabial fold is characteristic of canine space abscesses.
2. Canine space infections may lead to orbital cellulitis and cavernous sinus thrombosis.
3. Although these patients may drool when significant upper lip swelling is present, they typically do not have trismus, dysphagia, or odynophagia.



FIGURE 6.33 ■ **Canine Space Abscess.** Unilateral facial swelling lateral to the nose with associated redness and the typical loss of the nasolabial fold is shown. The maxillary canine tooth is usually the source of this process. (Photo contributor: Frank Birinyi, MD.)

LUDWIG ANGINA

Clinical Summary

Ludwig angina is defined as rapidly spreading bilateral cellulitis of the submandibular and sublingual spaces with associated tongue elevation. A characteristic painful, brawny induration is present in the involved tissue. The posterior mandibular molars are the usual odontogenic origin for the infection. Streptococcus, Staphylococcus, and Bacteroides species are the most common pathogens. Affected individuals are typically 20 to 60 years old, with a male predominance. Patients are usually febrile and may demonstrate impressive trismus, dysphonia, and odynophagia. Dysphagia and drooling are secondary to tongue displacement and oropharyngeal swelling. Potential airway compromise or spread of infection to the deep cervical layers and the mediastinum is possible.

Management and Disposition

Acute laryngospasm with airway compromise is a potentially life-threatening complication; thorough plans for definitive airway management should be prepared since up to one-third of patients require definitive airway placement. Broad-spectrum aerobic and anaerobic parenteral antibiotic therapy should be initiated. Analgesia should be given as needed. CT or MRI can be used to identify abscess location, but great care should be taken to ensure the patient can tolerate the imaging process. Definitive treatment requires surgical drainage of the abscess. Admission to the intensive care unit is indicated for airway surveillance and management.

Pearls

1. Brawny submandibular induration and tongue elevation are common and characteristic clinical findings.
2. The second mandibular molar is the most common site of origin for Ludwig angina.
3. Acute laryngospasm with sudden total airway obstruction may be precipitated by attempts at oral or nasotracheal intubation.



FIGURE 6.34 ■ **Ludwig Angina.** Note the diffuse submandibular swelling and fullness. Direct palpation of this area would reveal a characteristic brawny induration. Potential airway compromise is a key concern in all patients with Ludwig angina. (Photo contributor: Jeffrey Finkelstein, MD.)



FIGURE 6.35 ■ **Ludwig Angina.** Sagittal view on CT scan demonstrates fluid collection and abscess formation within the floor of the mouth along with superior displacement of the tongue in this patient with Ludwig angina. (Photo contributor: Jake Block, MD.)

PARAPHARYNGEAL SPACE ABSCESS

Clinical Summary

The parapharyngeal space, also known as the lateral pharyngeal or pharyngomaxillary space, is a pyramidal-shaped space with its apex at the hyoid bone and base at the base of the skull. Laterally it is bound by the internal pterygoid muscle and the parotid gland with the superior pharyngeal constrictor muscle acting as the medial border. The posterior aspect of this space is in close proximity with the carotid sheath and cranial nerves IX through XII. Presenting symptoms of parapharyngeal space abscesses include fever, dysphagia, odynophagia, drooling, and ipsilateral otalgia. Unilateral neck and jaw angle facial swelling in association with rigidity and limited neck motion is common. Potentially disastrous complications include cranial neuropathies, jugular vein septic thrombophlebitis, and erosion into the carotid artery. The origin of parapharyngeal abscesses may be from bacterial pharyngitis, sinuses, dentition, or lymphatic spread.

Management and Disposition

Preparations for definitive airway management via endotracheal intubation or surgery are vital. Early recognition and anticipation of other potentially disastrous complications should be considered and managed appropriately. Broad-spectrum antibiotic coverage for mixed aerobic and anaerobic infections should be initiated. Radiologic modalities used to assess parapharyngeal and other deep-space neck infections include contrast-enhanced CT, ultrasound, plain radiography, and MRI. Emergent otolaryngological or oral surgical consultation is warranted for definitive intraoperative incision and drainage of the abscess.

Pearl

1. Suspected oropharyngeal abscesses in association with cranial nerves IX through XII involvement are pathognomonic of parapharyngeal abscesses.



FIGURE 6.36 ■ **Parapharyngeal Space Abscess.** Unilateral facial, jaw angle, and neck swelling is seen in this patient. Nuchal rigidity may also be present. (Photo contributor: Sara-Jo Gahm, MD.)

TRENCH MOUTH (ACUTE NECROTIZING ULCERATIVE GINGIVITIS)

Clinical Summary

Painful, severely edematous interdental papillae are characteristic of acute necrotizing ulcerative gingivitis (ANUG), or trench mouth. Other associated features include the presence of ulcers with an overlying grayish pseudomembrane and a "punched out" appearance. The inflamed gingival tissue is friable, necrotic, and represents an acute destructive disease process of the periodontium. Fever, malaise, and regional lymphadenopathy are commonly associated signs. Patients may also complain of foul breath and a strong metallic taste. Poor oral hygiene, emotional stress, smoking, and immunocompromised states (eg, HIV, steroid use, diabetes) all predispose for ANUG. Anaerobic Fusobacterium and spirochetes are the predominant bacterial organisms involved. The anterior incisor and posterior molar gingival regions are the most commonly affected oral tissues. Acute herpetic, gonococcal, or streptococcal gingivostomatitis, aphthous stomatitis, desquamative gingivitis, and chronic periodontal disease may mimic ANUG.

Management and Disposition

Initial management includes warm saline irrigation, analgesics, topical anesthetics, hydrogen peroxide or chlorhexidine oral rinses, and antibiotic treatment with oropharyngeal coverage. Follow-up within 1 to 2 days is recommended. Patients with more advanced disease may require admission and oral surgical consultation.

Pearls

1. Dramatic relief of symptoms within 24 hours of initiating antibiotics and supportive treatment is characteristic.
2. Periodontal abscesses and underlying alveolar bone destruction are common complications of ANUG and require dental follow-up.
3. Gingivitis is a nontender inflammatory disorder and is not synonymous with ANUG.



FIGURE 6.38 ■ **Acute Necrotizing Ulcerative Gingivitis.** Excessive purulent drainage was associated with this patient's ANUG. (Photo contributor: R. Jason Thurman, MD.)

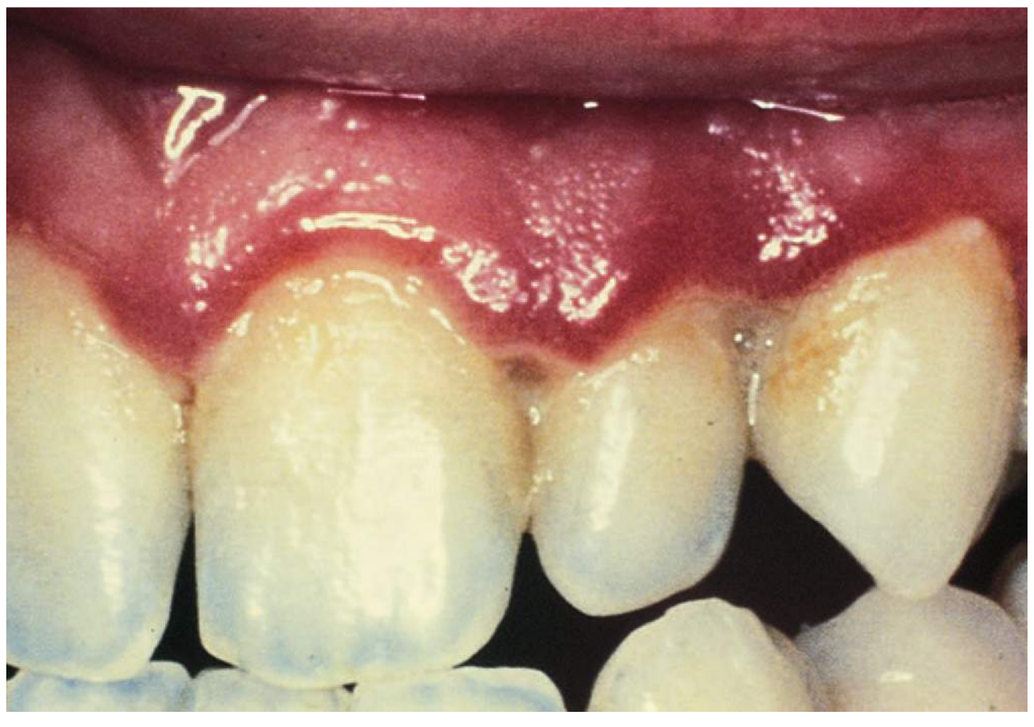


FIGURE 6.37 ■ **Acute Necrotizing Ulcerative Gingivitis.** Note the inflamed, friable, and necrotic gingival tissue. An overlying grayish pseudomembrane or punched out ulcerations of the interdental papillae are pathognomonic. (Photo contributor: David P. Kretzschmar, DDS, MS.)



FIGURE 6.39 ■ **Severe Acute Necrotizing Ulcerative Gingivitis.** Note the extensive gingival necrosis in this advanced case of trench mouth. (Photo contributor: David Effron, MD.)

ACID TOOTH EROSION (BULIMIA)

Clinical Summary

Bulimia nervosa is an eating disorder with significant associated physical complications. It is characterized by binge eating with self-induced vomiting, laxative use, dieting, and exercise to prevent weight gain. Patients with bulimia are at risk for damage to the dental enamel and dentin as a result of repeated episodes of vomiting with chronic exposure to regurgitated acidic gastric contents. The lingual dental surfaces are most commonly affected. In severe cases, all surfaces of the teeth may be affected. Trauma to the oral and esophageal mucosa may also result from induced vomiting. The quantity, buffering capacity, and pH of both the resting and stimulated saliva are found to be reduced, and salivary gland enlargement, most commonly the parotid, may occur as well.

Included in the differential diagnosis of acid tooth erosion are conditions that involve recurrent vomiting. Xerostomia is a condition of excessive mouth dryness (associated with Sjögren syndrome) that can also accelerate the process of enamel loss. Tooth abrasions and erosions may be brought about by the use of chewing tobacco, eating betel nuts (Indian paan), dentifrice use, bruxism, abnormal swallowing, and jaw clenching.

Management and Disposition

The initial emergency department (ED) management of patients with bulimia should address any medical complication of the disorder (hypokalemia, metabolic acidosis, etc). Hospitalization to stabilize medical complications and provide nutritional support may be indicated. Dental treatment should begin with vigorous oral hygiene to prevent further destruction of tooth structures. Regular professional fluoride treatments to cover exposed dentin and cosmetic procedures should be instituted once the patient is adequately stabilized psychologically. A multidisciplinary team approach is necessary and should involve psychiatry, internal medicine, and dental consultation as needed.

Pearls

1. The lingual surfaces of the teeth are the most commonly involved tooth surfaces.
2. Bruxism tends to cause enamel loss from occlusal and incisal dental surfaces.
3. The labial and buccal surfaces of the teeth tend to show enamel loss from repeat or prolonged chemical contact (eg, lemon sucking or tobacco products).



FIGURE 6.40 ■ **Acid Tooth Erosion (Bulimia).** Erosive dentin exposure of the maxillary teeth secondary to chronic vomiting. The involvement of the lingual dental surfaces is characteristic of bulimia. (Photo contributor: David P. Kretzschmar, DDS, MS.)



FIGURE 6.41 ■ **Acid Tooth Erosion (Snuff User).** Note the typical dentin exposure on the buccal dental surfaces resulting from prolonged snuff use and its accompanying acid erosion. (Photo contributor: David P. Kretzschmar, DDS, MS.)

THRUSH (ORAL CANDIDIASIS)

Clinical Summary

White, flaky, curd-like plaques covering the tongue and buccal mucosa with an erythematous base are typical of thrush. These lesions tend to be painless; however, painful inflammatory erosions or ulcers may be noted, particularly in adults. Predisposing factors include antibiotic use, inhaled and oral corticosteroids, radiation to the head and neck, extremes of ages, patients with immunologic deficiencies, and chronic irritation (eg, denture use and xerostomia). Colonization of surface epithelium by Candida occurs due to altered oral microflora. Hairy leukoplakia, lingual lichen planus, flecks of milk or food debris, and liquid antacid adhering to the tongue may be confused with candidiasis. Hairy leukoplakia cannot be removed with a tongue depressor (note Fig. 20.3). This helps differentiate this process from thrush or residue from ingested materials. Microscopic examination of the removed specimen for the presence of hyphae in potassium hydroxide mount will aid in the identification of Candida.

Management and Disposition

Nystatin oral tablets or swish and swallow suspension, fluconazole, or clotrimazole oral troches are therapeutic. Topical analgesic cocktails may provide comfort for patients (eg, liquid antacid with diphenhydramine, viscous lidocaine).

Pearls

1. Thrush is most common in premature infants and immunosuppressed patients.
2. In young adults, thrush may be the first sign of AIDS; a history of HIV risk factors should be elicited.
3. Failure of oral candidiasis to respond to topical antifungal agents may suggest an underlying immune deficiency.



FIGURE 6.42 ■ **Oral Candidiasis (Thrush).** Whitish plaques are seen here on the buccal mucosa. These plaques are easily removed with a tongue blade, differentiating them from lichen planus or leukoplakia. (Photo contributor: James F. Steiner, DDS.)



FIGURE 6.43 ■ **Oral Candidiasis (Thrush).** Extensive thrush is seen on the hard and soft palate of this immunocompromised patient. (Photo contributor: Lawrence B. Stack, MD.)

ORAL HERPES SIMPLEX VIRUS (COLD SORES)

Clinical Summary

Oral herpes simplex may present acutely as a primary gingivostomatitis or as a recurrence. Painful vesicular eruptions on the oral mucosa, tongue, palate, vermilion borders, and gingiva are highly characteristic. A 2- to 3-day prodromal period of malaise, fever, and cervical adenopathy is common. The vesicular lesions rupture to form a tender ulcer with yellow crusting and an erythematous margin. Pain may be severe enough to cause drooling and odynophagia, which can discourage eating and drinking, particularly in children. The disease tends to run its course in a 7- to 10-day period with nonscarring resolution of the lesions. Recurrent herpes labialis may present with an aura of burning, itching, or tingling prior to vesicle formation. Oral trauma, sunburn, stress, and any variety of febrile illnesses can precipitate this condition. Oral erythema multiforme or Stevens-Johnson syndrome, aphthous lesions, oral pemphigus, and hand-foot-mouth (HFM) syndrome are in the differential diagnosis. It should be noted that aphthous ulcers tend to occur on movable oral mucosa and rarely on immovable mucosa (ie, hard palate and gingiva). The vermilion border is a characteristic location for herpes labialis as opposed to aphthous lesions. Posterior oropharyngeal ulcerations with associated hand and foot lesions help to define HFM syndrome.



FIGURE 6.44 ■ **Herpes Simplex Virus (HSV) Stomatitis.** Note the extensive painful ulcerations on the patient's lower lip. A prodromal period of fever, malaise, and cervical adenopathy may herald the onset of these painful ulcerations. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Supportive care with rehydration and pain control is the mainstay of therapy. Temporary pain relief may be achieved with topical analgesics. Viscous lidocaine may be used as an oral rinse, 5 mL every 3 to 4 hours. Oral antiviral agents may be useful in adults with primary infections. Secondary infection of herpetic lesions should be treated with antibiotics.

Pearls

1. Fatal viremia and systemic involvement may occur in infants and children with herpetic gingivostomatitis.
2. Primary acute oral herpetic infection occurs most commonly in children and young adults.
3. Initiation of suppressive therapy with oral antivirals should be considered within the first 72 hours from symptom onset.



FIGURE 6.45 ■ **HSV Stomatitis.** Extensive vesicular lesions along the vermilion border and surrounding tissues are consistent with HSV infection. (Photo contributor: Frank Birinyi, MD.)

APHTHOUS ULCERS (CANKER SORES)

Clinical Summary

Aphthous ulcers are shallow 1 to 15 mm painful mucosal ulcers. A prodromal burning sensation may be noted 2 to 48 hours before an ulcer is noted. The initial lesion is a small white papule that ulcerates and enlarges over 48 to 72 hours. Lesions are typically round or ovoid with a raised yellow border and surrounding erythema. Multiple aphthous ulcers may occur on the lips, tongue, buccal mucosa, floor of the mouth, or soft palate. Spontaneous healing occurs in 7 to 10 days without scarring. The exact etiology is unknown. Deficiencies of vitamin B_{12}, folic acid, and iron as well as viruses have been implicated. Stress, local trauma, and immunocompromised states have all been cited as possible precipitators.

Primary or recurrent herpetic oral lesions may present with an almost identical prodrome and similar appearance. Herpetic lesions, unlike aphthous ones, tend to occur on the gingiva, hard palate, and vermilion border. Oral erythema multiforme may also present similarly to aphthous stomatitis; however, like oral herpes, it may tend to present with multiple vesicles in the early stages. Stevens-Johnson syndrome represents a severe form of erythema multiforme characterized by hemorrhagic anogenital and conjunctival lesions as well as oral lesions. Herpangina results from Coxsackie and echoviruses with oral ulcerations typically involving the posterior pharynx. Oral pemphigus should also be considered. Behçet disease can present with recurrent oral lesions, genital ulcers, and uveitis.

Management and Disposition

Supportive care, rehydration, and pain control constitute the focus of therapy. A topical anesthetic agent such as 2% viscous lidocaine or liquid antihistamine/antacid mix as an oral rinse every 3 to 4 hours is palliative. Use of oral antimicrobial rinses promotes healing. Protective dental paste may be applied every 6 hours to prevent irritation of lesions. Triamcinolone acetonide in an emollient dental paste three to four times daily may reduce pain and promote healing.

Pearls

1. Aphthous ulcers may be associated with Crohn disease.
2. Topical anesthetics may be used as a temporary adjunct in pain relief. Care must be taken to avoid overdose or complications such as methemoglobinemia from overuse.
3. Aphthous ulcers almost never occur on gums or hard palate.



FIGURE 6.46 ■ **Aphthous Ulcer (Single Lesion).** Raised yellow borders with surrounding erythema are typical of aphthous ulcers. (Photo contributor: James F. Steiner, DDS.)



FIGURE 6.47 ■ **Aphthous Ulcerations.** Note the multiple ulcers of various sizes located on the lip and gingival mucosa. These lesions rarely occur on the immobile oral mucosa of the gingiva or hard palate. (Photo contributor: James F. Steiner, DDS.)

STRAWBERRY TONGUE

Clinical Summary

Reddened hypertrophied lingual papillae, called strawberry tongue, are primarily associated with scarlet fever caused by group A streptococcal enterotoxin. The tongue initially appears white with the erythematous papillae sticking through the white exudate. After several days, the white coating is lost and the tongue appears bright red. Other signs of group A streptococcal infection include fever, an exudative pharyngitis, a scarlatiniform rash, and the presence of Pastia lines (petechial linear rash in the skin folds). Kawasaki syndrome may also present with an injected pharynx and an erythematous strawberry-like tongue. It is essential to make the distinction between streptococcal infection and Kawasaki syndrome, since the latter is associated with a high incidence of coronary artery aneurysm if left untreated. Also consider toxic shock syndrome (TSS), in which one-half to three-fourths of patients have pharyngitis with a strawberry-red tongue.



FIGURE 6.48 ■ **Strawberry Tongue.** Note the white exudate with bulging red papillae. The white coating is eventually lost after several days, and the tongue then appears bright red. (Photo contributor: Michael J. Nowicki, MD.)

Management and Disposition

Rapid streptococcal immunoassay testing may help expedite the diagnosis. Pharyngeal cultures are useful for confirming the diagnosis. Antistreptolysin O (ASO) titers can be used for confirmation in the convalescent stage if the diagnosis is in question. Penicillin is the drug of choice for group A streptococcus.

Pearls

1. Strawberry tongue initially appears white in color, with prominent red papillae bulging through the white exudate. After several days, the tongue becomes completely beefy red.

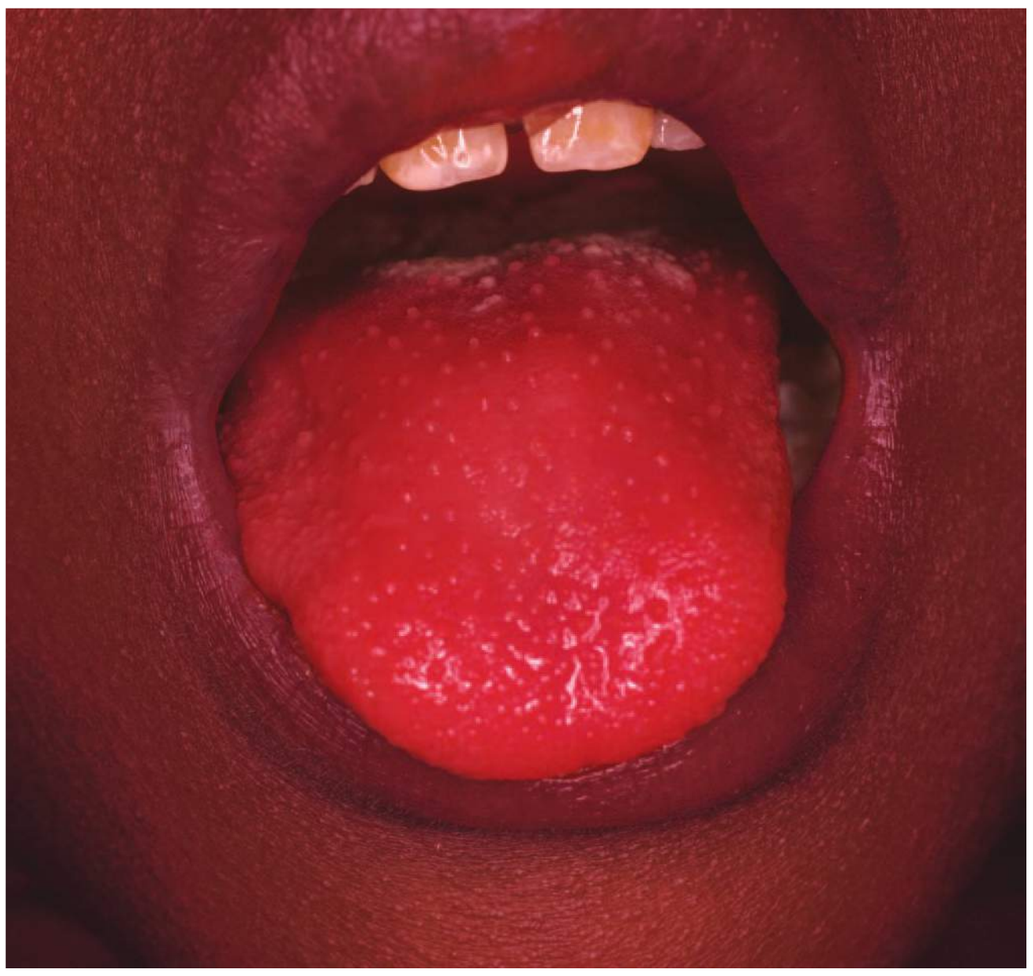


FIGURE 6.49 ■ **Strawberry Tongue.** This patient with scarlet fever demonstrates the bright red appearance of strawberry tongue after most of the white exudate is lost. (Photo contributor: Kevin J. Knoop, MD, MS.)

TORUS PALATINUS

Clinical Summary

Tori are benign nodular overgrowths of cortical bone, occurring in the midline of the palate where the maxilla fuses (palatinus) or on the mandible, typically on the lingual aspect of the molar teeth (mandibularis). Although their physical appearance may be alarming, there is generally no need for concern. Tori are covered by a thin epithelium, which is easily traumatized and ulcerated. These ulcerations tend to heal very slowly because of the poor vascularization of the tori. Torus palatinus, in particular, is slow growing and may occur at any age; however, it is most commonly noted prior to age 30 in adults, and affects women twice as frequently as men. There are a variety of oral conditions that may be confused with mandibular or palatal tori. Gingival fibromatosis, fibroma formation secondary to irritation, granulomas, abscesses, and oral neurofibromatosis located on the palate may resemble torus palatinus. Nodular bony enlargement in the oral cavity may also result from fibrous dysplasia, osteomas, and Paget disease. Oral malignancies may manifest themselves on the palate as primary lesions, though these are rare. Oral radiographs, CT scans, and biopsy may aid in differentiating these conditions.

Management and Disposition

Tori are normal structural variants and do not represent an inflammatory or neoplastic process. They require no treatment unless associated with a complication. Tori may enlarge enough to interfere with eating or speaking, and impair proper fitting of dental prostheses. For some patients, the mere presence of tori may be bothersome and undesirable. Referral is indicated for suspected malignancies or lesions of questionable origin.

Pearl

1. Torus palatinus and mandibularis are nontender and otherwise asymptomatic unless traumatized.



FIGURE 6.50 ■ **Torus Palatinus.** Note the nodular appearance and characteristic central palatal location. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 6.51 ■ **Torus Palatinus.** Due to location, abrasions and ulcerations can occur on the thin overlying epithelium secondary to trauma. (Photo contributor: R. Jason Thurman, MD.)

Clinical Summary

Gingival hyperplasia, or overgrowth of the gingiva, is a relatively rare condition. Most commonly, gingival hyperplasia is drug-induced, with phenytoin, cyclosporine, and calcium channel blockers most commonly implicated. Gingival hyperplasia may also occur from genetic disorders, myelodysplastic syndromes, granulomatous disease, and neoplastic processes, and may occur in pregnancy or puberty. Generally the condition is asymptomatic, but patients may present to the ED due to bleeding gums, traumatized hyperplastic tissue, dental pain, gingival soreness, or associated periodontal disease.

Management and Disposition

ED treatment is geared toward symptomatic relief and identification of the underlying etiology. History may reveal a causative medication, while laboratory testing should rule out underlying blood dyscrasias. Good dental hygiene and dental follow-up are essential. Removal or reduced dosing of the offending agent may be considered. Gingivoplasty or gingivectomy may be necessary in extreme cases.

Pearls

1. Gingival hyperplasia may occur in up to 50% of patients taking phenytoin.
2. Underlying malignancies, especially leukemias, should be considered in the differential diagnosis of patients presenting to the ED with gingival hyperplasia.



FIGURE 6.52 ■ **Gingival Hyperplasia.** This patient presented to the ED complaining of recurrent gingival trauma and bleeding. (Photo contributor: R. Jason Thurman, MD.)

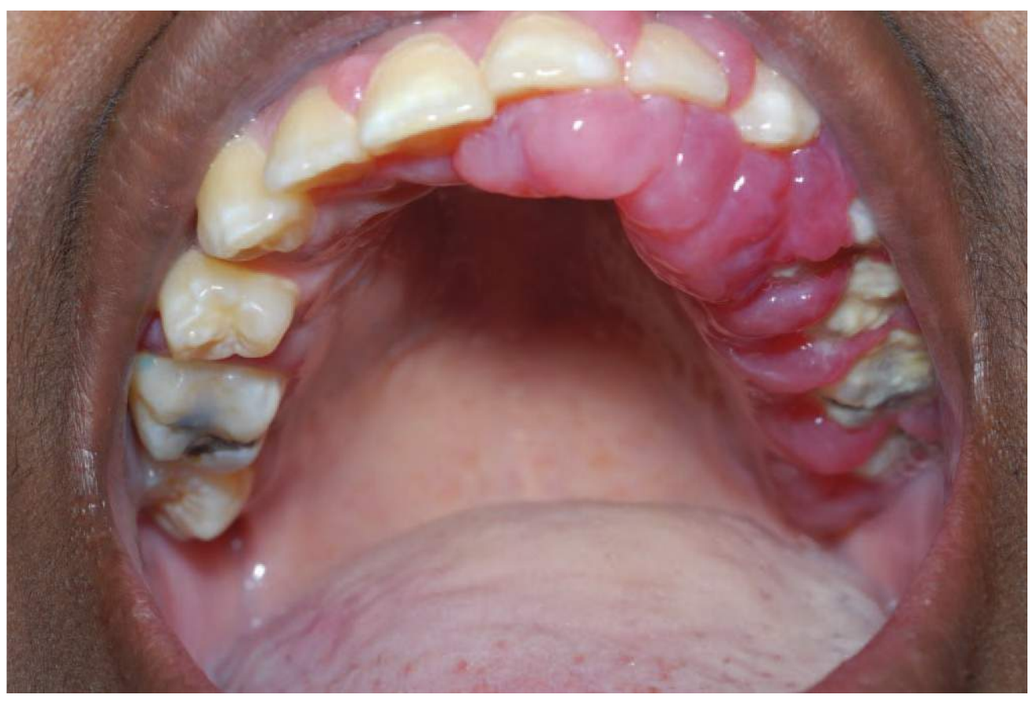


FIGURE 6.53 ■ **Gingival Hyperplasia.** Examination of the oral cavity of the patient in Fig. 6.52 reveals marked hyperplasia of the gingival tissues. (Photo contributor: R. Jason Thurman, MD.)

BLACK HAIRY TONGUE

Clinical Summary

Black hairy tongue (BHT) represents a benign reactive process characterized by hyperplasia and dark pigmentation of the tongue's filiform papillae. The elongated filiform papillae may reach up to 2 cm in length and vary in actual degree of pigmentation from light tan to black. Predisposing factors include excessive smoking, gastroesophageal reflux, poor oral hygiene, chemotherapy, and the use of broad-spectrum oral antibiotics. Pigment from consumed food, beverages, and tobacco products stains the entrapped food debris and desquamated papillary keratin. Some antibiotics may alter normal oral microflora and promote the growth of chromogenic organisms, also contributing to the tongue's discoloration. The darkly pigmented filament-like papillae give the tongue a black, hairy appearance. Males are more often affected than females; this condition very rarely occurs in children. Patients with BHT frequently note alterations of taste perception and fetid breath.

Geographic tongue and orolingual candidiasis may resemble more lightly pigmented forms of BHT. Similarly, dark discoloration of normal tongue papillae may also mimic BHT. This exogenous pigmentation of normal papillae may come from ingested food dyes and certain medications, such as bismuth-containing compounds, ketoconazole, and azidothymidine. The lack of hyperplastic filiform papillae with additional pigmentation of other oral mucosal surfaces may aid in distinguishing these conditions.

Management and Disposition

Improved oral hygiene with gentle tongue brushing and a reduction in the ingestion of exogenous pigment-containing substance represent the cornerstones of treatment. Removal of other predisposing factors (eg, antibiotic withdrawal and smoking cessation) will also promote resolution of this condition. The use of topically applied retinoid preparations and antifungal agents has been advocated for more refractory instances.

Pearls

1. BHT involves the superior aspect of the tongue.
2. The tongue is not always black and can be a tan or yellow color.



FIGURE 6.54 ■ **Black Hairy Tongue.** Hyperplasia of the filiform papillae on the dorsum of the tongue accompanied by deposition of dark pigment is characteristic of black hairy tongue. (Photo contributor: Department of Dermatology, National Naval Medical Center, Bethesda, MD.)

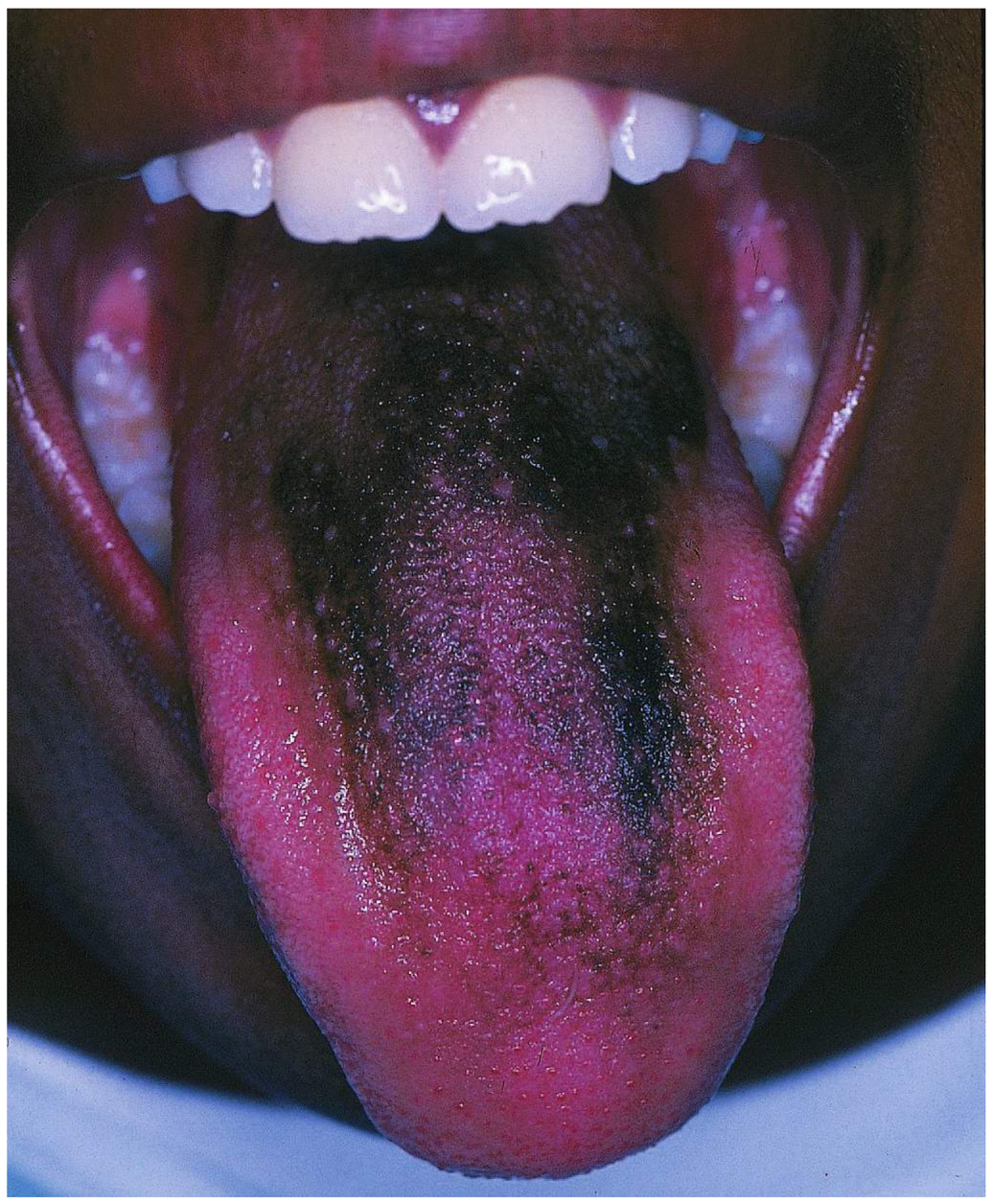


FIGURE 6.55 ■ **Black Hairy Tongue.** Deposition of black pigment secondary to bismuth ingestion. This patient ingested Pepto-Bismol. (Photo contributor: Kevin J. Knoop, MD, MS.)

Clinical Summary

Facial piercings are increasingly common. While external piercings, such as ear and eyebrow rings, are visibly apparent, intraoral and extraoral piercing may not be readily visible on initial inspection. An awareness and suspicion for recent lip, labret, tongue, or other oral mucosal piercings should be performed in any trauma patient as they may cause intraoral damage, produce artifact and diminished CT scan quality if not removed, and interfere during intubation. Common nontraumatic complications of intraoral include peri-piercing cellulitis, abscess formation, hemorrhage, penetrating wounds, dental injury, and granuloma formation around a stud. Though infrequent, systemic infection may result. If the ball comes off the post, it may be swallowed or, rarely, aspirated. Playing with tongue rings may result in lingual surface tooth abrasion, erosion, chipping, fracture, and gingival resorption.

Management and Disposition

Piercing should be removed prior to any CT scan if possible. For localized hemorrhage after a recent piercing, direct pressure should be applied. If bleeding persists, consider removal of the piercing. Any infection surrounding a piercing warrants removal of the stud as it will serve as a nidus for continued infection. Chest radiographs are required to evaluate for stud aspiration. Advise patients with chipped teeth to remove the bar or shorten the post.

Pearls

1. Remove piercings prior to cross-sectional imaging.
2. Piercings may act as a nidus for ongoing infection if not removed.
3. Ensure the prompt identification and removal of intraoral and extraoral piercings in any trauma patient potentially requiring emergent airway management, as the presence of jewelry can be a hindrance to successful intubation.



FIGURE 6.57 ■ **Oral Piercing Complication.** This patient suffered a painful injury when the stud of her lip ring came off, resulting in the penetration of her gingiva and inability to open her mouth. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 6.56 ■ **Tongue Piercing Cellulitis.** This patient presented with fever and tongue pain surrounding his piercing site. Note the erythema and swelling of the tongue and sublingual areas extending from the piercing site (central bubble). (Photo contributor: David Effron, MD.)



FIGURE 6.58 ■ **Oral Piercing Complication**. A close-up photograph of the oral piercing complication seen in Fig. 6.57. (Photo contributor: Lawrence B. Stack, MD.)

Chapter 7

CHEST AND ABDOMEN

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Scan QR code or go to www.ematlas.com for video related to chapter



Penetrating Chest Trauma. Victim of an assault suffered multiple gunshot wounds to his right chest. (Photo contributor: Lawrence B. Stack, MD.)

TRAUMATIC ASPHYXIA

Clinical Summary

The clinical findings of traumatic asphyxia are due to a sudden increase in intrathoracic pressure against a closed glottis. The elevated pressure is transmitted to the veins, venules, and capillaries of the head, neck, extremities, and upper torso, resulting in capillary rupture. Strangulation and hanging are common mechanisms. Survivors demonstrate plethora, ecchymoses, petechiae, and subconjunctival and retinal hemorrhages. Severe injuries may produce central nervous system injury with blindness, seizures, posturing, and paraplegia.

Management and Disposition

Treatment is supportive, with attention to other concurrent injuries. Long-term morbidity is related to the associated injuries.

Pearls

1. Facial petechiae are known as Tardieu spots.
2. One should be alert for associated rib and vertebral fractures.
3. Perthes syndrome is traumatic asphyxia following thoracic crush injury.



FIGURE 7.1 ■ **Traumatic Asphyxia.** This 45-year-old man was pinned when the truck he was working under fell on his chest. He was unable to breathe for 3 to 4 minutes until his coworkers rescued him. The violaceous coloration of the shoulders, face, and upper chest is apparent. (Photo contributor: Stephen W. Corbett, MD.)



FIGURE 7.2 ■ **Traumatic Asphyxia.** A closer view showing the petechial nature of this rash. The patient was observed in the hospital overnight and recovered completely. (Photo contributor: Stephen W. Corbett, MD.)



FIGURE 7.3 ■ **Tardieu Spots.** Facial petechiae, also known as Tardieu spots, are seen in this child after an accidental strangulation injury. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 7.4 ■ **Subconjunctival Hemorrhages.** Bilateral subconjunctival hemorrhages are seen after an intentional strangulation injury. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

A flail chest occurs when multiple rib fractures allow a section of the thoracic cage to move independently. The negative inspiratory pressure created by the diaphragm is less effective since the flail segment paradoxically moves in, creating problems with adequate ventilation. Pulmonary contusion, hemothorax, pneumothorax, and great vessel injuries frequently accompany a flail chest.

Management and Disposition

Pain control and pulmonary toilet are initial standard therapy. Mechanical ventilation should be reserved for those with respiratory compromise, not simply as a mechanism to stabilize the flail segment. Treatment of underlying pulmonary injuries and intensive care unit admission are required for these critically ill patients.

Pearls

1. Intercostal nerve blocks may help provide adequate analgesia so that pain does not prevent the patient from ventilating adequately.
2. Continuous positive airway pressure (CPAP) with patient-controlled analgesia may be as effective as mechanical ventilation.
3. Paradoxical movement of the flail segment is seen during the respiratory cycle in flail chest. As the rib cage expands with inspiration, the flail segment sinks into the chest. As it contracts in expiration, it balloons outward.





FIGURE 7.5 ■ **Flail Chest.** Localized blunt trauma to the left anterior chest 4 cm inferior to the midclavicle, with resultant flail segment. Positive intrathoracic pressure (**A**) and negative intrathoracic pressure (**B**) demonstrate the paradoxical movement of the flail segment. (Photo contributor: Lawrence B. Stack, MD.)

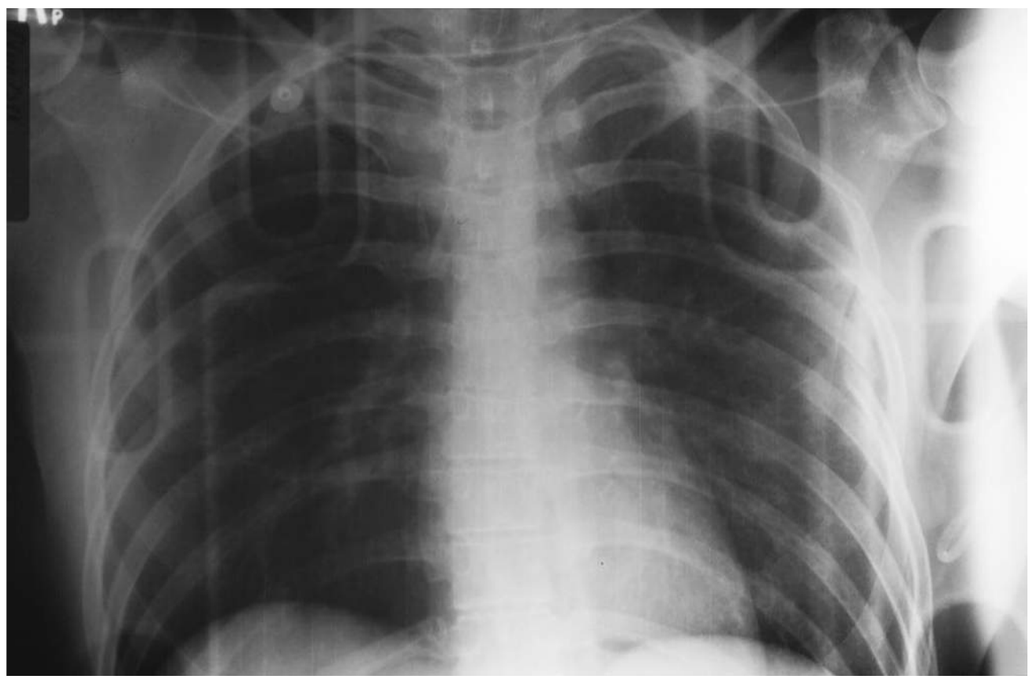


FIGURE 7.6 ■ **Flail Chest: Trauma Chest X-Ray.** Fractures in two places of left ribs 3 and 4 result in a flail segment. Ribs 2 and 5 have single fractures. This is the chest x-ray of the patient in Fig. 7.5. (Photo contributor: Lawrence B. Stack, MD.)

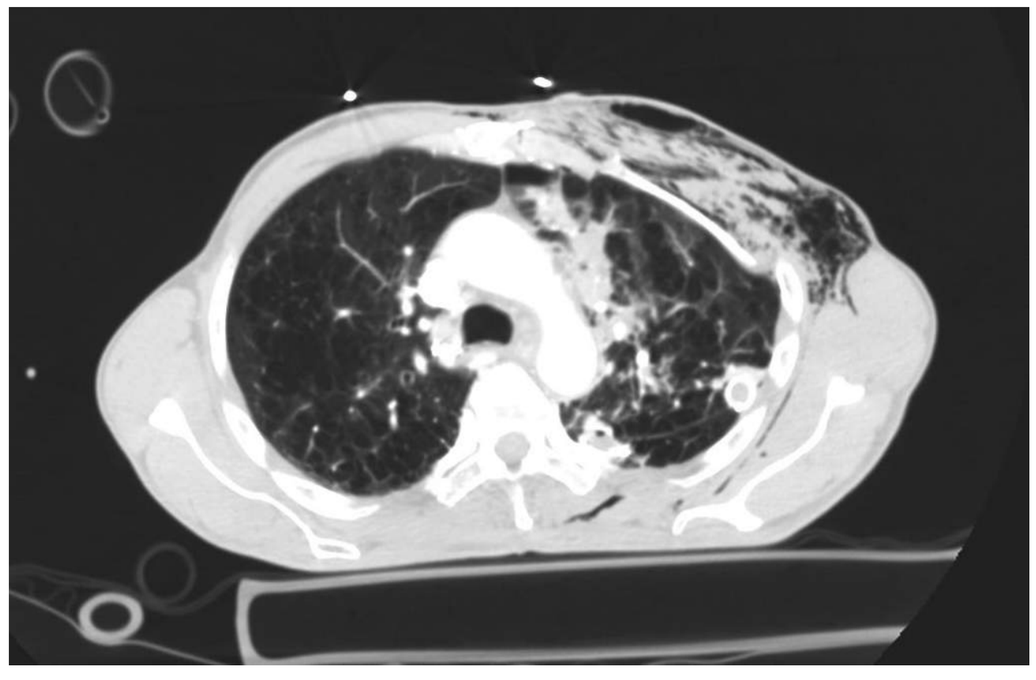


FIGURE 7.7 ■ **Flail Chest: Chest CT Scan.** A flail segment is seen of the left anterior chest on an axial cut of this chest CT scan. A chest tube is seen in the left pleural space. Subcutaneous emphysema is seen in the area of the fractures and tracking posteriorly. This is the chest CT of the patient in Fig. 7.5. (Photo contributor: Lawrence B. Stack, MD.)

STERNOCLAVICULAR DISLOCATION

Clinical Summary

Dislocations of the sternoclavicular joint (SCJ) are uncommon due to the strength of the supporting ligaments. Anterior dislocations are nine times more frequent than posterior dislocations. Posterior dislocations are clinically more important due to the potential for injury to underlying structures. SCJ dislocations typically occur from motor vehicle crashes and sports injuries. Examination findings of an anterior SCJ dislocation include tenderness and prominence of the proximal clavicle from the sternum. The arm is often held in adduction. Posterior SCJ dislocations may be more difficult to identify, as frequently they are associated with multiple injures. Pain and depression of the medial clavicle relative to the sternum may be seen. Superior and inferior dislocations may also be seen. CT scan through the SCJ is the best diagnostic study to evaluate this injury.

Management and Disposition

Orthopedic follow-up is required. Closed reduction of anterior SCJ dislocations is performed by placing the patient supine with a sandbag between their shoulders and placing downward pressure directly over the clavicle. A figure-of-eight clavicle harness is applied. Recurrence is common. Closed reduction of posterior SCJ dislocations may be attempted by an orthopedic surgeon by placing a towel clip around the medial clavicle and pulling the clavicle forward. Open reduction is often necessary.

Pearls

1. While typically it requires a tremendous force to cause an SCJ dislocation, spontaneous dislocations have been reported.
2. In the apical lordotic radiograph (serendipity view), the clavicular head will be higher than the unaffected side in anterior and lower than the unaffected side in posterior dislocations.
3. One-fourth of posterior SCJ dislocations result in mediastinal injury; they always require reduction.



FIGURE 7.8 ■ **Sternoclavicular Dislocation.** A 43-year-old man complains of left sternoclavicular pain after direct trauma to the shoulder. Prominent proximal clavicle is seen prompting CT scan confirmation. (Photo contributor: R. Jason Thurman, MD.)

Clinical Summary

Clavicle fractures are common and are classified by location into proximal, middle, and distal thirds. Most clavicle fractures occur in the middle third. Point tenderness and contusion typically overly the fracture site. Deformity is often seen. Proximal fractures are least common and require significant force, and, therefore, they may have underlying life-threatening mediastinal injuries. Displaced distal clavicle fractures suggest injury to the coracoclavicular and acromioclavicular (AC) ligaments requiring orthopedic follow-up.

Management and Disposition

Most clavicle fractures are identified on an AP view of the shoulder. A 45-degree AP cephalad view maybe helpful if AP films are normal and suspicion is high for fracture. A contrast-enhanced chest CT should be performed if a proximal fracture is identified, looking for mediastinal injuries. Nonoperative management using a simple sling, analgesics, and ice is adequate for most injuries. Orthopedic consultation is indicated for any medial fracture, tenting of the skin, fractures with 100% displacement, severely comminuted, and for displaced distal third fractures.

Pearls

1. Tenting requires closed reduction or surgical repair.
2. Stress radiographs (weighted views) help differentiate minimally or nondisplaced distal third fractures from an AC joint separation.
3. Glenoid neck fracture plus a clavicle fracture represents a "floating" shoulder and requires urgent orthopedic consultation.
4. Sixty percent of childhood clavicular fractures are nondisplaced.

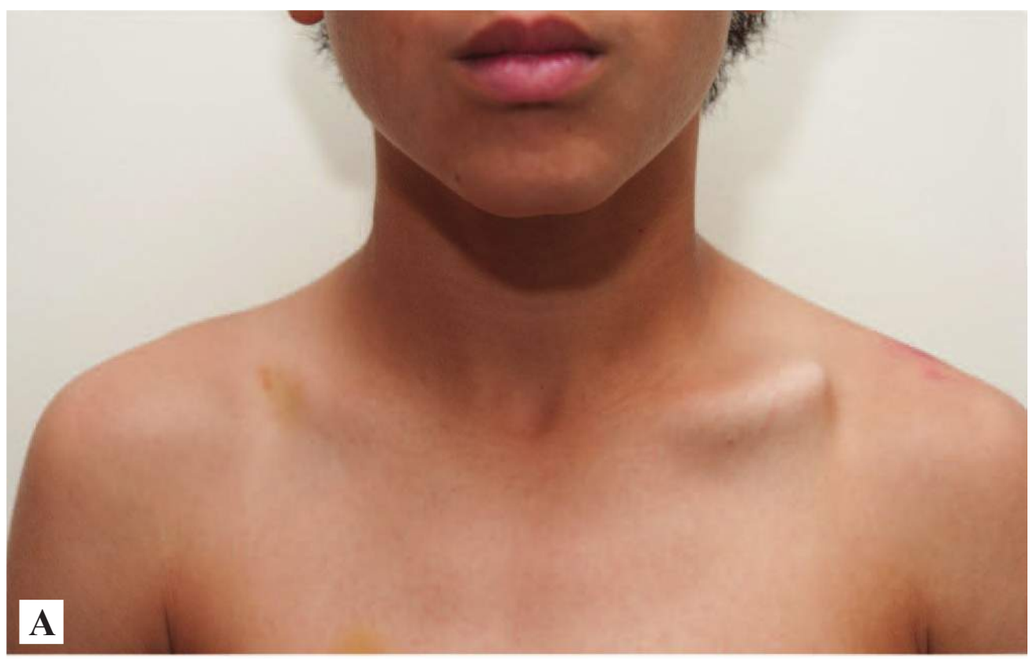


FIGURE 7.9 ■ **Clavicle Fracture.** This 12-year-old boy complains of pain, tenderness, and deformity over the mid left clavicle after an injury incurred during football drills. A "kick-stand" injury is seen on plain films (see Fig. 7.11). Fracture, though overriding, healed without reduction. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 7.10 ■ **Clavicle Fracture Radiograph.** Left mid clavicle fracture with 100% displacement and overriding fragments. A "kick-stand" injury is seen. Fracture, though overriding, healed without complication. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 7.11. ■ **Clavicle Fracture with Skin Tenting.** A 30-year-old unrestrained woman in a motor vehicle crash (MVC) complains of left clavicle pain. Skin tenting is seen. Closed reduction was required to avoid skin ischemia. (Photo contributor: R. Jason Thurman, MD.)

TENSION PNEUMOTHORAX WITH NEEDLE THORACENTESIS

Clinical Summary

A tension pneumothorax results when air enters but does not exit the pleural space. Air in the pleural space accumulates and compresses the ipsilateral lung and vena cava, with a rapid decrease in cardiac output. The contralateral lung may suffer ventilation/perfusion mismatch. Subcutaneous air, tracheal deviation, jugulovenous distention (JVD), and diminished or hyperresonant ipsilateral breath sounds can be clues. Subcutaneous emphysema may be visible on the neck and chest radiographs and is easily diagnosed by palpation. The released air from a tension pneumothorax can be heard escaping from a needle thoracostomy.

Management and Disposition

Treatment requires rapid recognition of the tension pneumothorax, frequently without benefit of chest radiographs. A 14-gauge or larger needle should be placed over the superior rib surface of the second interspace in the midclavicular line. A rush of air with improvement of vital signs confirms the diagnosis. If there is no immediate improvement, do not hesitate to place a second needle in the next interspace. A chest tube should be placed immediately.

Pearls

1. Do not overventilate patients with obstructive pulmonary disease. "Stacking" breaths trap air in the lungs and predispose to bleb rupture and pneumothorax. The pathophysiology of this disease requires a prolonged expiratory phase.
2. The diagnosis of a tension pneumothorax should be made clinically and treated immediately with needle thoracostomy and tube thoracostomy.
3. Tension pneumothorax may be a consequence of positive pressure ventilation in patients with undetected pneumothorax and a persistent air leak such as bronchopleural fistula.
4. Tension pneumothorax is a treatable cause of pulseless electrical activity (PEA).

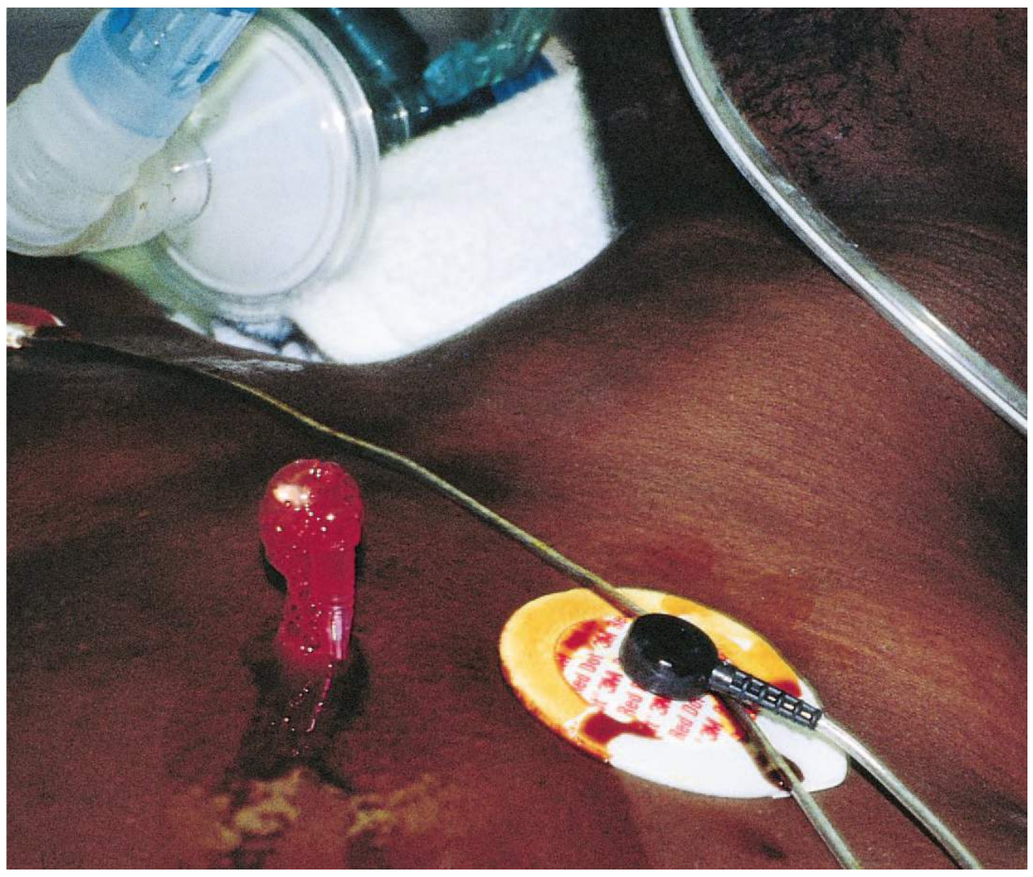


FIGURE 7.12 ■ **Tension Pneumothorax.** A 35-year-old man with severe asthma suffered respiratory arrest during transport by ambulance. He was intubated on arrival but soon became hard to ventilate and developed subcutaneous emphysema followed by hypotension. Needle thoracostomy produced a rush of air and bubbling from the needle with stabilization of vital signs. (Photo contributor: Stephen W. Corbett, MD.)

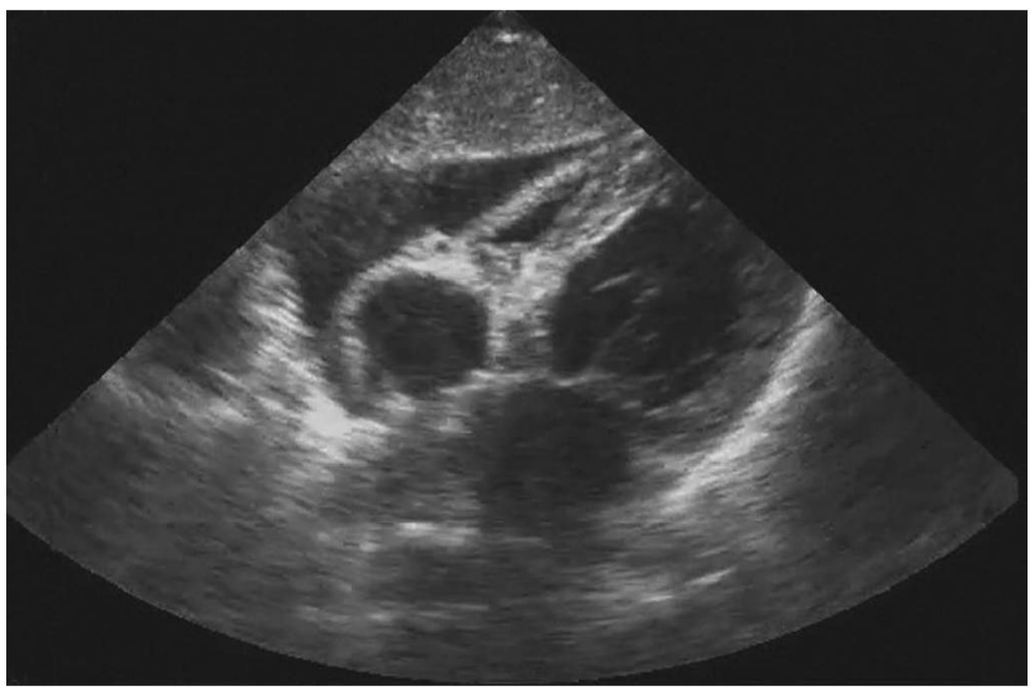


FIGURE 7.13 ■ **Deep Sulcus Sign.** Deepening of the left costophrenic angle is seen. Left to right mediastinal shift is also present. (Photo contributor: Lawrence B. Stack, MD.)

CARDIAC TAMPONADE WITH PERICARDIOCENTESIS

Clinical Summary

Beck's triad of acute cardiac tamponade includes JVD from an elevated central venous pressure (CVP), hypotension, and muffled heart sounds but is present only 10% to 40% of the time. JVD may not be seen in hypovolemic states such as trauma. The simultaneous appearance of all three physical signs is a late manifestation of tamponade and usually seen just prior to cardiac arrest. Symptoms include shortness of breath, orthopnea, dyspnea on exertion, syncope, and symptoms of inadequate perfusion. Causes include trauma (especially penetration wounds), malignancy, hypothyroidism, uremia, myocardial or aortic rupture, and pericarditis. Rapid filling of the pericardial sac (50-100 mL) is more likely to cause compromise.



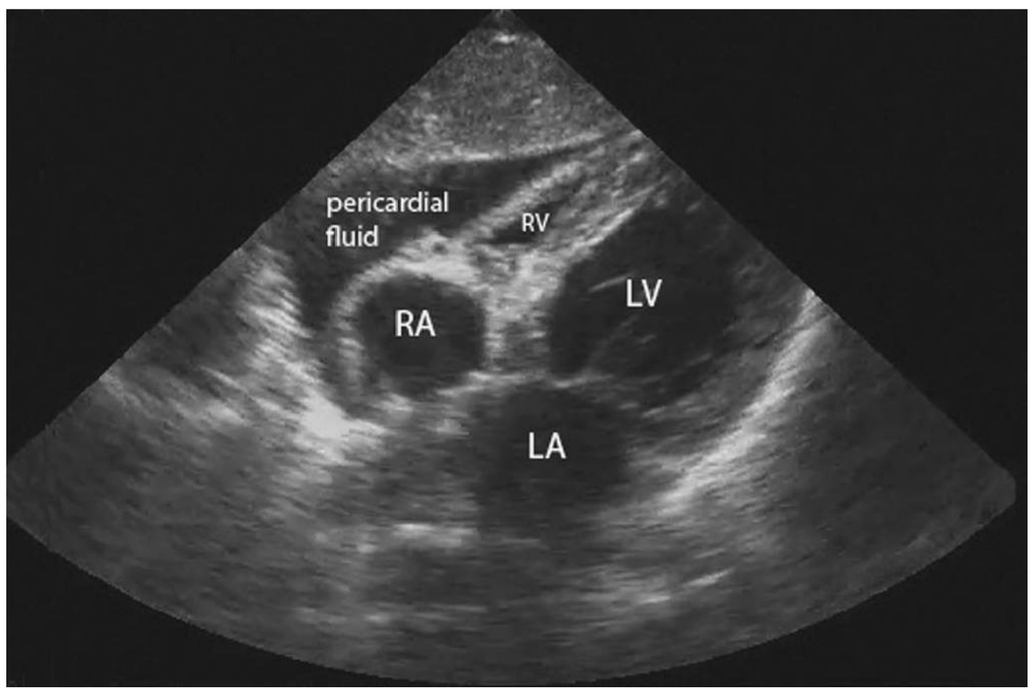


FIGURE 7.14 ■ **Ultrasound of Cardiac Tamponade.** This subxiphoid view of the heart reveals a large pericardial fluid collection and compression of the right ventricle (RV) suggesting tamponade physiology. (Photo contributor: Department of Emergency Medicine, Ultrasound Section, Vanderbilt University.)

Management and Disposition

The clinical diagnosis of tamponade requires suspicion and a careful evaluation of the signs and, when available, imaging techniques. Bedside ultrasonography provides rapid diagnosis and facilitates pericardiocentesis. Goals of emergency department (ED) pericardiocentesis include identification of pericardial effusion and removal of blood or other fluid from the pericardial space to relieve the tamponade.

Pearls

1. Electrical alternans seen on a 12-lead electrocardiography (ECG) suggests pericardial effusion.
2. Cardiac tamponade may cause a narrow pulse pressure, pulsus paradoxus (loss of palpable pulses on inspiration), and low-voltage ECG.
3. Ultrasonographic identification of pericardial fluid in patients with penetration chest trauma may lead to life-saving pericardiocentesis or thoracotomy.
4. Cardiac tamponade is a treatable cause of PEA.

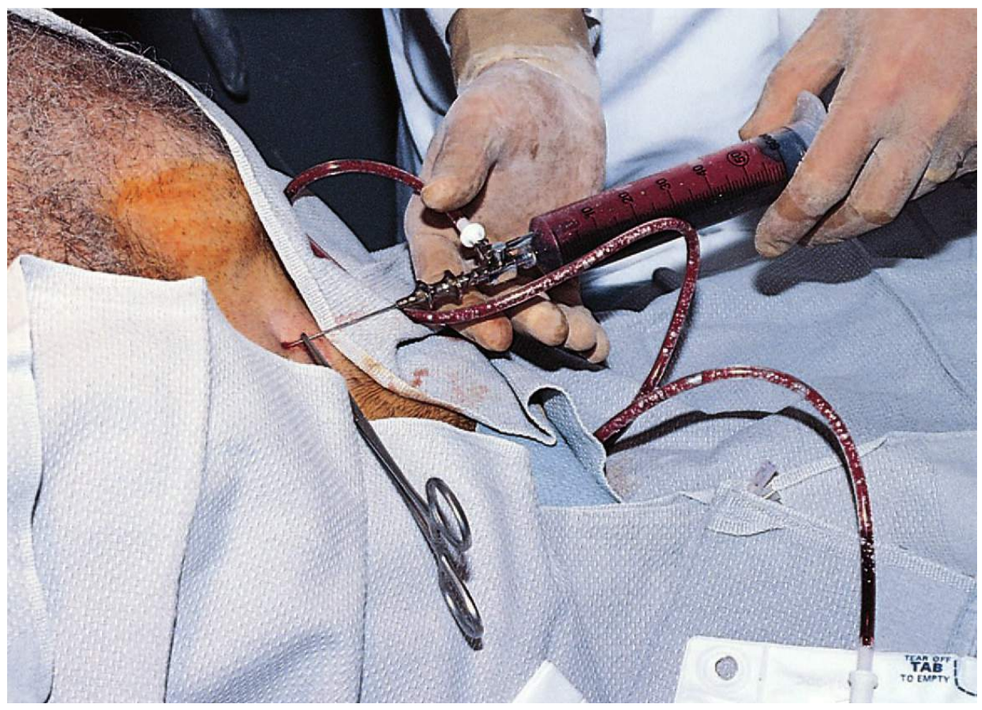


FIGURE 7.15 ■ **Emergency Department Pericardiocentesis.** A positive pericardiocentesis in a patient with a sudden onset of shortness of breath and electrical alternans. (Photo contributor: Lawrence B. Stack, MD.)

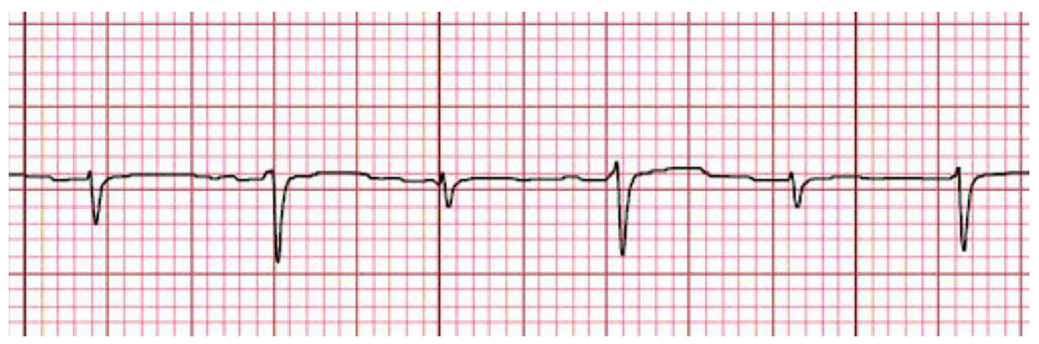


FIGURE 7.16 ■ **Electrical Alternans.** Lead V4 demonstrates a beat-to-beat change in axis, diagnostic of electrical alternans, suggesting pericardial effusion. Low voltage may also be a clue for pericardial effusion. (Photo contributor: R. Jason Thurman, MD.)

Clinical Summary

ED thoracotomy is a resuscitative procedure performed in patients with penetrating chest trauma, who have lost signs of life in the presence of prehospital or ED personnel. Thoracotomy in the ED has specific goals once the chest is opened: relief of cardiac tamponade, support of cardiac function (internal cardiac compressions, cross-clamping the aorta to improve coronary perfusion, and internal defibrillation), and control of hemorrhage from the heart, pulmonary vessels, thoracic wall, and great vessels.

Management and Disposition

Patients with penetrating thoracic trauma who lose their vital signs on arrival or have unstable vital signs despite resuscitation should receive an immediate thoracotomy in the ED. Survival rates following ED thoracotomy for penetrating trauma are 9%; stab wounds fare much better than gunshot wounds. Patients with blunt trauma who lose their vital signs en route to the ED should not undergo an ED thoracotomy, since they rarely survive. Surgical support should be obtained as soon as possible.

Pearls

1. Injuries potentially responsive to ED thoracotomy include cardiac tamponade, pulmonary parenchymal and tracheobronchial injuries, large-vessel injuries, air embolism, and penetrating heart injuries.
2. ED thoracotomy should be performed immediately once the indications have been met, since the likelihood of survival is greater when this is performed earlier in the resuscitation.
3. Stab wounds and measurable ED vital signs are the best predictors of survival.



FIGURE 7.17 ■ **Emergency Department Thoracotomy.** An unsuccessful resuscitative emergency department thoracotomy with pericardiotomy in a patient with penetrating chest trauma who lost signs of life in the field after the paramedics arrived at the scene. (Photo contributor: Lawrence B. Stack, MD.)

SEAT BELT INJURY

Clinical Summary

Two- and three-point seat belt restraints have reduced mortality and the severity of trauma due to motor vehicle crashes; however, they occasionally produce injury. Lap/shoulder belts are known to produce abdominal (most common), thoracic, and spinal injuries. The "seat belt sign," abrasions or ecchymoses to the neck, chest, and abdomen in the pattern of the belt, occurs in less than 20% of patients, but is associated with a four-fold risk of intrathoracic and an eight-fold risk of intra-abdominal injury.

Management and Disposition

Patients with a mechanism for significant trauma or with other injuries requiring admission need to undergo a careful search for related injures. Patients discharged home from the ED should be given appropriate precautions to monitor for a delayed injury presentation.



FIGURE 7.18 ■ **Seat Belt Injury.** Ecchymosis from the lap belt is evident. A subtle Destot's sign, which is inguinal or perineal ecchymosis, in a patient with a pelvis fracture. (Photo contributor: R. Jason Thurman, MD.)

Pearls

1. Up to 36% of those with a seat belt sign have an abdominal injury requiring laparotomy.
2. Lap-belt-only passengers are more likely to have Chance fractures and small bowel injury if they have an abdominal seat belt abrasion after a motor vehicle crash.

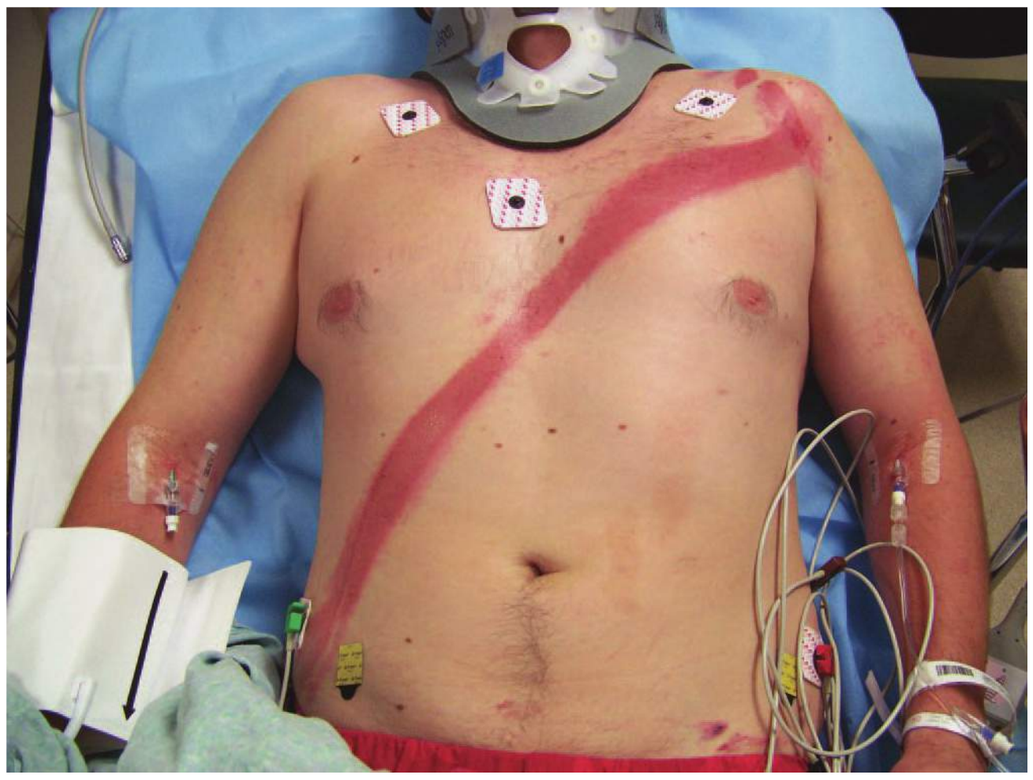


FIGURE 7.19 ■ **Seat Belt Injury.** Abrasions from a three-point restraint causing rib fractures and a pneumothorax. (Photo contributor: Brad Russell, MD.)



FIGURE 7.20 ■ **Seat Belt Injury.** Neck abrasions from a three-point restraint in a patient involved in a head-on motor vehicle crash. (Photo contributor: David Effron, MD.)

Clinical Summary

Bluish to purplish periumbilical discoloration (Cullen sign) and flank discoloration (Grey Turner sign) represent retroperitoneal hemorrhage that has dissected through fascial planes to the skin. Retroperitoneal blood may also extravasate into the perineum, causing a scrotal hematoma or inguinal mass. This hemorrhage may represent a hemodynamically significant bleed.

Cullen sign and Grey Turner sign are most frequently associated with hemorrhagic pancreatitis and are seen in 1% to 2% of cases, and typically are seen 2 to 3 days after onset. These signs may also be seen in ruptured ectopic pregnancy, severe trauma, leaking or ruptured abdominal aortic aneurysm, coagulopathy, or any other condition associated with bleeding into the retroperitoneum.

Management and Disposition

Treatment of patients with Grey Turner sign or Cullen sign depends on the etiology. Because the hemorrhage may represent a hemodynamically significant bleed, cardiovascular stabilization after airway stabilization is of the utmost importance. Once the patient has been stabilized, the source of bleeding can be elicited by selected laboratory tests (complete blood cell count, amylase, lipase, human chorionic gonadotropin, and diagnostic studies [ultrasound, computed tomography]). Because of the severity of diseases associated with Grey Turner and Cullen signs, these patients are usually admitted to the hospital.

Pearls

1. Mortality rate in patients with Cullen or Grey Turner sign may be as high as 37%.
2. These signs are typically seen 2 to 3 days after the acute event.
3. These signs are seen in only 1% to 2% of patients with hemorrhagic pancreatitis.



FIGURE 7.21 ■ **Grey Turner and Cullen Signs.** This patient displays both flank and periumbilical ecchymoses characteristic of Grey Turner and Cullen signs. (Photo contributor: Michael Ritter, MD.)



FIGURE 7.22 ■ **Grey Turner Sign.** A 68-year-old man with flank ecchymosis due to hemorrhagic pancreatitis. (Photo contributor: Stephen W. Corbett, MD.)

IMPALED FOREIGN BODY

Clinical Summary

Stab wounds cause injury to tissue in their path. Stab wounds to the chest, in addition to causing pneumothorax or hemothorax, may also cause life-threatening injuries to the heart and major blood vessels. One quarter of anterior stab wounds do not penetrate the peritoneum. Half of those that do penetrate require no surgical repair. For these reasons, local exploration, diagnostic peritoneal lavage (DPL), focused assessment with sonography for trauma (FAST), and serial examinations are often used as a management strategy. Penetrating flanks may be evaluated with triple-contrast computed tomography (CT). The size of the external wound frequently underestimates the internal injury. Impaled foreign bodies to the chest or abdomen pose a complex problem. The object inflicting the injury may also be preventing significant blood loss and therefore should be removed by the trauma surgeon in the operating room.

Management and Disposition

Initial stabilization of the patient (intravenous fluid resuscitation, oxygen, and monitoring), obtaining appropriate laboratory studies including blood type and cross-matching, and resource mobilization (trauma team) are important steps in the initial management of penetrating chest or abdominal trauma. Prior to surgical evaluation, stabilization of the impaled foreign object should be performed to prevent further injury.

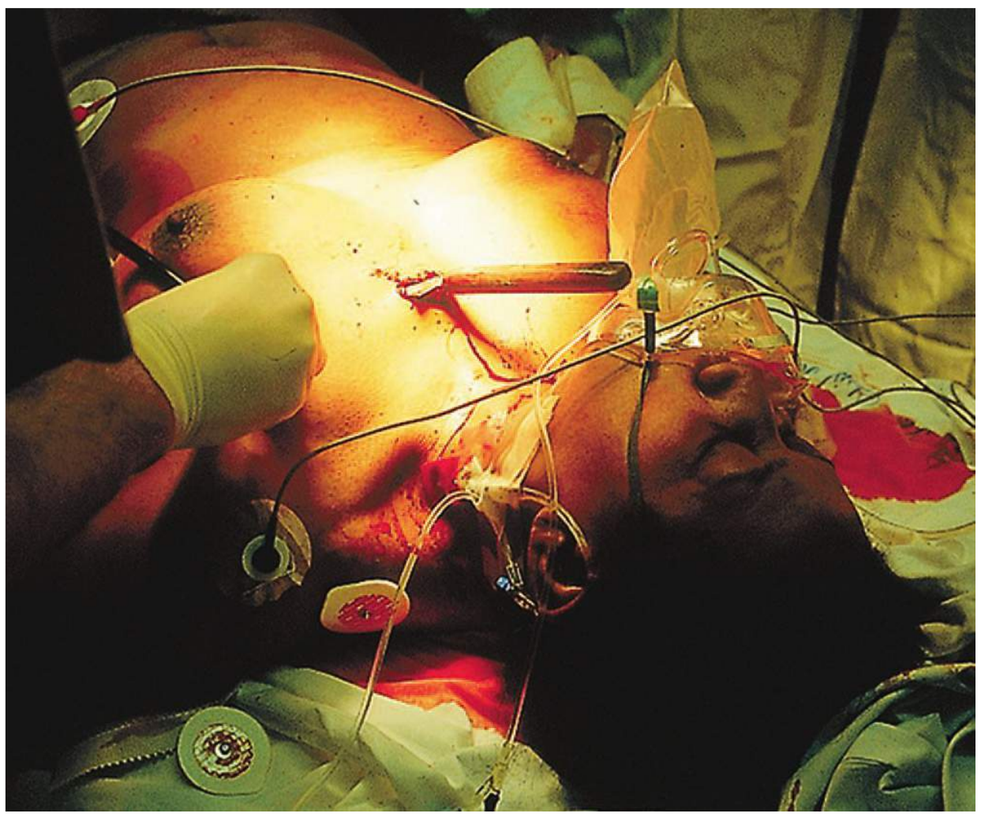


FIGURE 7.23 ■ **Impaled Chest Wound.** This patient was stabbed in the chest with a butcher knife in a family dispute. The knife was stabilized by EMS providers at the scene and removed in the operating room. Injury was isolated to the right atrium. (Photo contributor: Kevin J. Knoop, MD, MS.)

Pearl

1. Impaled chest or abdominal foreign bodies should be removed only by a trauma surgeon in a controlled setting.

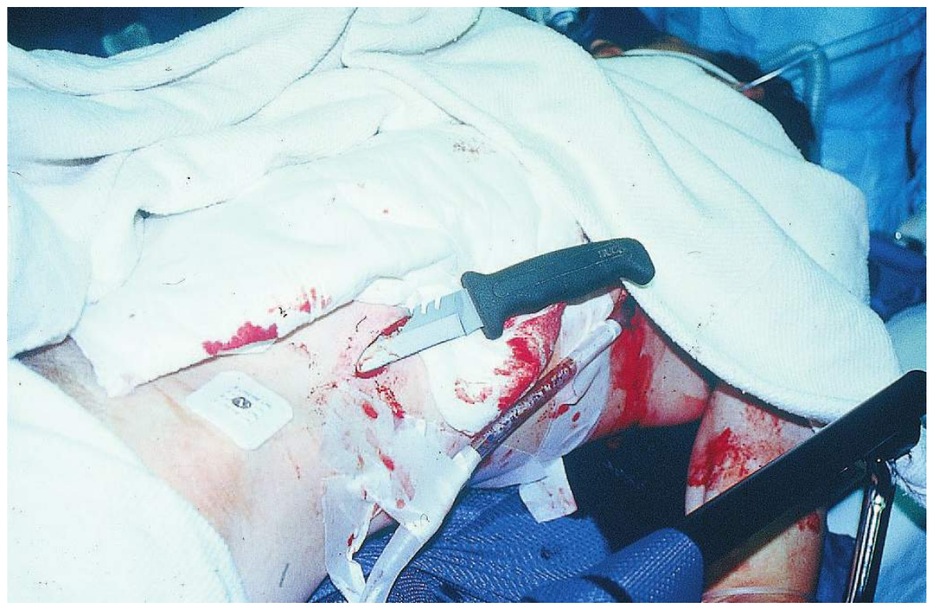


FIGURE 7.24 ■ **Impaled Abdominal Foreign Body.** Impaled knife to the left abdomen after a domestic argument. (Photo contributor: Ian D. Jones, MD.)



FIGURE 7.25 ■ **Impaled Nail in the Abdomen.** Impaled 16-penny nail to the right abdomen after an accidental discharge from a pneumatic nail driver. The nail penetrated the peritoneal cavity, but did not injure any organ. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 7.26 ■ **Impaled Chest Foreign Body.** Impaled car window frame to the left chest causing rib fractures and a pulmonary laceration. (Photo contributor: R. Jason Thurman, MD.)

ABDOMINAL EVISCERATION

Clinical Summary

Evisceration of abdominal contents usually occurs after a stab or slash wound to the abdomen. It is an indication for laparotomy. Other indications for laparotomy in penetrating abdominal trauma include unexplained shock and evidence of blood in the stomach, bladder, or rectum.

Consideration of the anatomic boundaries of the abdomen is important in differentiating abdominal injuries from penetrating chest or retroperitoneal injuries.

Management and Disposition

Initial stabilization (intravenous fluid resuscitation, oxygen, and monitoring), obtaining appropriate laboratory studies including a blood type and cross-matching, and resource mobilization (notifying surgical team, operating room, and anesthesiology) are important steps in the initial management of penetrating abdominal trauma. Eviscerated bowel should be covered with saline-soaked gauze. Antibiotics with enteric coverage should be provided.

Pearls

1. Evisceration, unexplained shock, or blood in the stomach, bladder, or rectum is an indication for laparotomy.
2. Selected patients with stab wounds to the abdomen and peritoneal penetration may be conservatively observed for delayed complications. Some centers are using a nonoperative approach for patients with gunshot wounds to the abdomen as well.
3. As many as 25% of patients with stab wounds to the abdomen can be discharged from the ED based on a negative wound exploration.
4. Although striking, evisceration should not distract the provider from the ABCs of a thorough resuscitation.



FIGURE 7.27 ■ **Abdominal Evisceration.** Self-induced evisceration with bowel perforation and spillage of food particles is clearly seen in this photograph. This patient went directly to the operating room. (Photo contributor: Lawrence B. Stack, MD.)

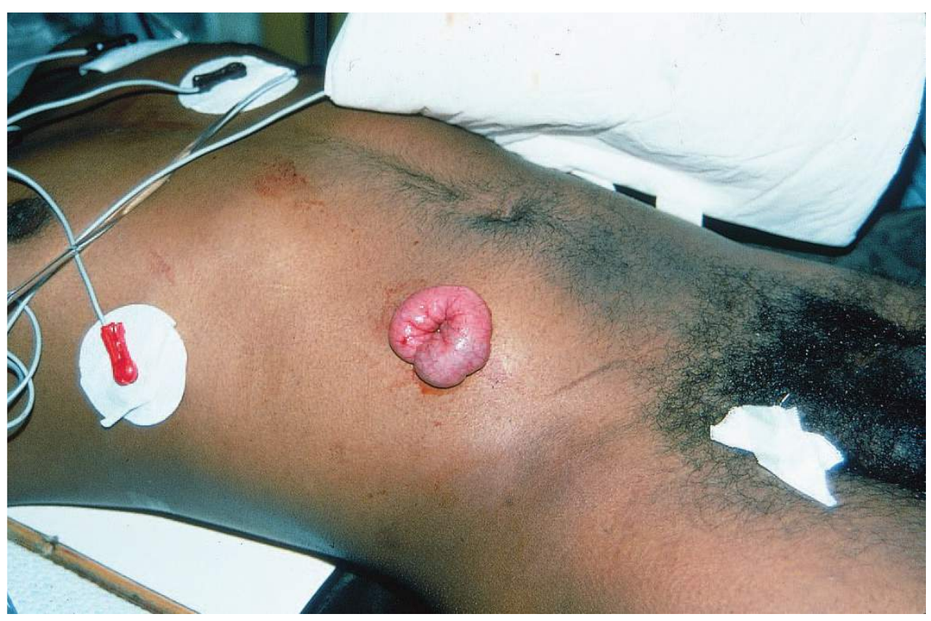


FIGURE 7.28 ■ **Abdominal Evisceration.** Evisceration of small bowel after assault and stab wound to the right lower abdomen. (Photo contributor: Frank Birinyi, MD.)

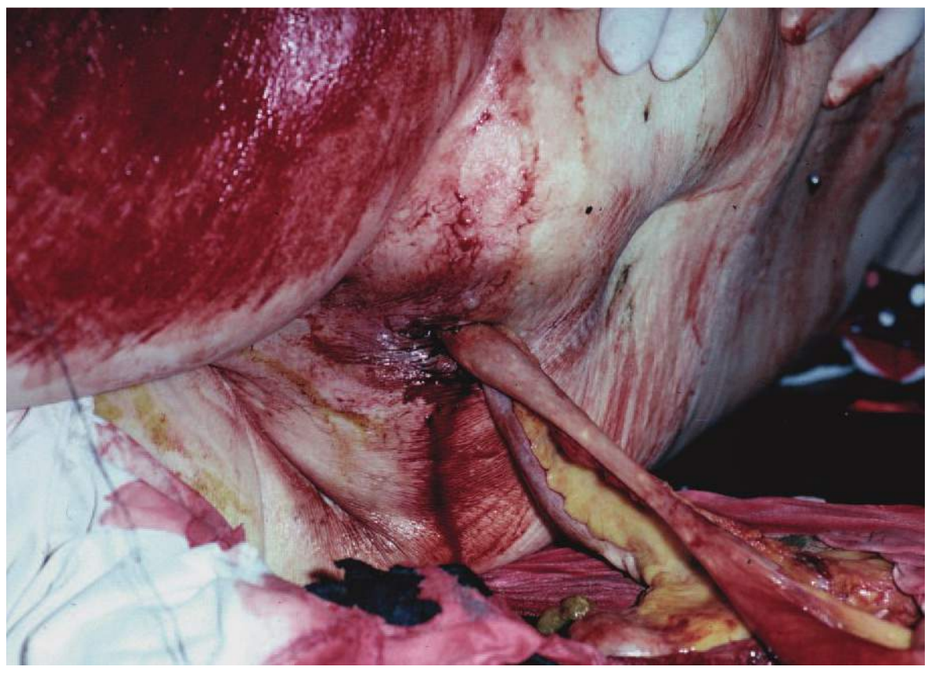


FIGURE 7.29 ■ **Bowel Evisceration through the Anus.** High-energy blunt abdominal trauma to this elderly man resulted in evisceration of small bowel and omentum through his anus. (Photo contributor: Alan B. Storrow, MD.)

TRAUMATIC ABDOMINAL HERNIA

Clinical Summary

Blunt traumatic abdominal hernia is defined as herniation through disrupted musculature and fascia associated with adequate trauma, without skin penetration, and no evidence of a prior hernial defect at the site of injury. This occurs when a considerable blunt force is distributed over a surface area large enough to prevent skin penetration but small enough to cause a focal defect in the underlying fascia or muscle wall. Most of these injuries are due to seat belt injuries in motor vehicle crashes; handlebar injuries are the second most common cause. Up to 44% of these patients require bowel resection.

CT of the abdomen and pelvis with intravenous contrast is the preferred diagnostic study for the evaluation of a traumatic abdominal hernia. Ultrasound may play a limited role in the diagnosis of abdominal wall hernia.

Management and Disposition

Identification and treatment of life-threatening associated injuries take priority over the hernia. The hernial defect should be repaired after the patient has been stabilized.

Pearls

1. Abdominal hernia due to blunt trauma is a rare injury, most frequently due to seat belt injuries in motor vehicle crashes.
2. CT scan is the preferred diagnostic study for abdominal wall hernias.

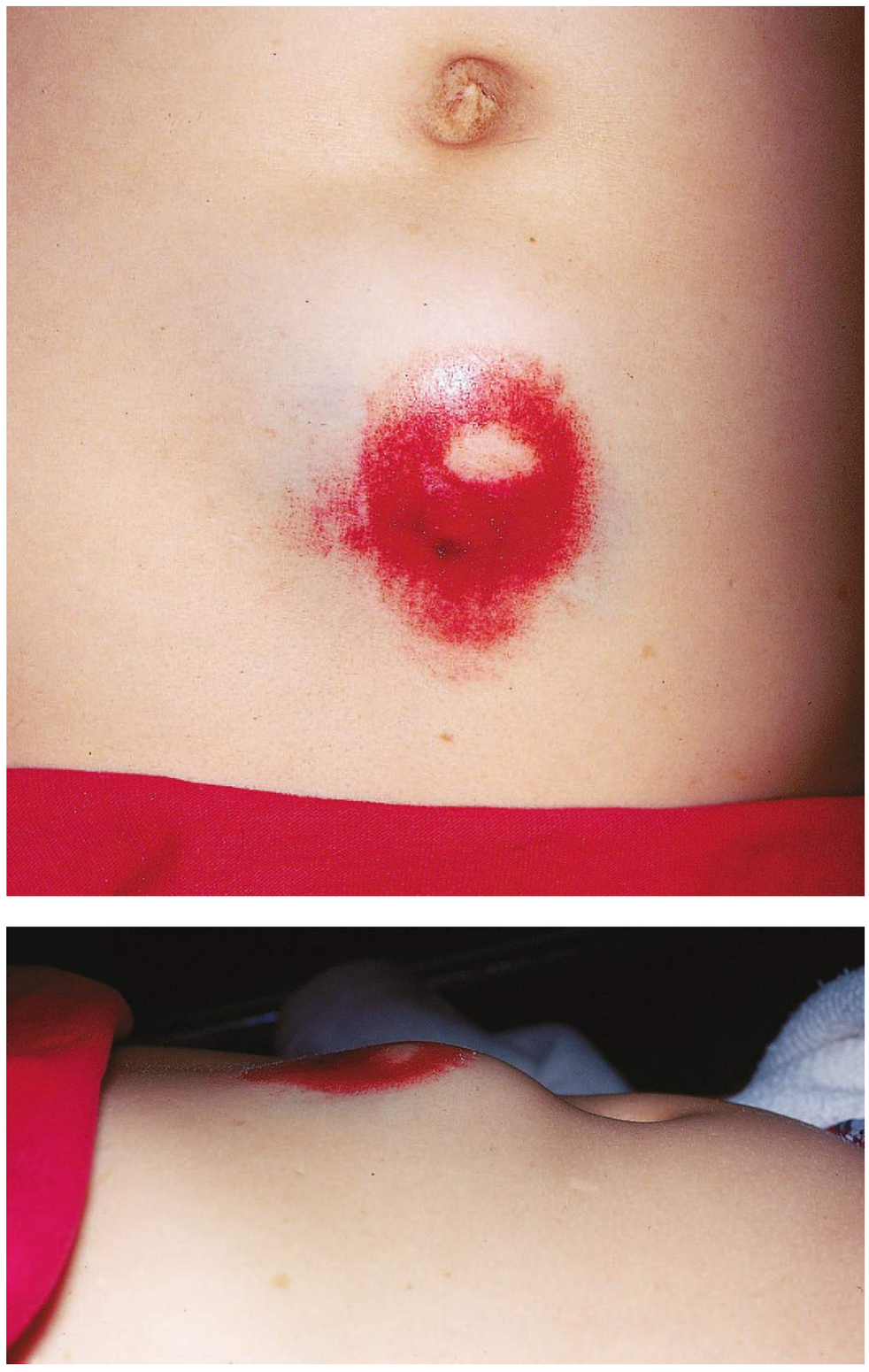


FIGURE 7.30 ■ **Traumatic Abdominal Wall Hernia.** This 5-year-old boy suffered a traumatic hernia from a handlebar injury. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 7.31 ■ **CT Scan, Abdominal Wall Hernia.** Abdominal contents are seen extruding through a fascial defect. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Pelvic fractures are most often the result of motor vehicle crashes or falls and are fraught with complications. The pelvis should be regarded as a ring; identification of one fracture or dislocation should prompt surveillance for another. Trauma to the genitourinary (GU) tract is suggested by blood at the urethral meatus, a high-riding prostate, gross hematuria, or scrotal hematoma. Spinal nerves, the lumbosacral plexus, sacral plexus, and the major lower extremity peripheral nerves, such as the sciatic, femoral, obturator, and pudendal nerves, are found in close proximity to the pelvis. A neurologic examination of the lower extremities should include a rectal examination to assess tone and temperature sensation. The iliac arteries, veins, and their branches are also enveloped by the bony architecture of the pelvis and severe hemorrhage is a common complication. While ecchymosis of the anterior abdominal wall, flank, sacral, or gluteal region suggests hemorrhage, there may be no outward signs of a severe hemorrhage. Blood found during rectal or vaginal examination may indicate a puncture wound from the fracture.

The presence of a pelvic fracture is associated with mortality rates as high as 20% (5% in children).

Management and Disposition

All patients with a suspected pelvic fracture should undergo a radiographic evaluation with an anteroposterior (AP) x-ray of the pelvis. Fractures through the sacrum are subtle and may be appreciated by noting disruption of the sacral arcuate lines or sacroiliac joint widening. A CT scan is often necessary to identify associated injuries. A retrograde urethrogram may also be necessary if a GU injury is suspected. Angiography by interventional radiology with selective embolization may be performed to control arterial bleeding. In the face of a widened pubic symphysis or "open book" pelvic fracture and continued hemodynamic instability, orthopedic consultation for emergent external fixation can help to reduce blood vessel tension and reduce hemorrhage.

Pearls

1. A pelvic binder or a sheet tied around the pelvis may be used to temporarily stabilize pelvic fractures.
2. Posterior pelvic fractures are more likely to result in neurovascular injuries, while anterior pelvic fractures are more likely to cause urogenital injuries.
3. FAST exam is less sensitive (26%) in detecting hemoperitoneum in patients with pelvic fractures. A negative FAST is therefore inadequate to eliminate the need for laparotomy in an unstable patient.



FIGURE 7.32 ■ **Pelvic Fracture.** Scrotal and perianal ecchymosis is seen in this patient with a vertical shear pelvic fracture due to a fall. (Photo contributor: Lawrence B. Stack, MD.)

RESPIRATORY RETRACTIONS

Clinical Summary

Increased respiratory effort may be manifested by increased respiratory rate, increased chest wall excursion, and retractions of the less rigid structures of the thorax. Retractions of the sternum, suprasternal notch, intercostal retractions, and paradoxical abdominal movement reflect increased respiratory effort. This may be due to obstructive disease such as asthma or upper airway obstruction, pneumonia, or restrictive disease. The presence of stridor, wheezing, or rhonchi will help distinguish the cause.

Management and Disposition

An aggressive search for the cause of the retractions is required to direct therapy. Rapid evaluation of the airway for patency and breathing for oxygenation should be done immediately on presentation. High-flow oxygen by face mask is appropriate for patients in respiratory distress. Preparations for securing an airway should be underway for those patients in severe distress or respiratory failure. Arterial blood gas analysis may help identify impending respiratory failure. Routine measures for the mildly symptomatic patient depend on the cause of the retractions. For asthma or exacerbations of chronic obstructive pulmonary disease (COPD), nebulized β_2 agonists and steroid therapy may be appropriate. Patients with croup may require nebulized epinephrine or dexamethasone as initial therapy. Foreign-body aspiration requires imaging and consultation for confirmation of the suspected diagnosis and removal.

Pearls

1. Retractions are best observed with the patient at rest and the chest exposed.
2. Retractions from obstructive airway disease can be intercostal and supraclavicular and are usually accompanied by nasal flaring, increased expiratory phase, and increased respiratory rate.



FIGURE 7.33 ■ **Sternal Retractions.** Sternal retractions in a patient with croup. (Photo contributor: Stephen W. Corbett, MD.)





FIGURE 7.34 ■ **Suprasternal Retractions.** Suprasternal retractions in an adolescent with severe asthma. (Photo contributor: Kevin J. Knoop, MD, MS.)

Clinical Summary

This symptom complex develops from obstruction of venous drainage from the upper body, resulting in increased venous pressure, which leads to dilation of the collateral circulation. Superior vena cava (SVC) syndrome is most commonly caused by malignant mediastinal tumors. Dyspnea; swelling of the face, upper extremities, and trunk; chest pain, cough, or headache may be present. Physical findings include dilation of collateral veins of the trunk and upper extremities, facial edema and erythema (plethora), cyanosis, and tachypnea.

Management and Disposition

Radiation therapy is the optimal treatment for most malignant mediastinal tumors causing SVC syndrome. An exception is small cell carcinoma which responds better to chemotherapy. Elevating the head of the bed, oxygen, and administration of corticosteroids and diuretics initiated in the ED may provide temporary relief pending definitive therapy.

Pearls

1. SVC syndrome is most commonly caused by malignant mediastinal tumors.
2. Treatment of most mediastinal tumors causing SVC syndrome is radiation therapy.
3. CT scan of the chest is the diagnostic modality of choice for patients with SVC syndrome.
4. Signs of decreased cardiac output, cerebral edema, and laryngeal edema are life-threatening findings.



FIGURE 7.36 ■ **Superior Vena Cava Syndrome.** A middle-aged woman with plethora, distended superficial upper thorax veins, and headache with SVC syndrome due to lymphoma.



FIGURE 7.35 ■ **Superior Vena Cava Syndrome.** A 27-year-old man with SVC syndrome. Note the prominent collateral veins of the chest and neck. (Photo contributor: William K. Mallon, MD.)



FIGURE 7.37 ■ **Facial Plethora.** A 53-year-old woman presents with shortness of breath and facial plethora. She has SVC syndrome due to lung cancer. (Photo contributor: R. Jason Thurman, MD.)

SUPRACLAVICULAR ADENOPATHY

Clinical Summary

Adenopathy or masses in the supraclavicular fossa should heighten suspicion for metastatic or locally invasive disease. A Virchow node, also called a sentinel node (Troisier sign), is left supraclavicular node in the area where the thoracic duct enters the SVC. This node, located behind the sternocleidomastoid muscles, suggests metastatic abdominal cancer, particularly gastric cancer spread via lymphatics. Carcinoma affecting right supraclavicular nodes often arises from cancer of the breast or lung and are typically lateral to a Virchow node.

A Pancoast tumor involves the apical lung and may affect contiguous structures such as the brachial plexus, sympathetic ganglion, vertebrae, ribs, SVC, and recurrent laryngeal nerve (more common for left-sided tumors). Horner syndrome, extremity edema, nerve deficits, hoarseness, and SVC syndrome may result. Erosion of tumor through the chest wall can cause compression of venous outflow, with resultant JVD.

Management and Disposition

Treatment depends on the staging and type of tumor. The SVC syndrome can be treated acutely with radiation and diuretics. Thrombolytic therapy has been used successfully in some cases of acute vena caval thrombosis.

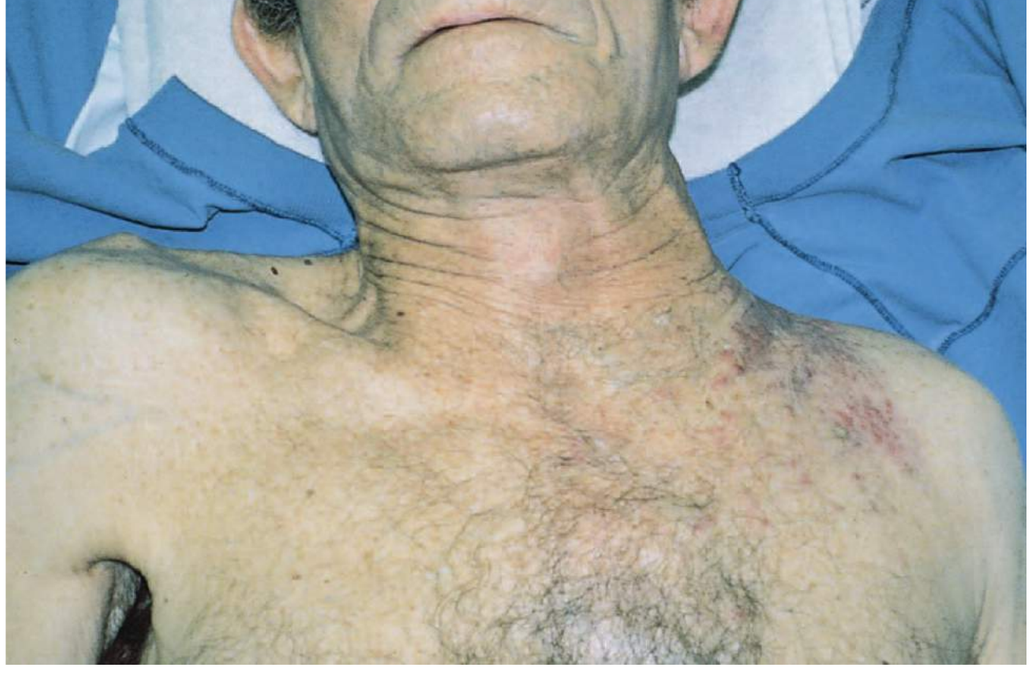


FIGURE 7.38 ■ **Apical Lung Mass.** This 68-year-old man cigarette smoker complained of cough and weight loss. A chest radiograph shows a left apical tumor. There is erosion of the tumor into the chest wall, with an indurated supraclavicular and infraclavicular mass. Moderate JVD is apparent, suggesting venous outflow obstruction. (Photo contributor: Stephen W. Corbett, MD.)

Pearls

1. Thrombosis may cause acute decompensation with edema, plethora, and airway collapse.
2. A Virchow node is made more evident if the patient performs a Valsalva.



FIGURE 7.39 ■ **Virchow Node.** This middle-aged woman presents with multiple complaints including lateral neck swelling. She was diagnosed with abdominal lymphoma. (Photo contributor: David Effron, MD.)



FIGURE 7.40 ■ **Left Supraclavicular Node.** This 16-year-old patient developed left supraclavicular swelling and intermittent fever. She was diagnosed with lymphoma. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 7.41 ■ **Right Supraclavicular Node.** This 25-year-old patient developed right supraclavicular swelling and intermittent fever. She was diagnosed with lymphoma. (Photo contributor: Alan B. Storrow, MD.)

Clinical Summary

Central venous (right atrial) pressure is reflected by distention of the internal or external jugular veins. Distention greater than 4 cm above the sternal angle of Louis with the head of the bed elevated 30 to 60 degrees should be considered abnormal. The presence of crackles, murmurs, rubs, percussed hyperresonance, or crepitus may help disclose the etiology.

The abdominojugular test (previously known as hepatojugular reflux) is performed by looking for JVD with pressure on the right upper quadrant. It is a marker for right ventricle dysfunction, constrictive pericarditis, cardiac tamponade, and tricuspid regurgitation.

Causes of JVD include right ventricular failure, left ventricular failure, biventricular failure, parenchymal lung disease, pulmonary hypertension, pulmonic stenosis, restrictive pericarditis, pericardial tamponade, SVC syndrome, pulmonary embolus, valvular disease, tension pneumothorax, increased circulating blood volume, and atrial myxoma. Temporary venous engorgement may result from Valsalva maneuver, positive pressure ventilation, and Trendelenburg position.

Management and Disposition

Treatment varies depending on the cause. Preload reduction may help in cases of congestive heart disease. Reversal of a traumatic etiology with needle thoracostomy or pericardiocentesis may be required.

Pearls

1. Right-sided myocardial infarction may produce JVD with clear lung fields.
2. JVD may be absent in the presence of the above-listed causes if hypovolemia is present.



FIGURE 7.42 ■ **Jugulovenous Distention.** An engorged external jugular vein is noted as it crosses the sternocleidomastoid muscle into the posterior triangle of the neck and disappears beneath the clavicle to join the brachiocephalic vein and the superior vena cava. This patient has severe congestive heart failure. (Photo contributor: Lawrence B. Stack, MD.)

CAPUT MEDUSAE

Clinical Summary

Veins of the abdomen normally are scarcely visible within the abdominal wall. Engorged veins, however, are often visible through the normal abdominal wall. Engorged veins forming a knot in the area of the umbilicus are described as caput medusae. The extent of associated findings depends on the underlying etiology. It is usually secondary to liver cirrhosis, with subsequent portal hypertension and development of circulation circumventing the liver.

Management and Disposition

Treatment is directed at the underlying cause. This finding by itself does not require acute treatment.

Pearl

1. Caput medusae have the same clinical significance as the more common pattern of venous engorgement.
2. If pressure on a prominent abdominal wall vein results in flow of blood to the head, the likely cause is inferior vena cava obstruction. If the flow is to the feet, it is caput medusa.

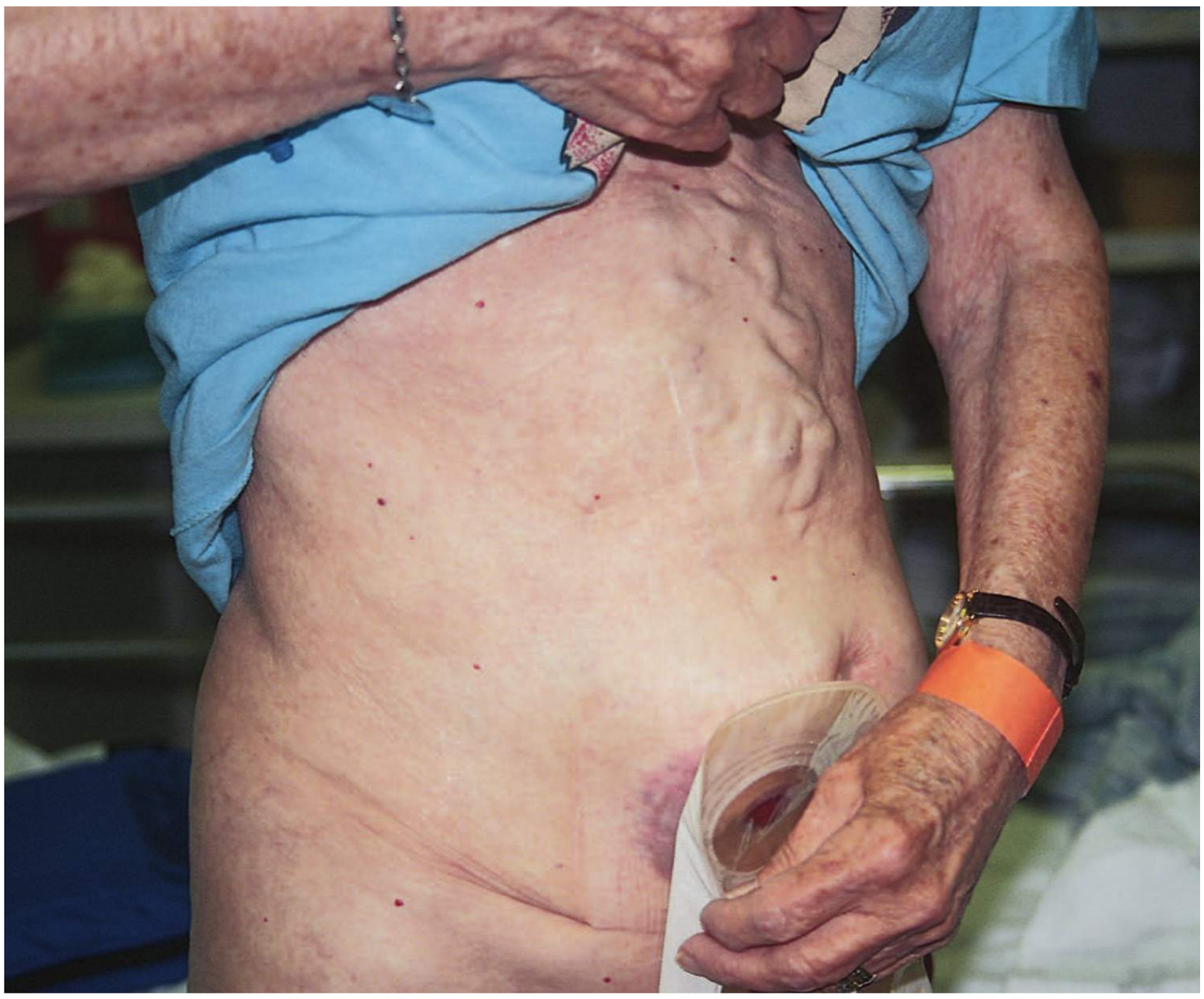


FIGURE 7.43 ■ **Caput Medusae.** This elderly female with alcoholic cirrhosis has engorged abdominal veins in the knotted appearance consistent with caput medusae. (Photo contributor: Gary Schwartz, MD.)

Clinical Summary

A hernia is a tissue protrusion through an abnormal body cavity opening. Most abdominal wall hernias occur at the groin and umbilicus. Incarceration is defined as the inability to reduce the protruding tissue to its normal position. Strangulation occurs when the blood supply of the hernia's contents is obstructed and tissue necrosis ensues. An incisional hernia may manifest clinically as a mass or palpable defect adjacent to a surgical incision and can be reproduced by having the patient perform the Valsalva maneuver. Obesity and wound infection, which interfere with wound healing, predispose to the formation of incisional hernias. The defect of an indirect inguinal hernia is the internal (abdominal) inguinal ring and may be manifest in either sex by a bulge over the midpoint of the inguinal ligament that increases in size with Valsalva maneuver. A fingertip placed into the external ring through the inguinal canal may palpate the defect. A direct hernia may be manifested by a bulge midway adjacent to the pubic tubercle and may be felt by the pad of the finger placed in the inguinal canal. The defect is in the posterior wall of the inguinal canal. Direct inguinal hernias are usually painless and occur in males. Femoral canal hernias are more common in women and are prone to both strangulation and incarceration.

Nausea and vomiting may be present if incarceration with bowel obstruction occurs. Strangulation can lead to fever, peritonitis, and sepsis.

Management and Disposition

When patients present without clinical evidence of strangulation (fever, leukocytosis, elevated lactate, systemic signs of toxicity), reduction should be attempted. In the presence of these signs, prompt surgical consultation is warranted for surgical reduction. Reduction in the ED is facilitated with systemic analgesia (as most patients present with significant pain), placing the patient in the supine position, and applying a cold pack to the hernia. Routine consultation for operative repair is indicated in asymptomatic patients with reducible hernias.

Pearls

1. Acutely strangulated or incarcerated hernias require immediate surgical evaluation.
2. Direct inguinal hernias are usually painless.
3. Evaluation and treatment of concomitant exacerbating conditions (cough, constipation, vomiting) prevent recurrences.

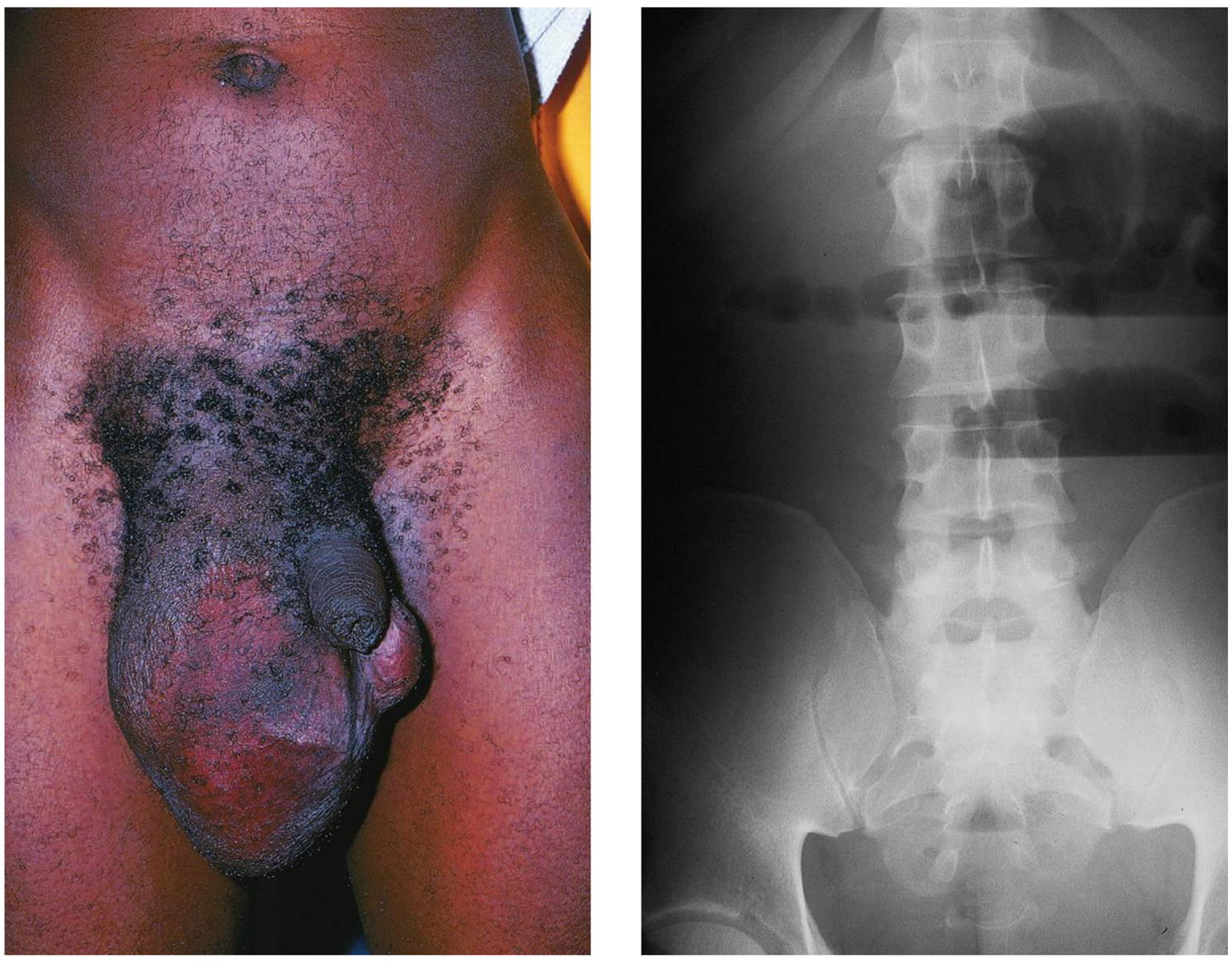


FIGURE 7.44 ■ **Indirect Inguinal Hernia.** This 35-year-old man has an incarcerated indirect inguinal hernia with small bowel obstruction seen on the upright abdominal film. (Photo contributor: Lawrence B. Stack, MD.)

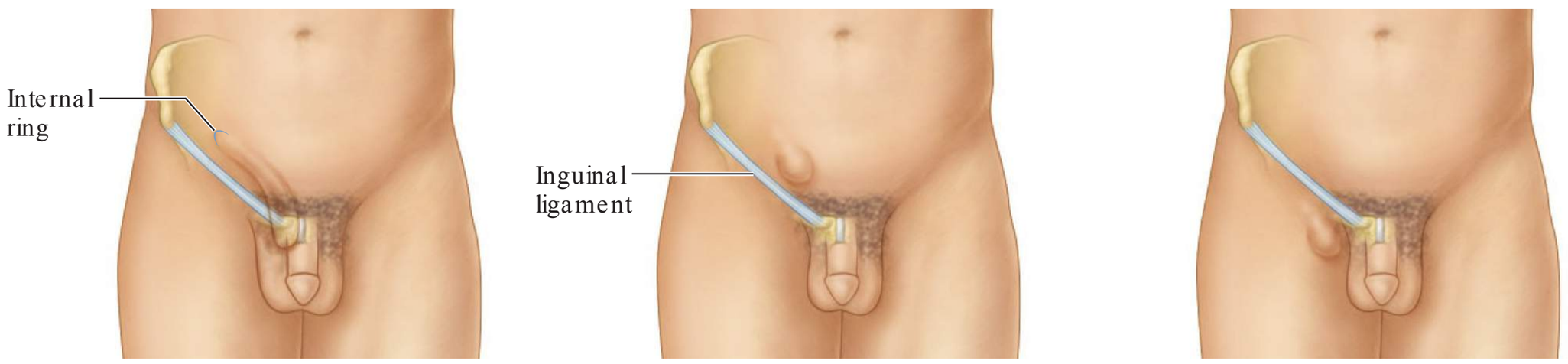


FIGURE 7.45 ■ **Hernia Types.** Drawings of the different hernia types using the landmarks of the inguinal ligament and pubic tubercle.



FIGURE 7.46A ■ **Incisional Hernia.** An asymptomatic incisional hernia in an obese male which developed after coronary artery bypass graft. The CT in Fig. 7.46B demonstrates a loop of bowel protruding through the abdominal wall defect. (Photo contributor: Lawrence B. Stack, MD.)

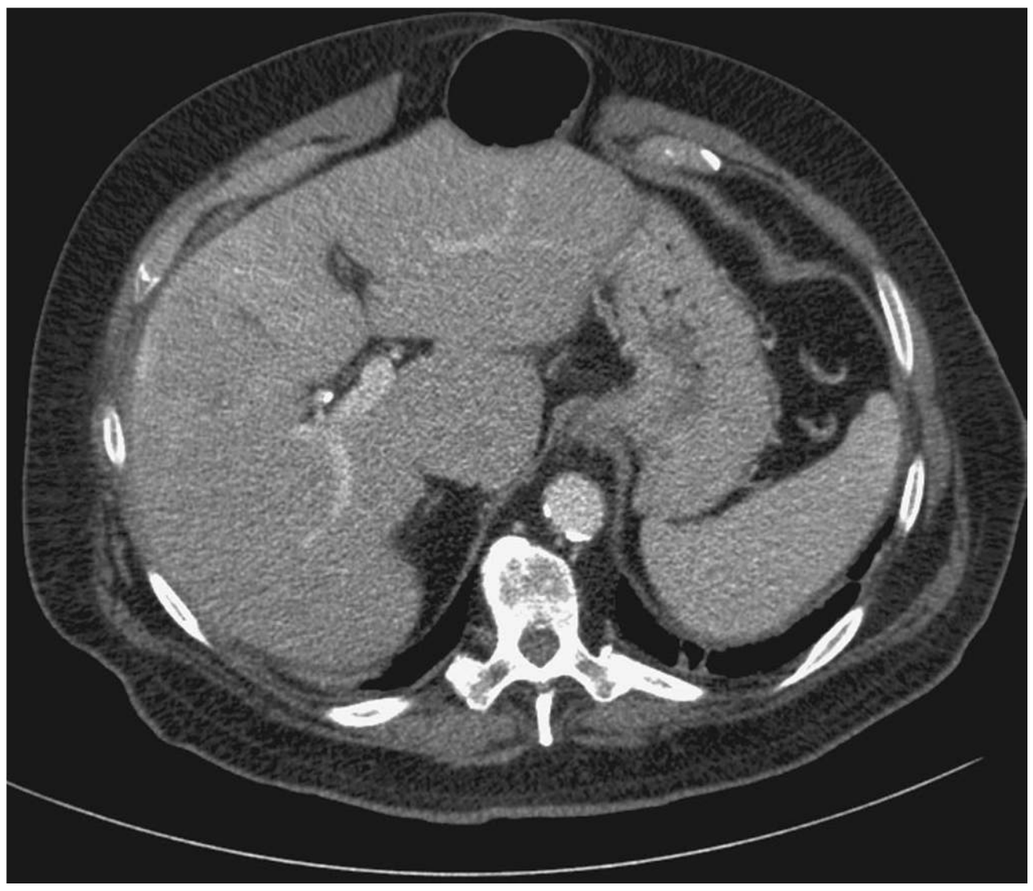


FIGURE 7.46B ■ **Incisional Hernia CT Scan.**



FIGURE 7.47 ■ **Indirect Inguinal Hernia.** A left indirect inguinal hernia in a female patient. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 7.48 ■ **Direct Inguinal Hernia.** A direct inguinal hernia. Note the bulge adjacent to the left pubic tubercle. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

The umbilicus is a common site of abdominal hernias. Predisposing conditions in adults most commonly include ascites and prior abdominal surgery. The size of the defect determines the symptomatology and incidence of incarceration, with smaller defects resulting in more pronounced symptoms and an increased incidence of incarceration. Pain is located in the area of the fascial defect. Contents of the hernia may be palpable and tender. Symptoms of obstruction (nausea, vomiting, and abdominal distention) may be present. If the hernia becomes strangulated, erythema of the overlying skin with fever and hypotension may occur.



FIGURE 7.49 ■ **Strangulated Umbilical Hernia.** The skin overlying a strangulated umbilical hernia is erythematous and tender. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Reduction is attempted in the stable patient without clinical evidence of strangulation. Treatment of any predisposing conditions (ie, abdominal paracentesis in the patient with tense ascites) may cause spontaneous reduction and avoid progression of the hernia to strangulation. Routine consultation for elective repair is indicated in asymptomatic patients with reducible hernias.

Pearls

1. Umbilical hernias in children usually resolve without treatment.
2. Umbilical hernias in adults usually become worse and require elective repair.



FIGURE 7.50A ■ **Umbilical Hernia.** A 53-year-old man with umbilical pain and swelling. (Photo contributor: Lawrence B. Stack, MD.)

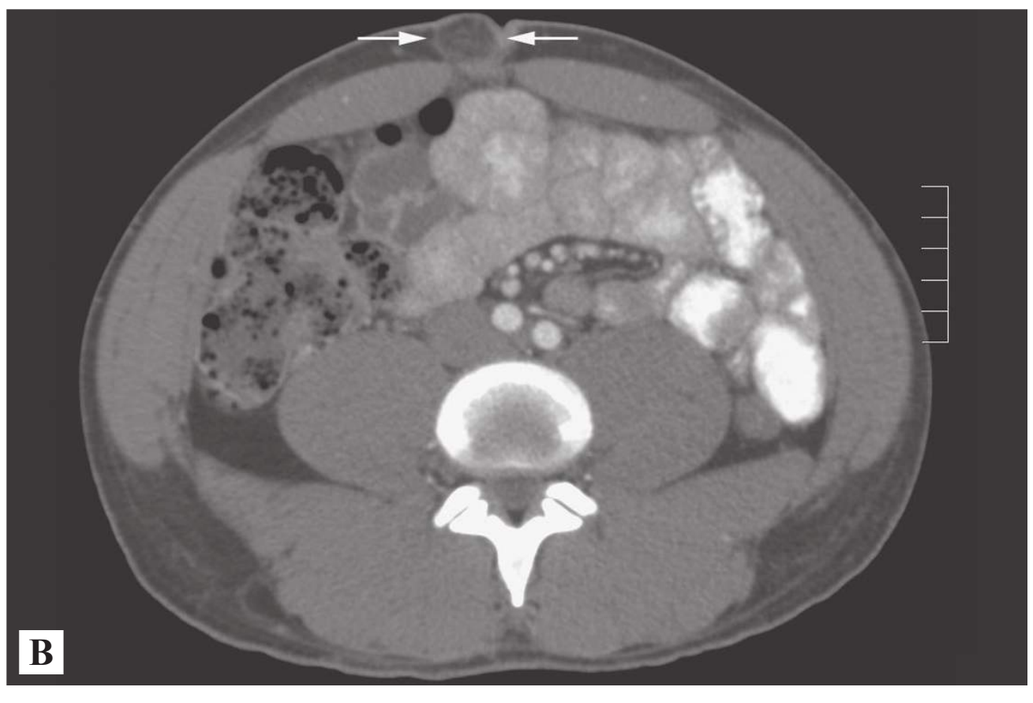


FIGURE 7.50B ■ **Umbilical Hernia CT Scan.** CT reveals omentum in the umbilical hernia defect (arrows). (Photo Contributor: Lawrence B. Stack, MD.)

PATENT URACHAL DUCT

Clinical Summary

When the vestigial urachal duct is not obliterated during development, drainage can occur from the bladder to the umbilicus. Urachal cysts may also be present, detectable as a painful mass between the umbilicus and pubis when they become infected or by ultrasound. Rarely, adenocarcinoma may form in these remnants.

Management and Disposition

Acute treatment is usually not required unless an infection is evident. Routine urologic consultation for surgical revision is indicated. A retrograde study with radiopaque dye will outline the patent duct.

Pearl

1. This finding should prompt a careful search for other urogenital anomalies.

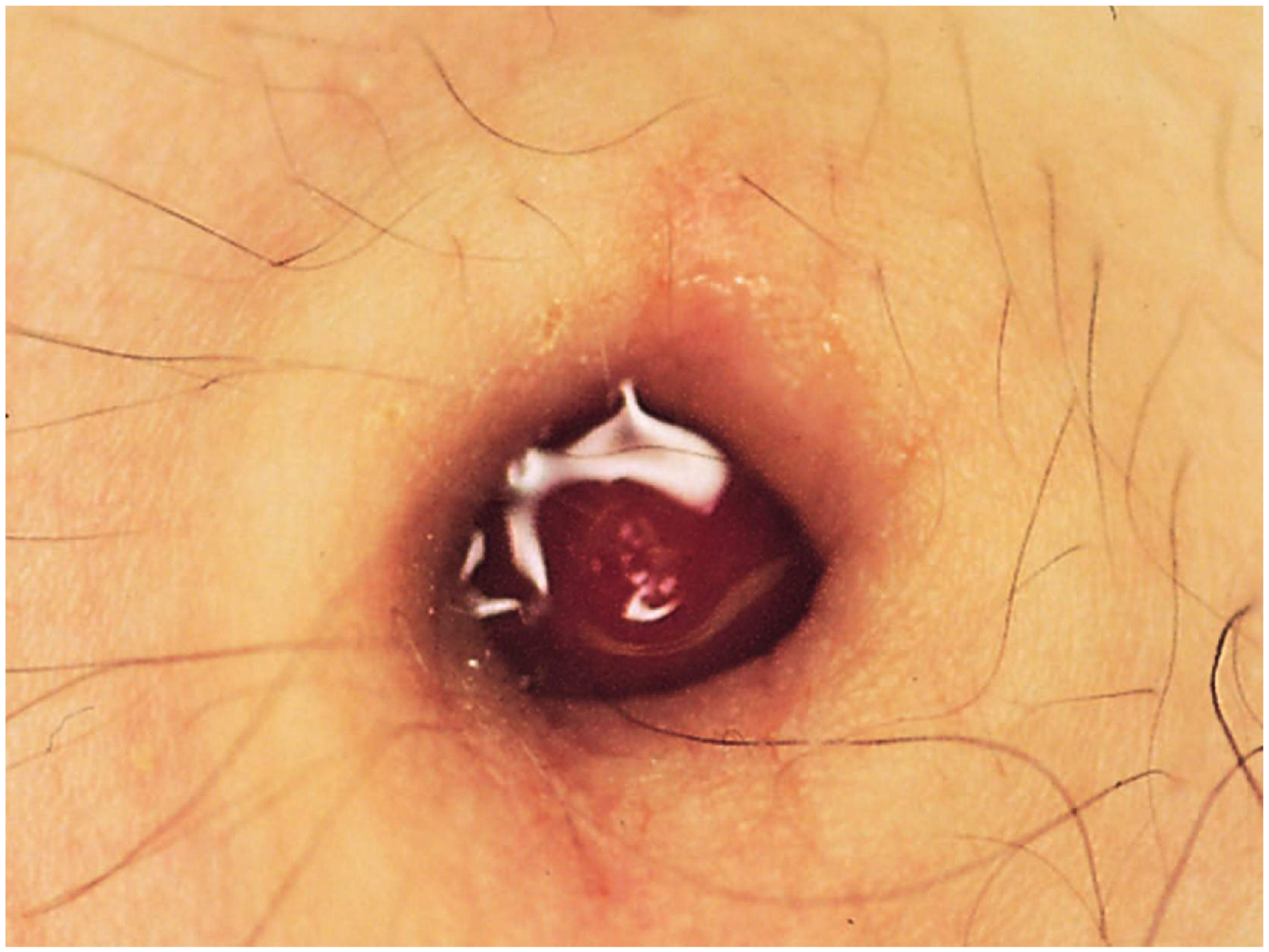


FIGURE 7.51 ■ **Patent Urachal Duct.** This 19-year-old man presented to the emergency department with clear fluid (urine) draining from the umbilicus, suggestive of a patent urachal duct. (Photo contributor: Kevin J. Knoop, MD, MS.)

SISTER MARY JOSEPH NODE (NODULAR UMBILICUS)

Clinical Summary

A Sister Mary Joseph node is a metastasis manifesting as a periumbilical nodule secondary to abdominal or pelvic cancers (one half are gastrointestinal, one quarter are gynecologic). Cancers of the colon may cause pain, change in bowel habits, anemia, and obstruction. In general, left-sided cancers cause obstruction, whereas right-sided tumors may have significant metastases before they create signs and symptoms. These metastases typically involve peritoneal and omental spread with distant metastases to the liver. Spread to the umbilicus is colloquially known as the Sister Mary Joseph node.

Prognosis is poor if cancer is the cause (survival time of 10 months).

Management and Disposition

Prompt referral for staging and treatment of the tumor is indicated. Other signs and symptoms (from obstruction, blood loss, malnutrition, and pain) should be addressed and treated.



FIGURE 7.52 ■ **Sister Mary Joseph Node.** Sister Mary Joseph nodule of patient with ovarian cancer. (Photo contributor: Stephen W. Corbett, MD.)

Pearls

1. Virchow node, presenting as a supraclavicular mass, also heralds bowel carcinoma.
2. A Sister Mary Joseph node is commonly due to gastric carcinoma.
3. All umbilical masses require evaluation.



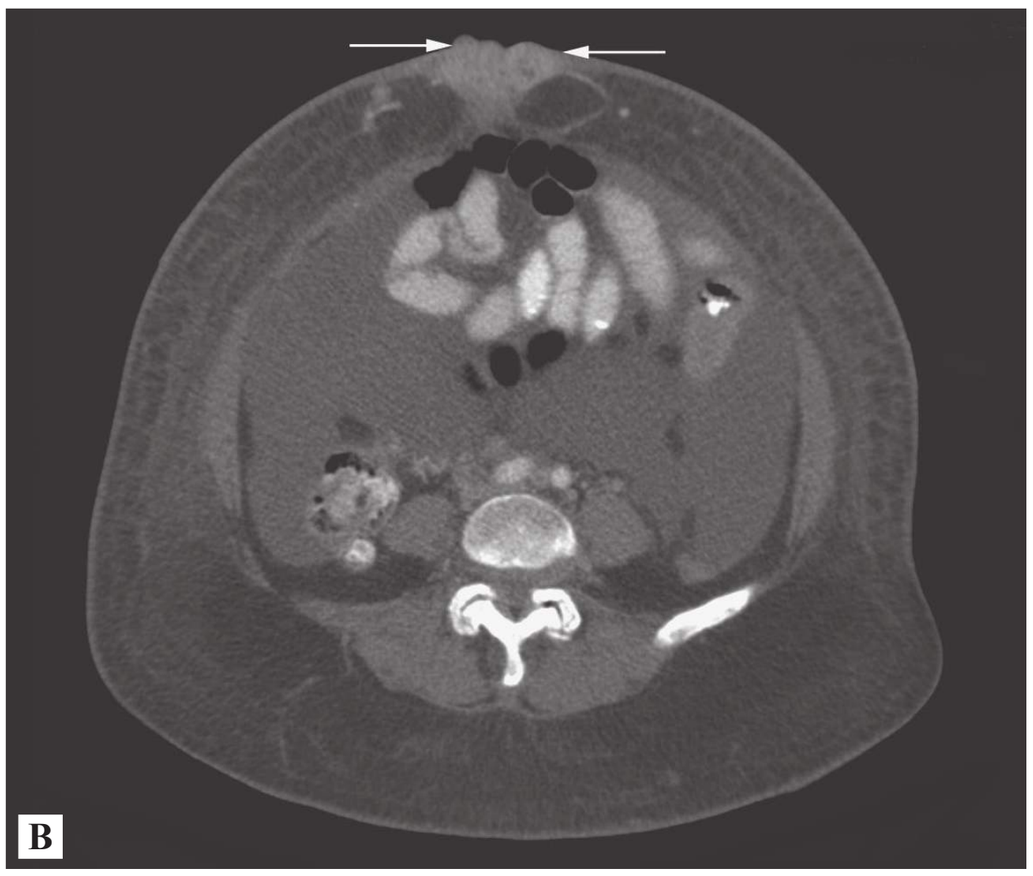


FIGURE 7.53 ■ **Sister Mary Joseph Node.** This 63-year-old woman presents with abdominal swelling and ascites. She was diagnosed with ovarian cancer. Axial CT scan of the abdomen at the level of the umbilicus demonstrates ascitic fluid and the umbilical nodularity (arrows). (Photo contributor: R. Jason Thurman, MD.)

ABDOMINAL DISTENSION

Clinical Summary

Abdominal distention may be a symptom—described by the patient as the feeling of being bloated—or a sign, a protuberance of the patient's abdomen. Obesity, ascites, pregnancy, neoplasms, aneurysm, tympanites (excess gas), organomegaly, and constipation are important etiologies to consider in the differential.

In obesity, the abdomen is uniformly rounded, while an increase in girth and fat concurrently accumulates in other parts of the body.

In patients with ascites, there may be shifting dullness, a fluid wave, bulging flanks, or hepatomegaly. The profile of the fluid-filled abdomen of ascites is a single curve from the xiphoid process to the pubic symphysis. The umbilicus may be everted, and there may be prominent superficial abdominal veins. Other physical findings suggestive of ascites include shifting dullness and a fluid wave.

In patients with neoplasms, there may be a palpable mass.

In gravid patients, fetal heart tones may be present and fetal motion may be felt. The pregnant abdomen profile shows the outward curve to be more prominent in the lower half of the abdomen. The umbilicus may be everted in the last trimester of pregnancy. Prominent abdominal wall veins may also be seen.

In patients with excess gas from bowel obstruction, there may be absent or high-pitched bowel sounds and absence of bowel movements or flatus. Excess abdominal air can be located in the lumen of the stomach or intestines or free in the peritoneum. This abdominal profile is a single curve from the xiphoid process to the pubic symphysis. Nausea, vomiting, decreased bowel sounds, and colicky pain are present in



FIGURE 7.54 ■ **Ascites.** Ascites in a male with alcoholic cirrhosis. Note the everted umbilicus and prominent superficial abdominal veins. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 7.55A ■ **Ascites with Paracentesis.** Midline approach to a paracentesis in the patient with ascites. (Photo contributor: Lawrence B. Stack, MD.)

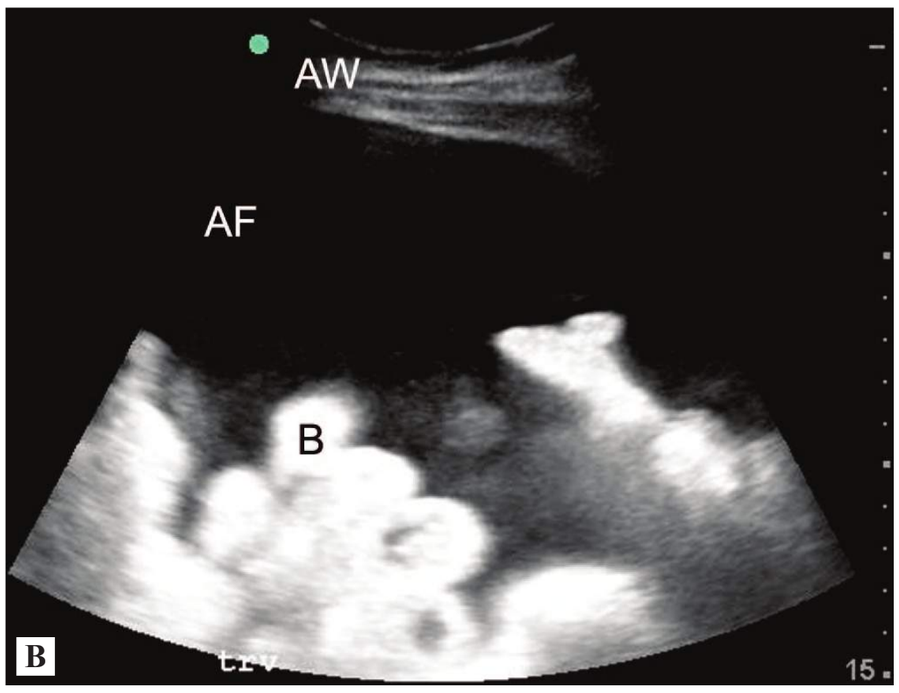


FIGURE 7.55B ■ **Ascites on Ultrasound.** Ascitic fluid (AF) seen between the abdominal wall (AW) and bowel (B) on this bedside ultrasound. (Photo contributor: Jeremy Simpson Boyd, MD.)

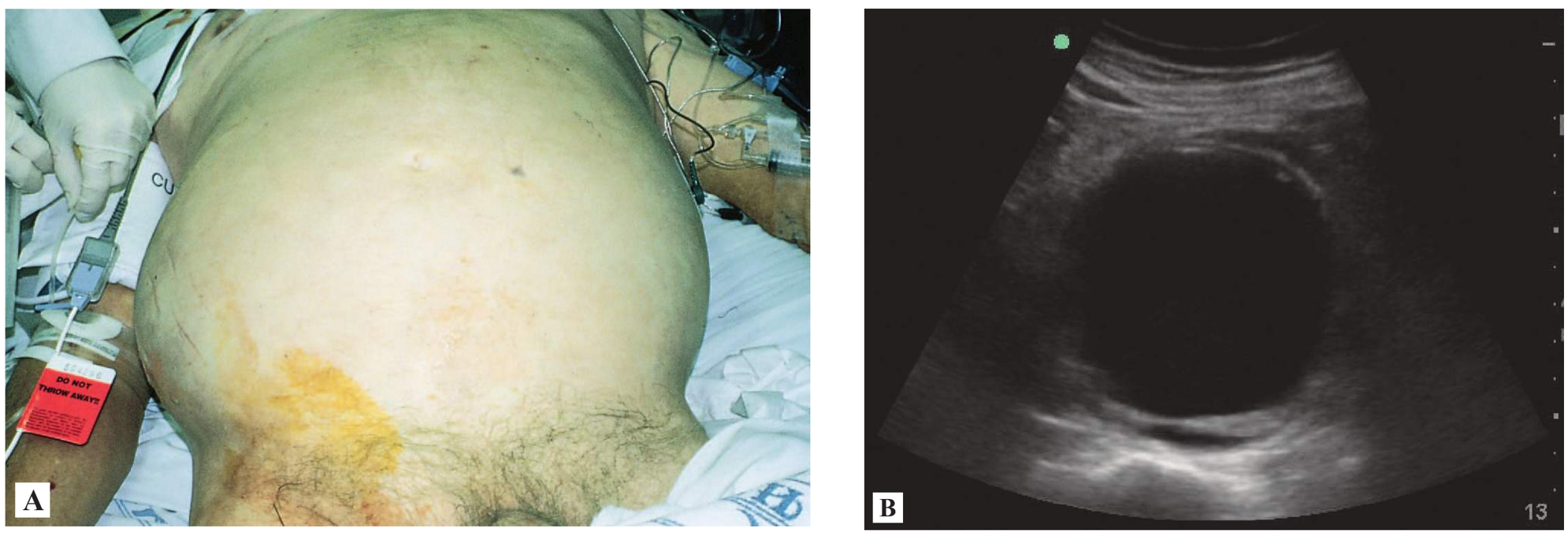


FIGURE 7.56 ■ **Abdominal Aortic Aneurysm.** (**A**) The abdomen of a patient with a leaking abdominal aortic aneurysm. Note the mottled abdominal wall and the prominent curvature of the right side of the abdomen. (Photo contributor: Stephen W. Corbett, MD.) (**B**) Eight-centimeter abdominal aortic aneurysm seen in cross-section on an ultrasound in different patient. (Photo contributor: Jeremy Simpson Boyd, MD.)



FIGURE 7.57 ■ **Gravid Abdomen.** The abdomen of a woman at 39 weeks' gestation. Note the abdominal wall striae, everted umbilicus, and prominent superficial abdominal wall veins. (Photo contributor: Stephen W. Corbett, MD.)

a small bowel obstruction. Large bowel obstruction may be accompanied by feculent vomiting and absent production of flatus.

The abdominal profile of a patient with a leaking abdominal aortic aneurysm shows a mottled abdominal wall reflective of hypoperfusion of this structure. There may be a curve of the midabdomen to either side of the aorta, more often on the left. Palpation of a pulsatile mass supports the diagnosis. Ultrasound or CT of the abdomen will confirm the diagnosis.

Management and Disposition

Treatment varies widely depending on the cause; thus, emergent management is directed at determining the etiology. Life-threatening causes (aneurysm, obstruction, neoplasms) require stabilization and referral for definitive treatment.

Pearl

1. The "six f's" can categorize conditions causing abdominal distention: fat, flatus, fetus, fluid, feces, and fatal growth.



FIGURE 7.58 ■ **Pseudoobstruction.** An 85-year-old woman was brought from a nursing home with a complaint of abdominal distention and pain for 1 to 2 days. An eventual diagnosis of Ogilvie syndrome, or pseudoobstruction of the large bowel, was made. This is usually seen in debilitated patients and can be treated with decompression. (Photo contributor: Stephen W. Corbett, MD.)



FIGURE 7.59 ■ **Constipation.** An 11-year-old boy complains of abdominal pain and abdominal distention. Stool is palpated throughout the abdomen. Flat and upright plain films of the abdomen reveal a large stool burden. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Mild trauma may produce hematomas of the rectus sheath. This injury results in intense abdominal pain, which can mimic an acute abdomen. The diagnosis is difficult by physical exam since most hematomas are in the posterior rectus sheath and are not palpable or visible. Palpation of the abdominal wall may reveal a tender mass that is accentuated by contraction of the rectus. Ultrasound and CT can confirm the diagnosis.

Major traumatic force, especially when belted in a vehicle crash, may result in a massive abdominal wall hematoma that requires intervention. Active extravasation, demonstrated by a contrast blush within the hematoma, suggests active bleeding within the hematoma.

Management and Disposition

Assuming that there is no underlying blood dyscrasia or coagulopathy, hematomas of the rectus sheath usually resolve in 1 to 2 weeks. Expanding abdominal wall hematomas with active extravasation of blood may require embolization by interventional radiology.

Pearl

1. Fothergill sign is enhancement of a rectus sheath hematoma when the abdominal wall is tensed. The mass should not cross the midline and should be easier to palpate with abdominal muscle contractions. Intra-abdominal masses are more difficult to palpate with such contractions.
2. Carnett sign is also assessed after the abdominal wall muscles are tensed; decreased pain suggests an intra-abdominal source, increased pain suggests an abdominal wall source.



FIGURE 7.60 ■ **Abdominal Wall Hematoma.** This 50-year-old man with chronic obstructive pulmonary disease developed right-lower-quadrant pain after an episode of coughing. A repeat examination on the second visit showed clearly visible ecchymosis. There was no coagulopathy and amylase was normal. A CT scan revealed a 10- by 8-cm hematoma in the right rectus abdominis sheath. (Photo contributor: Stephen W. Corbett, MD.)



FIGURE 7.61 ■ **Abdominal Wall Hematoma.** A 60-year-old woman, restrained passenger in a MVC, has an expanding abdominal wall hematoma. Contrasted CT scan of the abdomen (Figure 7.62) reveals contrast extravasation into the hematoma. (Photo contributor: Lawrence B. Stack, MD.)

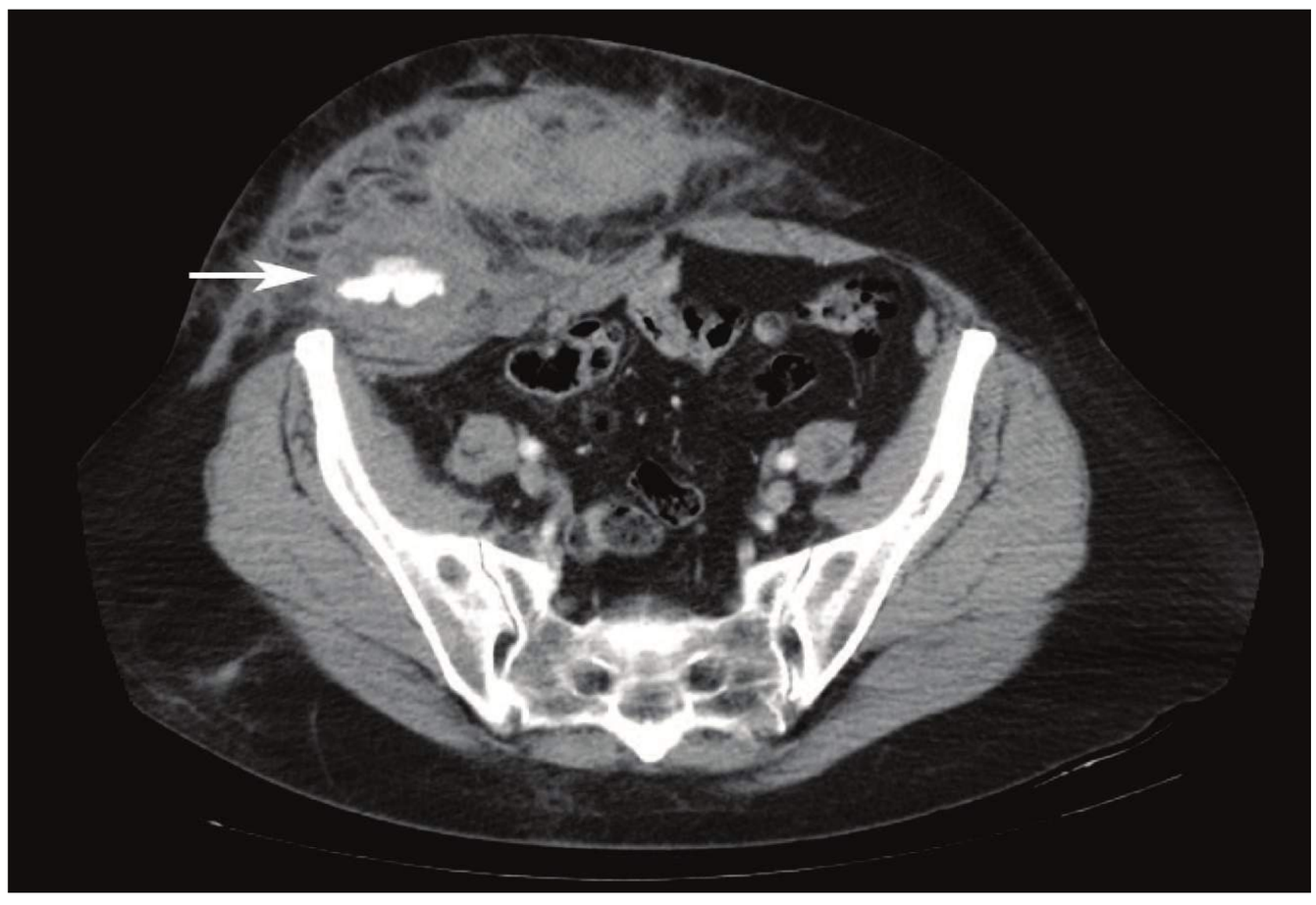


FIGURE 7.62 ■ **CT of Abdominal Wall Hematoma.** Contrast-enhanced CT of the patient in Fig. 7.61 demonstrates active extravasation of contrast (arrow) into the hematoma. Embolization of the hematoma ceased the hemorrhage. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Pectus excavatum is a common developmental deformity of the anterior chest wall present in 0.25% of births. The cause is not well understood and may be related to intrauterine growth effects or muscle and connective tissue abnormalities within the thorax. In many cases, there is a family history of the condition.

The defect is a concavity of the anterior chest wall. Although the condition is occasionally associated with Marfan, scoliosis, and congenital heart disease, most patients are asymptomatic. In some, there is slightly decreased exercise tolerance, felt to be the effect of the consequence of the increased work of breathing caused by the chest wall mechanics.

Pectus carinatum is a chest wall deformity where the sternum protrudes forward and may involve the mid and lower sternum (chondrogladiolar prominence: 95%) or upper sternum (chondromanubrial prominence: 5%). It is an uncommon deformity and becomes evident in late childhood and worsens during adolescence. Most patients are asymptomatic and repair is most often for cosmesis.

Management and Disposition

No definite treatment is needed, although cosmetic repairs of the anterior chest wall are sometimes recommended.

Pearl

1. Congential heart disease and Marfan syndrome are present in 1.5% of patients with pectus excavatum.
2. Pectus excavatum may create a radiological artifact that appears like a right middle lobe opacity.

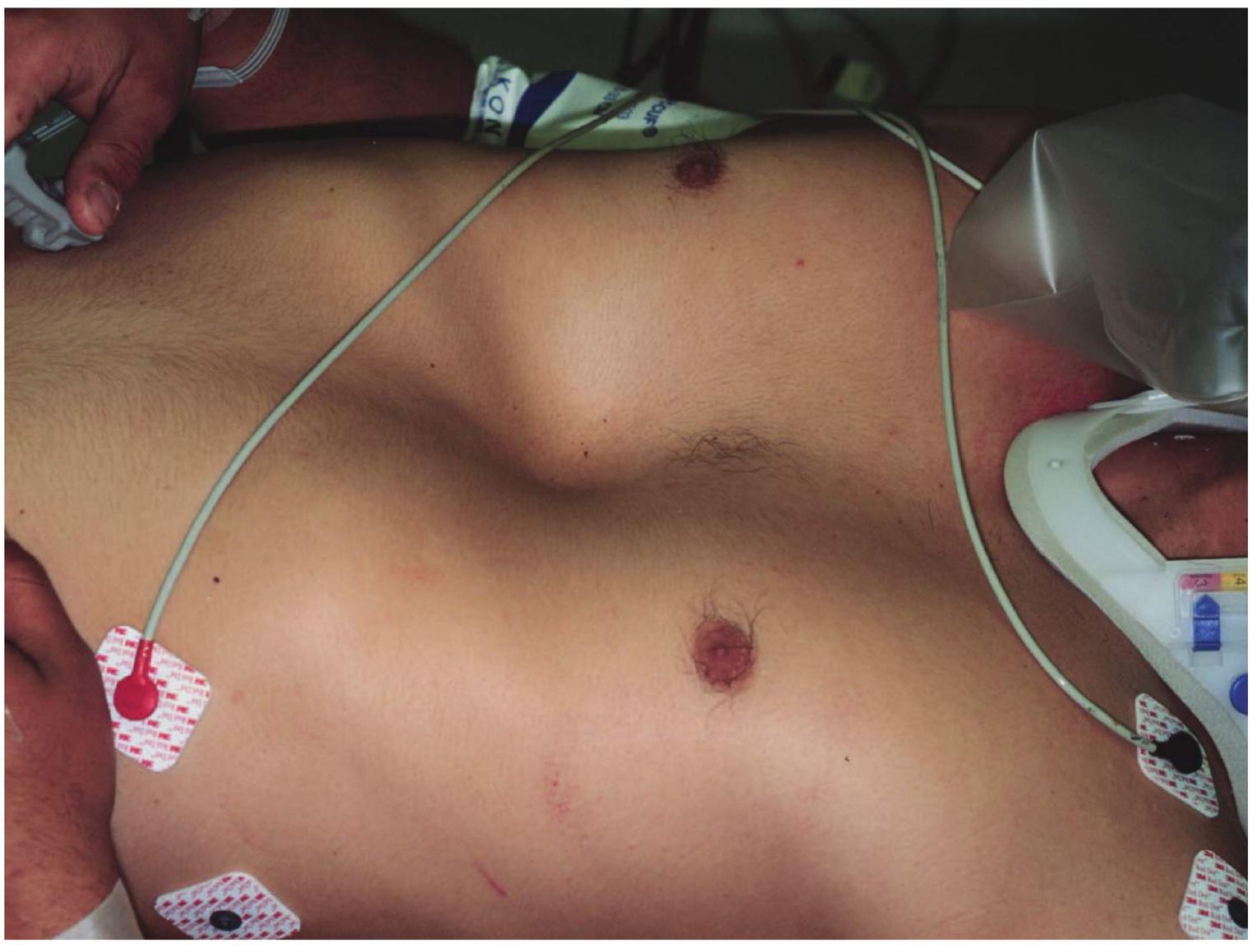


FIGURE 7.63 ■ **Pectus Excavatum.** A 26-year-old man presents to the emergency department with multisystem trauma after a motor vehicle crash. A longstanding pectus deformity is seen here. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 7.64 ■ **Pectus Carinatum.** A 14-year-old boy with sore throat was found to have pectus carinatum deformity of the chondrogladiolar prominence type. He had no chest complaint. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

When a stoma prolapses, bowel telescopes out on itself, lengthening the stoma. A stoma of the ileum may prolapse in 3% to 11% of patients over a lifetime. Causes of stomal prolapse include stomal construction difficulties, abdominal wall abnormalities such as obesity, increased intra-abdominal pressure, and weak abdominal musculature. Clinical findings of stomal prolapse include increase in size and length of the stoma; edema of the mucosa; bleeding; and if ischemic may be dusky, cyanotic, or purple in color.

Management and Disposition

Emergent surgical consultation should occur in patients with a gangrenous stoma or if the prolapse is not reducible. To reduce a prolapsed stoma, have the patient lie supine or in slight Trendelenburg to decrease intra-abdominal pressure. Apply continuous gentle pressure on the prolapsed stomal tissues, into the abdominal cavity. If the bowel is edematous, a cold compress or osmotic therapy using table sugar applied for 15 minutes before reduction attempt may reduce the edema. If stomal prolapse reduction is successful, general surgery follow-up should be arranged as soon as possible.

Pearls

1. Oral or intravenous diazepam may help facilitate prolapse reduction.
2. Your hospital stoma nurse may be a great resource to assist with management of stoma complications.



FIGURE 7.65 ■ **Prolapsed Stoma.** A 32-year-old man with inflammatory bowel disease and ileostomy presents with a prolapsed stoma. Note the bleeding, edema, and dusky appearance of the ileal mucosa. (Photo contributor: Lawrence B. Stack, MD.)

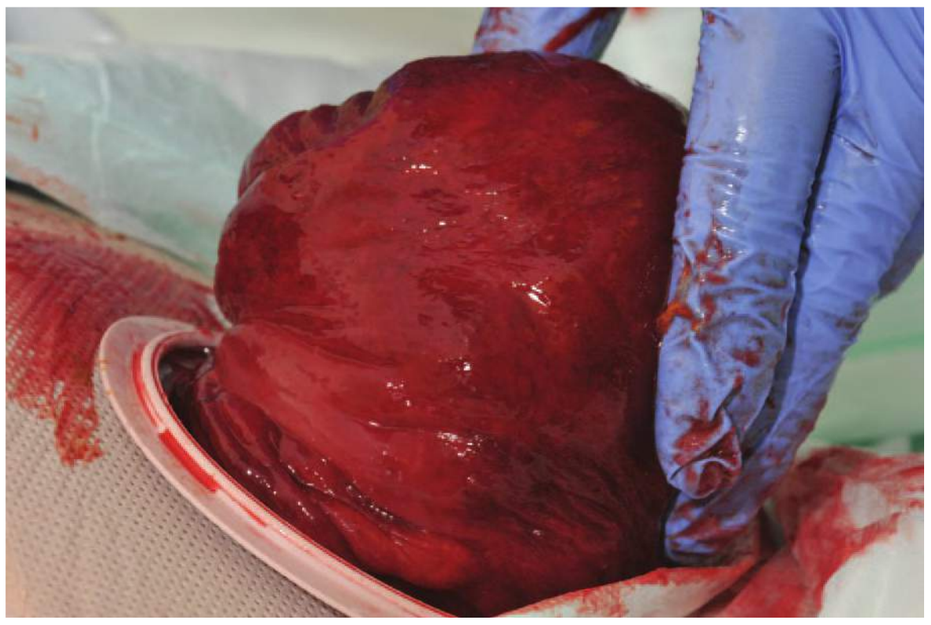


FIGURE 7.66 ■ **Prolapsed Stoma.** A 48-year-old man with pain and bleeding from stoma which was placed after a partial colectomy for complex diverticulitis. The bowel appears dusky and is oozing blood. (Photo contributor: Nakul Shekhawat, MD, MPH.)

PRUNE BELLY (EAGLE-BARRETT) SYNDROME

Clinical Summary

Prune belly syndrome (PBS) is a congenital disorder characterized by a partial or complete lack of abdominal musculature, severe urinary tract abnormalities, and bilateral cryptorchidism. The syndrome is typically recognized at birth or by ultrasound in utero. Severe renal dysplasia resulting in end-stage renal disease (ESRD) occurs in 50% of patients. ESRD and its complications are the most common reason a patient with PBS comes to the ED. Patients with PBS are at higher risk for recurrent urinary tract infection (UTI), chronic constipation, splenic torsion, and midgut and cecal volvulus.

Management and Disposition

Patients with PBS will most commonly frequent the ED because of complications of ESRD and their hemodialysis, continuous ambulatory peritoneal dialysis, or transplant. Because patients with PBS are at higher risk for splenic torsion and midgut or cecal volvulus, CT scan should be performed if sudden onset of severe abdominal pain occurs in these patients. Frequent UTIs and chronic constipation may prompt ED visits.

Pearls

1. About one-half of patients progress to ESRD.
2. Chronic constipation is a common symptom in patients with PBS.
3. Gastrointestinal abnormalities are seen in 30% of patients with PBS.

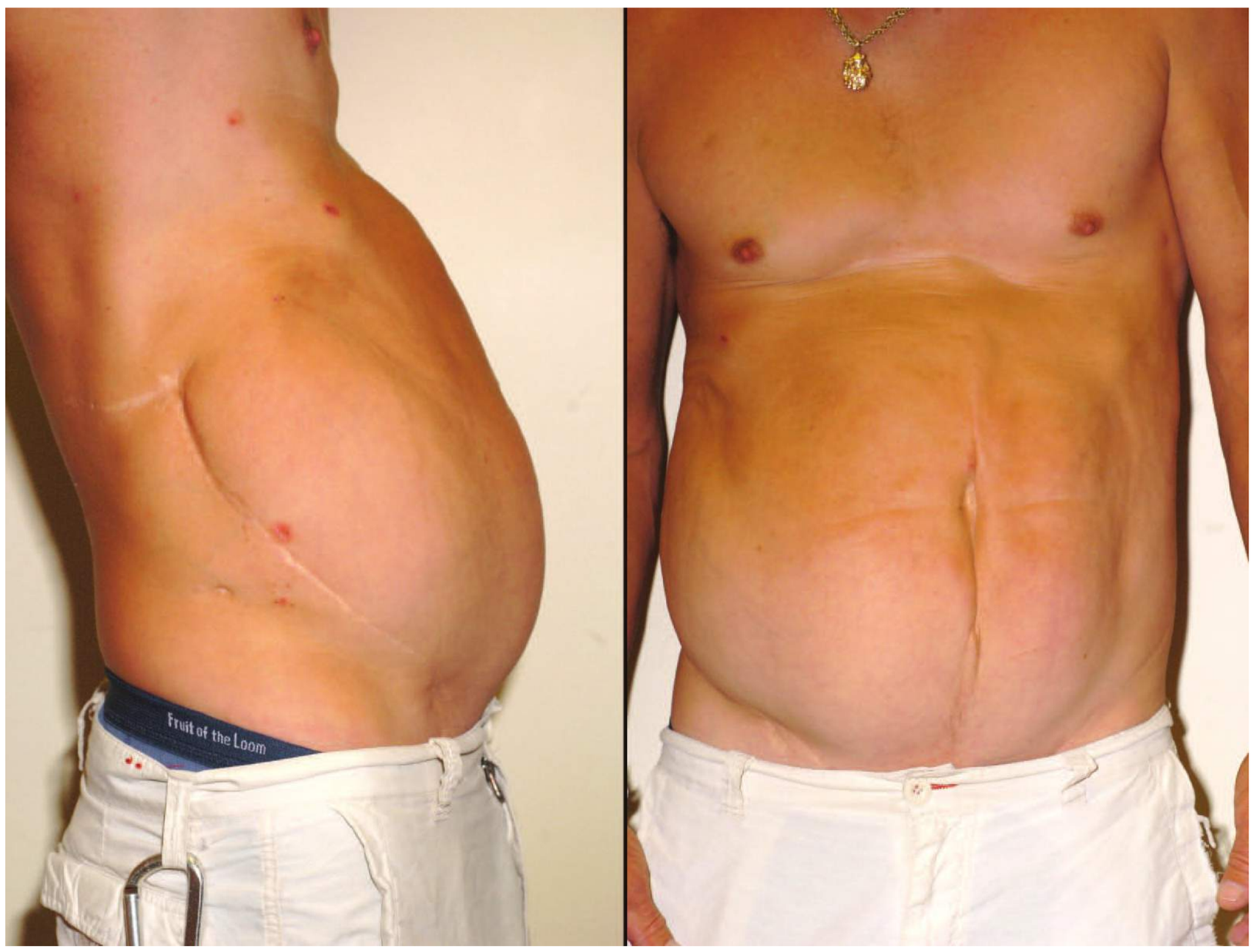


FIGURE 7.67 ■ **Prune Belly Syndrome.** A 44-year-old man with PBS, s/p renal transplant, comes to the ED for abdominal pain and was ultimately diagnosed with constipation. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 7.68 ■ **Prune Belly Syndrome.** A 2-year-old with PBS on hemodialysis comes to the ED for fever. Patient was bacteremic due to a dialysis catheter infection. (Photo contributor: Lawrence B. Stack, MD.)

Chapter 8

UROLOGIC CONDITIONS

Jeffery D. Bondesson

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Priapism. Aspiration of the corpora cavernosa followed by injection of α-adrenergic agents such as phenylephrine through the same needle is demonstrated by combining two syringes with a three-way stopcock. (Photo contributor: David Effron, MD.)

TESTICULAR TORSION

Clinical Summary

Young men (average age 16-17.5 years) complain of the sudden onset of pain in one testicle, followed by swelling of the affected testicle, reddening of the overlying scrotal skin, lower abdominal pain, nausea, and vomiting. An examination reveals a swollen, tender, retracted testicle that often lies in the horizontal plane (bell-clapper deformity). The spermatic cord is frequently swollen on the affected side. In delayed presentations, the entire hemiscrotum may be swollen, tender, and firm. The urine is usually clear with a normal urinalysis. In one-third of cases, there is a peripheral leukocytosis.



FIGURE 8.1 ■ **Testicular Torsion.** Swollen, tender hemiscrotum, with erythema of scrotal skin and retracted testicle. (Photo contributor: Stephen W. Corbett, MD.)

Management and Disposition

Obtain urologic consultation immediately and prepare to go to the operating room without delay. Doppler ultrasound or technetium scanning may be helpful if these procedures will not delay surgery. In the interim, detorsion may be attempted if the patient is seen within a few hours of onset: open the affected testicle like a book, that is, the right testicle turned counterclockwise when viewed from below and the left testicle turned clockwise. Pain relief is immediate. Decreased pain prompts additional turns (as many as three) to complete detorsion; increased pain prompts detorsion in the opposite direction. Do not delay operative intervention for ancillary studies since testicular infarction will occur within 6 to 12 hours after torsion.

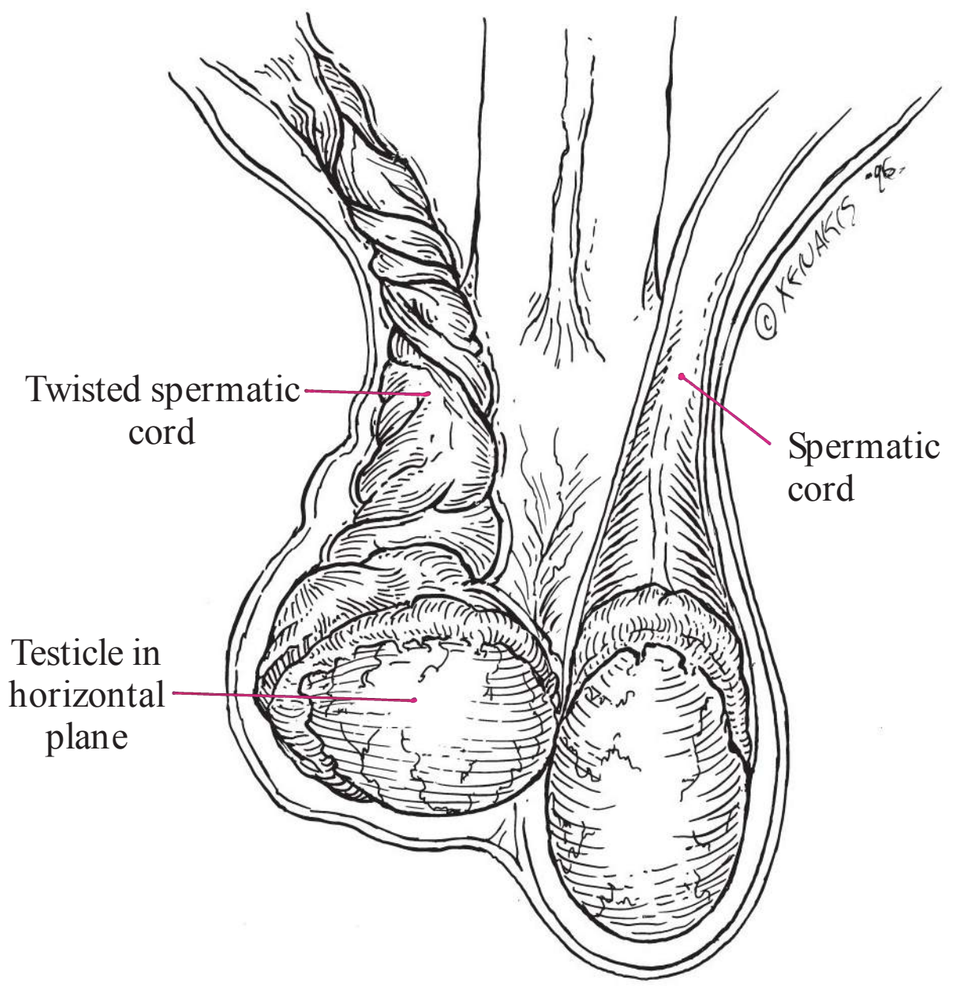


FIGURE 8.2 ■ **Bell-Clapper Deformity.** Twisting of the spermatic cord causes the testicle to be elevated with a horizontal lie. Lack of fixation to the posterior scrotum predisposes the freely movable testicles to rotation and subsequent torsion. Asymptomatic patients with bell-clapper deformity are at risk for torsion.

Pearls

1. The cremasteric reflex is almost always absent in testicular torsion.
2. Patients may report similar, less severe episodes that spontaneously resolved in the recent past.
3. Half of all torsions occur during sleep.
4. Abdominal or inguinal pain is sometimes present without pain to the scrotum.
5. The age of presentation has a bimodal pattern, since torsion is more prevalent during infancy and adolescence.





FIGURE 8.3 ■ **Testicular Torsion.** A retracted testicle consistent with early testicular torsion (minimal edema) is seen in both of these patients. (**A**) (Photo contributor: David W. Munter, MD, MBA.) (**B**) (Photo contributor: Emergency Medicine Department, Naval Medical Center Portsmouth.)



FIGURE 8.4 ■ **Testicular Torsion.** Swollen, tender scrotal mass. (Photo contributor: Patrick McKenna, MD.)



FIGURE 8.5 ■ **Testicular Torsion**. A red, swollen hemiscrotum is seen in this right testicular torsion in a 14-year-old. (Photo contributor: Lawrence Heiskell, MD.)

TORSION OF A TESTICULAR OR EPIDIDYMAL APPENDIX

Clinical Summary

Vestigial remnants in the embryology of the scrotum are often found on the superior portions of the testicle or the epididymis. These appendages, which have no known function, are occasionally on a stalk that is subject to torsion. This most commonly occurs in boys up to 16 years of age but has been reported in adults. The patient complains of sudden pain around the superior pole of the testicle or epididymis as the appendix undergoes necrosis and inflammation. Early in the course, palpation of a firm, tender nodule in this area will confirm the diagnosis.

Management and Disposition

Obtain urologic consultation immediately. Differentiating from the more emergent testicular torsion is the key responsibility. Ancillary studies are generally not helpful in making this diagnosis unless it presents very early in its course. A urinalysis is generally normal. The characteristic physical signs of a small, tender, upper-pole nodule along with a color Doppler ultrasound showing good flow to the testicle may mitigate the need for emergent surgery. With later presentations or an equivocal ultrasound, the diagnosis may not be made with confidence before surgery. If surgery is not deemed necessary by the urologic consultant, analgesics and rest are all that is required. The appendix will involute and calcify in 1 to 2 weeks.

Pearls

1. Stretching of the scrotal skin across the necrotic nodule will occasionally reveal a bluish discoloration of the nodule, called the "blue-dot sign." This is pathognomonic for torsion of the appendix.
2. A reactive hydrocele may accompany appendiceal torsion. When the hydrocele is transilluminated, the blue-dot sign may be revealed.



FIGURE 8.7 ■ **Blue-Dot Sign.** A blue-dot sign is seen in an older patient. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 8.6 ■ **Blue-Dot Sign.** A blue-dot sign is caused by torsion of the testicular appendix. It is best seen with the skin held taut over the testicular appendix. (Photo contributor: Javier A. Gonzalez del Rey, MD.)

Clinical Summary

The onset of scrotal pain typically occurs over hours and is often referred to the ipsilateral inguinal canal or lower abdominal quadrant. Recent urinary tract instrumentation or urinary tract infection is a risk factor. Early in the course, a tender, indurated, edematous epididymis is palpated separately from the nontender testicle. Late presentations will have generalized scrotal swelling and tenderness, making examination and differentiation more difficult. The urinalysis reveals pyuria or bacteriuria half of the time and the peripheral white blood cell count is frequently elevated. Patients can present with fever and signs of sepsis.

Management and Disposition

Direct outpatient treatment of younger men (<35 years) at sexually transmitted organisms. Older men tend to have infections caused by organisms in common with urinary tract infection. Refer adolescents and children for urological evaluation to rule out congenital anomalies, which are common in nongonococcal infections in this age group. Consider admission and IV antibiotics for febrile patients.

Pearls

1. Elevation of the affected hemiscrotum while supine may provide relief of symptoms (Prehn sign).
2. Evaluate older men for urinary retention, as this is a frequent cause of epididymitis.
3. Testicular tumors are most frequently misdiagnosed as epididymitis.
4. The absence of pyuria or bacteriuria does not exclude the diagnosis.
5. Referred pain to the lower quadrants can mimic appendicitis or diverticulitis.

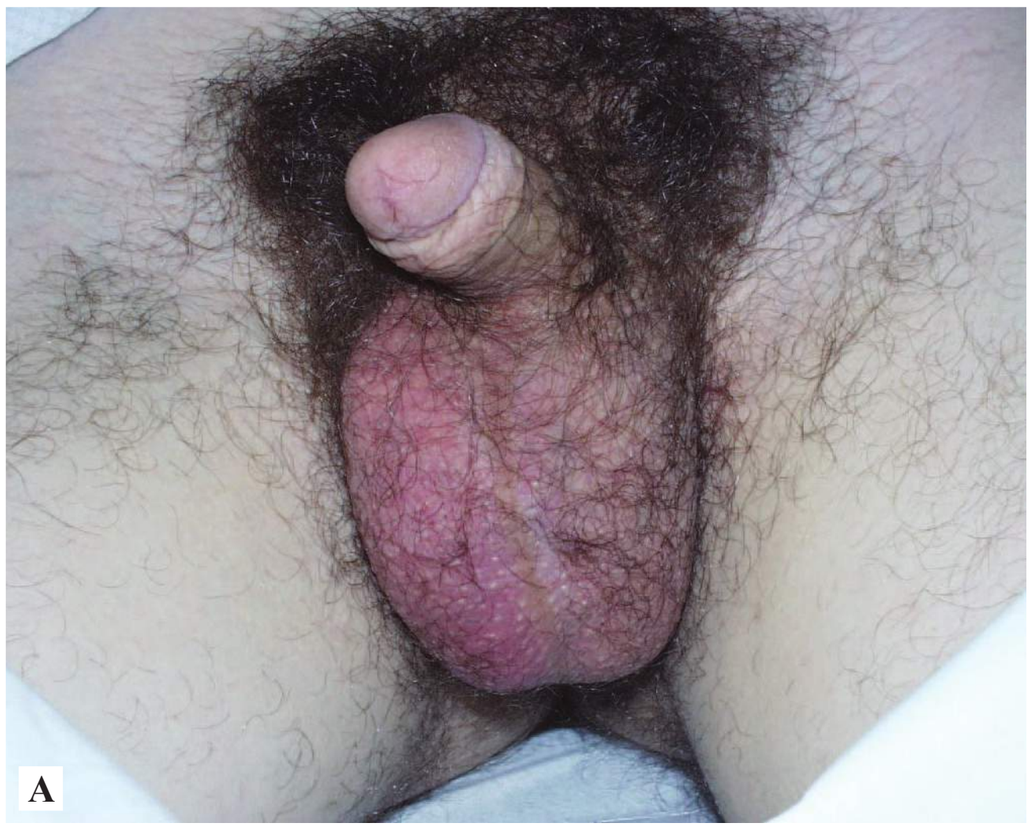


FIGURE 8.8 ■ **Acute Epididymitis.** (**A**) Swelling of the right hemiscrotum and tenderness of the inferior posterior portion of the testicle. (Photo contributor: Emergency Medicine Department, Naval Medical Center Portsmouth.) (**B**) Diffuse and erythema swelling of the entire scrotum. (Photo contributor: Cyril Thomas, PAC.)

ORCHITIS

Clinical Summary

Orchitis has a variable onset and is mildly uncomfortable or severely painful. It is most frequently a complication of mumps infection and more rarely other viruses. Bacterial orchitis is rarer still and associated with concurrent epididymitis. Mumps orchitis occurs 4 to 7 days after parotid symptoms with testicular pain and swelling. It is unilateral 70% of the time with a contralateral infection developing later 30% of the time. The testicle is swollen and tender, sparing the epididymis. The overlying scrotal skin can be edematous and erythematous. Constitutional symptoms of malaise, headache, myalgias, and fever are common.

Management and Disposition

Supportive care with analgesics, hot or cold packs, and scrotal elevation is sufficient for mumps orchitis. Bacterial orchitis is treated the same as epididymitis.



FIGURE 8.9 ■ **Orchitis.** Unilateral testicular pain, swelling, scrotal erythema, and edema are seen in this patient with parotitis. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. Pain and swelling from mumps orchitis mimics testicular torsion mandating Doppler ultrasonography to differentiate.
2. An enlarged, tender epididymis or boggy, tender prostate supports bacterial orchitis.
3. Preceding or concurrent parotid swelling supports mumps orchitis.



FIGURE 8.10 ■ **Orchitis.** Swelling and erythema of the right hemiscrotum in a patient with orchitis. (Photo contributor: David Effron, MD.)

HYDROCELE

Clinical Summary

Most hydroceles occur in older patients and develop gradually without any significant symptoms. Hydrocele presents as a soft, pear-shaped, fluid-filled cystic mass anterior to the testicle and epididymis that will transilluminate. However, it can be tense and firm and will transilluminate poorly if the tunica vaginalis is thickened. Almost all hydroceles in children are communicating, resulting from the same mechanism that causes inguinal hernia. A persistent, narrow processus vaginalis acts like a one-way valve, thus permitting the accumulation of dependent peritoneal fluid in the scrotum. Acute symptomatic hydroceles are rarer and can occur in association with epididymitis, trauma, or tumor.

Management and Disposition

In an acute hydrocele, direct treatment at discovering a possible underlying cause. A positive urinalysis may point toward an infectious etiology. Transillumination helps demonstrate whether the mass is cystic or solid. Ultrasound can be very helpful in imaging the scrotal contents and delineating the composition of the mass. Acute hydroceles should not be considered benign and require referral to a urologist to rule out tumor or infection. Refer chronic accumulations to a urologist on a more routine basis for elective drainage.

Pearls

1. Ten percent of testicular tumors have a reactive hydrocele as the presenting complaint.
2. An inguinal hernia with a loop of bowel in it may emit bowel sounds.
3. Hydroceles are almost never symptomatic.
4. Acute reactive hydroceles may be caused by infection, trauma, or torsion.

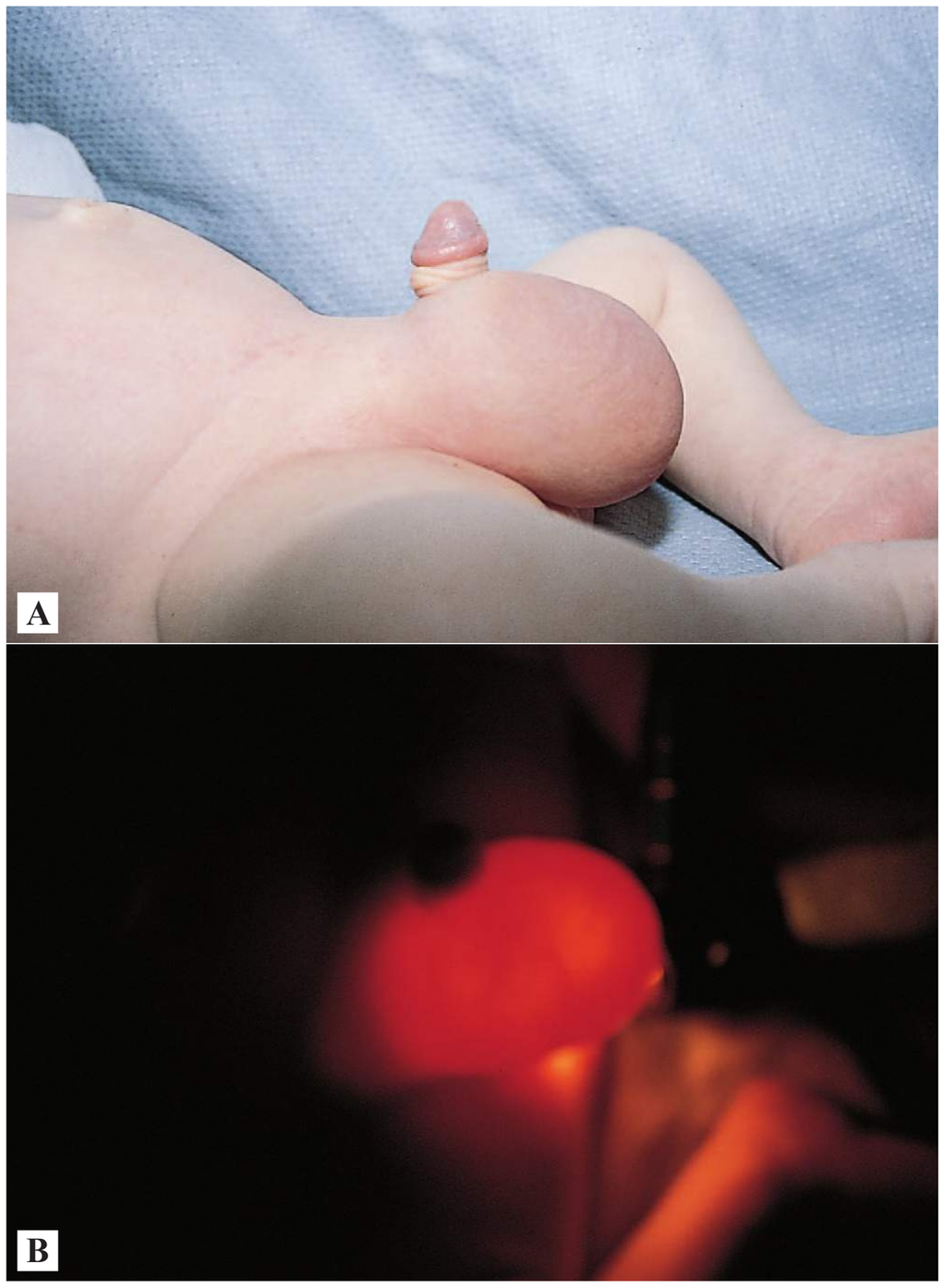


FIGURE 8.11 ■ **Hydrocele.** Painless swelling in the scrotum of a young boy (**A**). Transillumination of the swelling (**B**) identifies the hydrocele. (Photo contributor: Michael J. Nowicki, MD.)

Clinical Summary

In testicular tumor, a painless, firm testicular mass is palpated, with the patient often complaining of a "heaviness" of his testicle. If the patient presents early, the mass will be distinct from the testis, whereas later presentations will have generalized testicular or scrotal swelling. These lesions occasionally present with pain due to infarction of the tumor.

Management and Disposition

Refer patients promptly to a urologist for surgical exploration.

Pearls

1. Acute hydroceles and hematoceles should prompt the physician to consider a tumor as the cause.
2. Pain from tumor infarction is usually not as severe as pain due to torsion or epididymitis.
3. Findings of an unexplained supraclavicular lymph node, abdominal mass, or chronic nonproductive cough resistant to conventional therapy should prompt a testicular examination for tumor.

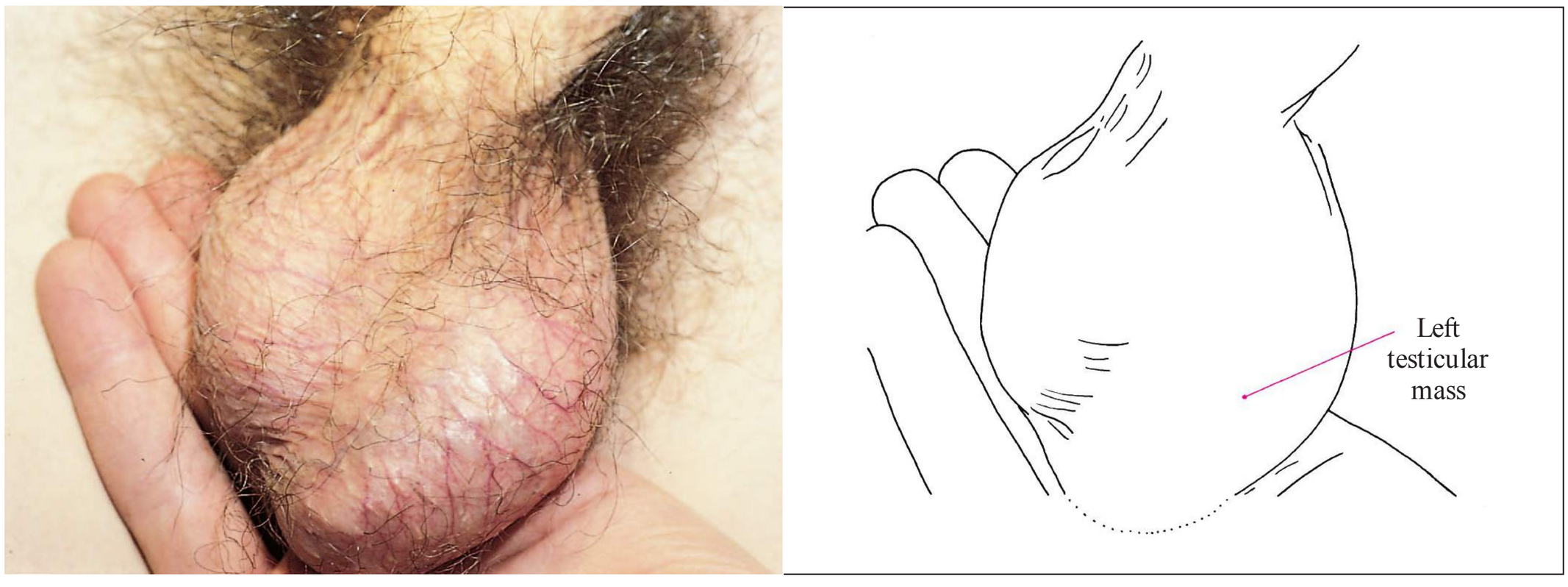


FIGURE 8.12 ■ **Testicular Tumor.** This painless left testicular mass is highly suspicious for tumor, as proved to be the case in this patient. (Photo contributor: Patrick McKenna, MD.)

SCROTAL ABSCESS

Clinical Summary

A scrotal abscess is a suppurative mass with surrounding erythema involving the superficial layers of the scrotal wall. The usual history given is progressive swelling of a small pustule or papule with increasing pain and induration or fluctuance. Constitutional symptoms and fever are generally absent.

Management and Disposition

Using local anesthesia, simply make a stab incision and drain the abscess. Instruct the patient to use a sitz bath and to change the dressing frequently. An alternative method of treatment to ensure adequate wound drainage is to unroof the abscess by circumferential excision. Immunocompromised patients may require intravenous antibiotics and admission.

Pearl

1. If the patient appears ill out of proportion to the superficial appearance, suspect that this mass is the point of a deep scrotal abscess.



FIGURE 8.13 ■ **Scrotal Abscess.** Suppurative mass on the scrotum. (Photo contributor: David Effron, MD.)

Clinical Summary

Fournier gangrene, a form of necrotizing fasciitis, most frequently occurs in a middle-aged diabetic man who presents with swelling, erythema, and severe pain of the entire scrotum, but it is also known to occur in women. In men, the scrotal contents often cannot be palpated because of the marked inflammation. The patient has constitutional symptoms with fever and frequently is in shock. There is often a history of recent urethral instrumentation, an indwelling Foley catheter, or perirectal disease. A localized area of fluctuance cannot be appreciated.

Management and Disposition

These patients require aggressive fluid resuscitation and early surgical consultation for immediate debridement and surgical drainage. Give broad-spectrum antibiotics effective against gram-positive, gram-negative, and anaerobic organisms as soon as possible in the emergency department. There is anecdotal experience that treatment is enhanced by hyperbaric oxygen.

Pearls

1. Pain out of proportion to the clinical findings may represent an early presentation of Fournier gangrene.
2. A plain pelvic radiograph may reveal subcutaneous air.
3. Fournier gangrene is usually quite painful but has been known to present with only a mildly uncomfortable necrosis of the scrotal wall and exposed testis.



FIGURE 8.14 ■ **Fournier Gangrene.** Middle-aged diabetic man with fever and scrotal pain. Final diagnosis is polymycrobial Fournier gangrene. (Photo contributor: Eugene C. Eiland, MD.)



FIGURE 8.15 ■ **Fournier Gangrene.** Swollen, tender, erythematous labia, perineum, and inner thighs in a female patient with Fournier gangrene. (Photo contributor: Daniel L. Savitt, MD.)



FIGURE 8.16 ■ **Fournier Gangrene.** Necrosis of overlying scrotal skin along with swelling, erythema, high fever, and severe pain was noted in this diabetic patient with Fournier gangrene. (Photo contributor: R. Jason Thurman, MD.)

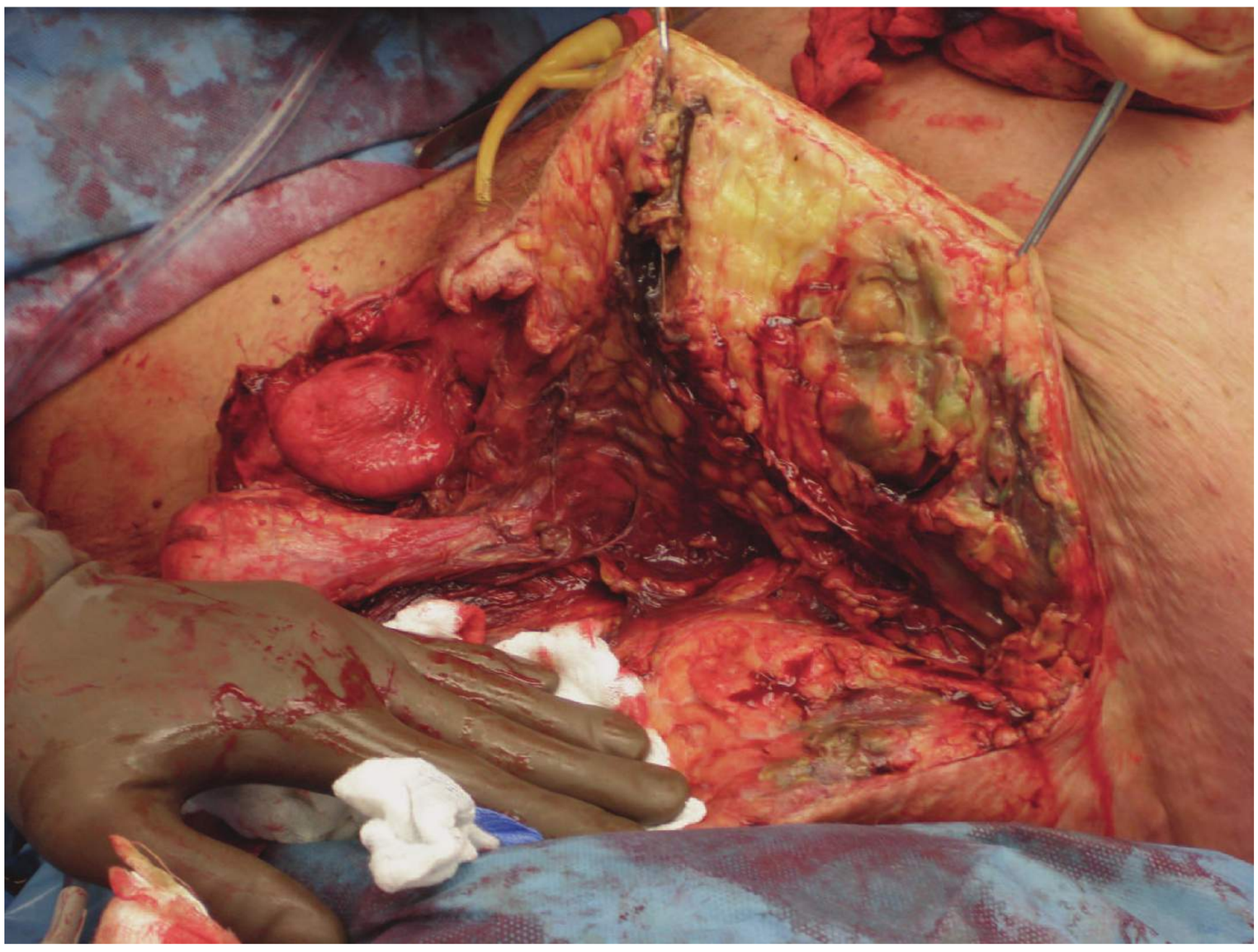


FIGURE 8.17 ■ **Fournier Gangrene.** The extent of necrosis in Fournier gangrene can be formidable as seen in this patient. (Photo contributor: R. Jason Thurman, MD.)

Clinical Summary

Paraphimosis is the entrapment of a retracted foreskin that cannot be reduced behind the coronal sulcus. Pain, swelling, and erythema are common. If severe, the constriction causes edema and venous engorgement of the glans, which can lead to arterial compromise with subsequent tissue necrosis. In contrast to paraphimosis, phimosis is the inability to retract the foreskin.

Management and Disposition

Squeeze the glans firmly for 5 minutes to reduce the swelling. This can lead to successful reduction of the foreskin. If manual reduction fails, consult a urologist for local infiltration of anesthesia with vertical incision of the constricting band.

Pearls

1. In the presence of arterial compromise, if a urologist is not immediately available, anesthetize and incise the constricting band.
2. The patient should be referred to a urologist for circumcision if successfully reduced.
3. Phimosis is "physiologic" in young boys (generally <5 to 6 years old).
4. Phimosis, if "reduced" (retracted proximally over the glans), can cause a paraphimosis—a true emergency.

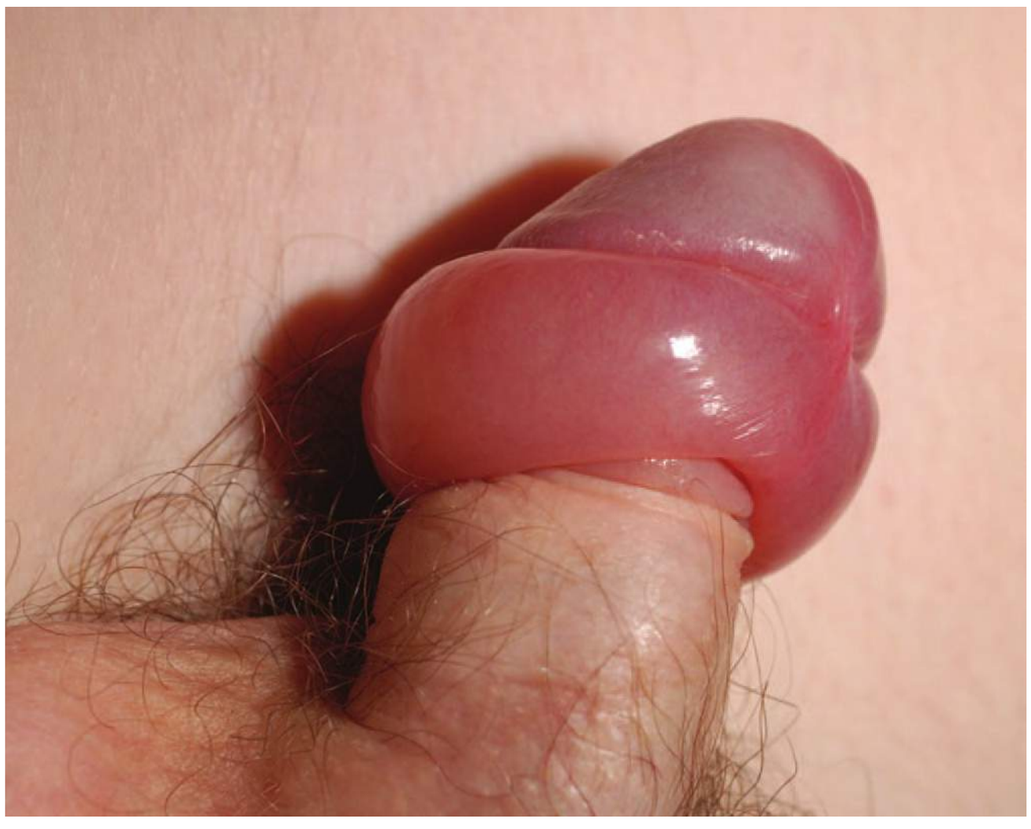


FIGURE 8.18 ■ **Paraphimosis.** Moderate edema of retracted foreskin, which is entrapped behind the coronal sulcus. (Photo contributor: Lawrence B. Stack, MD.)

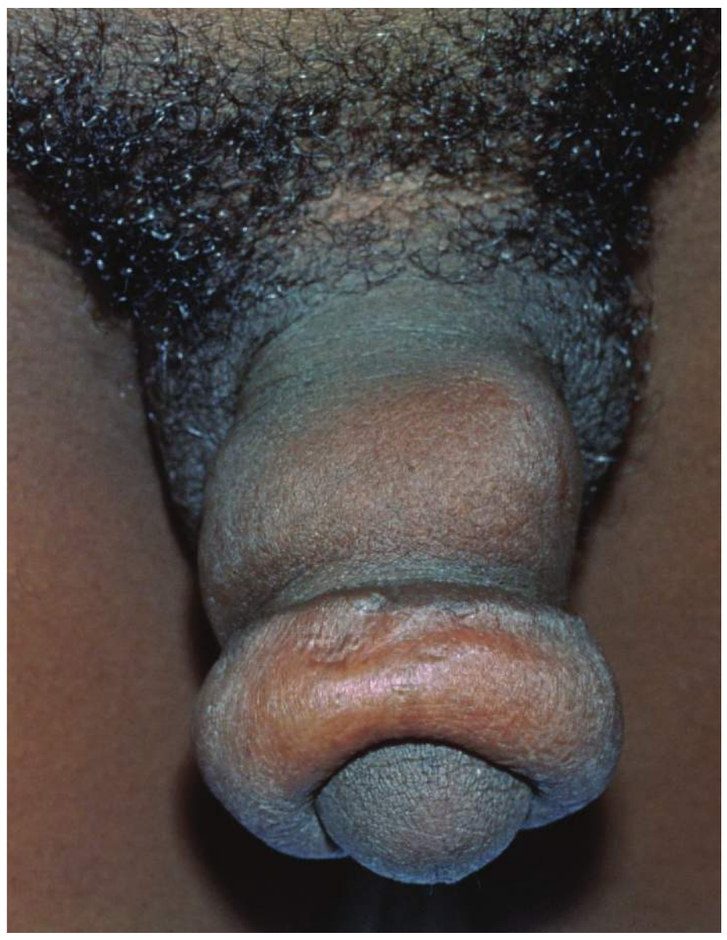


FIGURE 8.19 ■ **Paraphimosis.** Edema of retracted foreskin, which is entrapped behind the coronal sulcus. (Photo contributor: David Effron, MD.)



FIGURE 8.20 ■ **Phimosis.** Phimosis in a young patient is physiologic, but also may have obstruction from meatal stenosis and scarring. (Photo contributor: Alan B. Storrow, MD.)

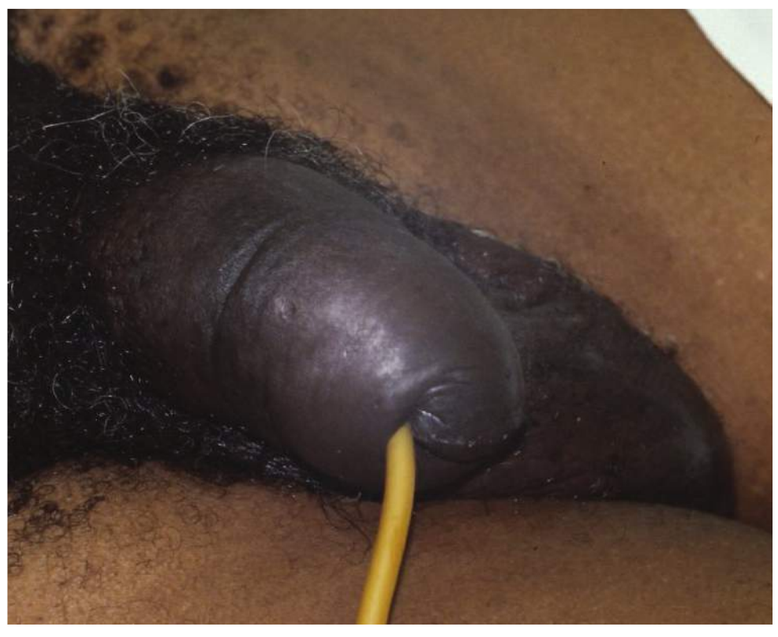


FIGURE 8.21 ■ **Phimosis.** Phimosis in an adult which required catheterization to relieve obstruction. (Photo contributor: David Effron, MD.)

PRIAPISM

Clinical Summary

These patients present with persistent, usually painful erection due to pathologic engorgement of the corpora cavernosa. Patients may present acutely or several days after their first symptoms. The glans penis and corpus spongiosum are not engorged and remain flaccid. The physiology is either arterial, which is generally traumatic, or veno-occlusive. Reversible causes of veno-occlusive disease include sickle cell disease, direct injection of erectile agents, and leukemic infiltration. Nonreversible causes include idiopathic ones (the most common), spinal cord lesions, and medications.

Management and Disposition

Treat diseases that are associated with reversible priapism. Ice packs to the perineum are frequently unsuccessful. Terbutaline given orally or subcutaneously occasionally reverses priapism. Aspiration of blood from the corpus cavernosum can lead to detumescence and should be followed by a compressive dressing. Injectable erectile agents can be reversed by aspiration followed by intracavernous injection of α-adrenergic agents such as phenylephrine. Obtain urologic consultation immediately for traumatic or persistent priapism despite initial treatment, with close urologic follow-up for those that are successfully reversed in the emergency department.

Pearls

1. Advise patients that impotence is a frequent complication of priapism, regardless of the length of the symptoms or the success of any treatment.
2. A prolonged erection with a flaccid glans and corpus spongiosum confirms priapism.
3. Urinary retention often accompanies priapism.



FIGURE 8.23 ■ **Priapism.** Aspiration of the corpora cavernosa is demonstrated. (Photo contributor: David Effron, MD.)



FIGURE 8.22 ■ **Priapism.** A painful persistent erection due to pathologic engorgement of the corpora cavernosa is seen in this patient with sickle cell disease. The glans penis and corpus spongiosum are not engorged. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 8.24 ■ **Traumatic Priapism.** A persistent erection is seen in this trauma victim who has sustained a cord injury. (Photo contributor: R. Jason Thurman, MD.)

Clinical Summary

Urethral injury is rarely an isolated event; it is often associated with multiple trauma. Anterior urethral injuries are most often the result of a straddle injury and may present late (many patients are still able to void) with a local infection or sepsis from extravasated urine. Posterior urethral injuries occur in motor vehicle and motorcycle accidents and are usually the result of pelvic fractures. Patients have blood at the urethral meatus, cannot void, and have perineal bruising. In men, the prostate is often boggy or free-floating or may not be palpable at all if there is a retroperitoneal hematoma between the prostate and the rectum.

Management and Disposition

Do not allow urethral instrumentation such as Foley catheterization prior to a retrograde urethrogram with highly concentrated water-soluble contrast. If there is only a partial anterior tear, gently attempt catheterization but abandon it at the first sign of resistance. If catheterization is unsuccessful and whenever there is a posterior tear, place a suprapubic catheter with a trocar if relief of bladder distention is required prior to operative repair.

Pearls

1. Foley catheter insertion is contraindicated in patients with a suspected urethral injury prior to a retrograde urethrogram.
2. Urethral injury should be suspected in the multiple-trauma patient who is unable to void or has blood at the meatus, a high-riding prostate, or perineal trauma.
3. Vaginal lacerations due to trauma in women should prompt consideration of a urethral tear.
4. Occasionally, urine from an anterior urethral tear will extravasate into the scrotum, causing marked swelling.
5. Posterior injuries are frequently associated with other intra-abdominal injury.



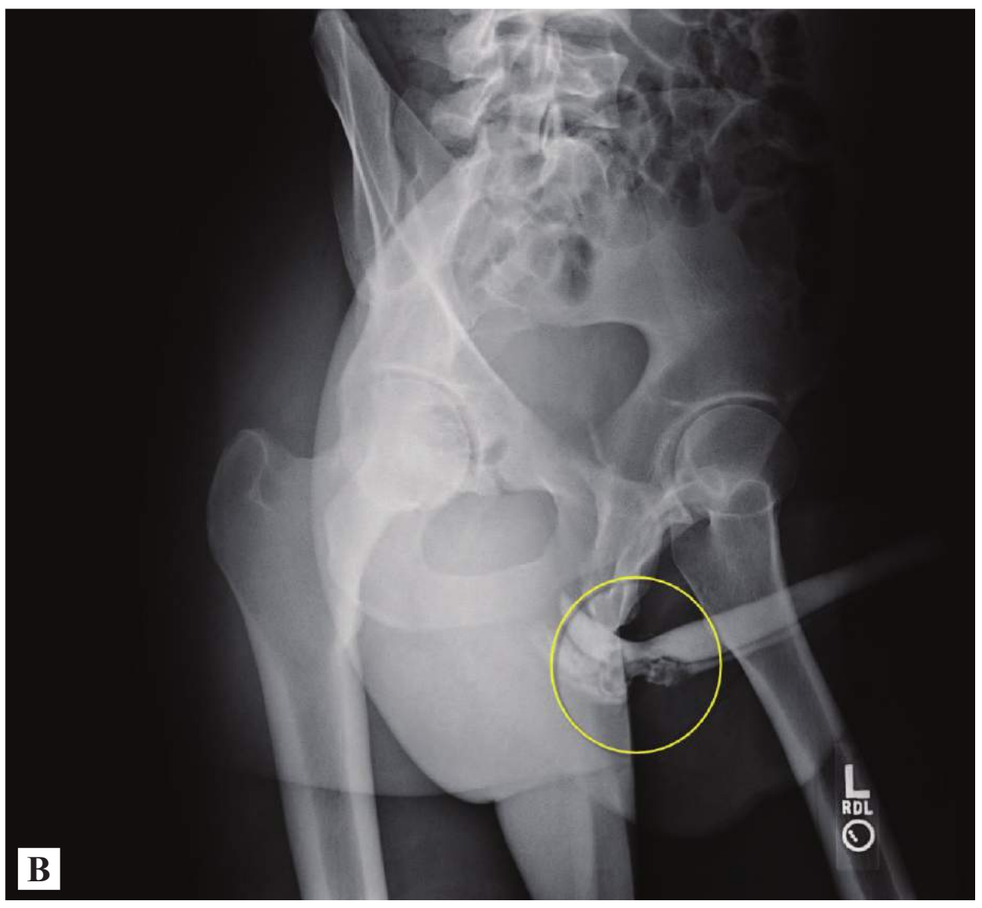


FIGURE 8.25 ■ **Urethral Rupture.** (**A**) Blood at the urethral meatus in a patient with an anterior urethral rupture secondary to a straddle injury. (**B**) A retrograde urethrogram shows extravasation of dye indicating rupture. (Photo contributor: David Effron, MD.)

FRACTURE OF THE PENIS

Clinical Summary

Patients usually present complaining of trauma during sexual arousal and often relate a sudden "snapping" sound or sensation, pain, and deformity, which is caused by a tearing of the tunica albuginea. The shaft of the penis is swollen and often angulated at the fracture site.

Management and Disposition

If the patient cannot urinate, obtain a retrograde urethrogram to rule out urethral injury. Obtain urologic referral as they frequently are taken immediately to the operating room for repair.

Pearls

1. Patients sometimes concoct elaborate stories which are not sexually related surrounding the circumstances of injury, but penile fracture most commonly occurs during sexual arousal.
2. Penile implants are also subject to injury in a similar fashion.

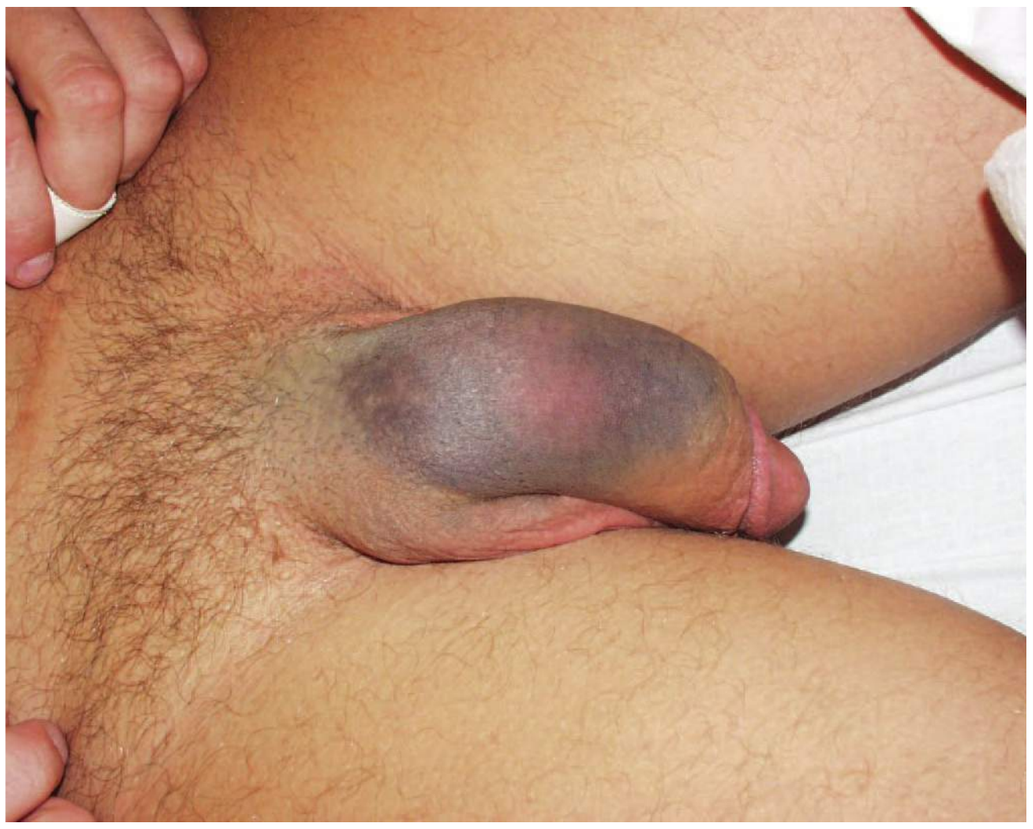


FIGURE 8.27 ■ **Fractured Penis**. Penile fracture with ecchymosis and edema without angulation. (Photo contributor: Emergency Medicine Department, Naval Medical Center Portsmouth.)



FIGURE 8.26 ■ **Fractured Penis.** Penile fracture with ecchymosis and angulation. (Photo contributor: Stephen W. Corbett, MD.)

Clinical Summary

A hair or filament that wraps around the penis can lead to a surgical emergency. The constricting band will impair distal venous and lymphatic drainage causing more edema and further impairing drainage in a vicious cycle. The ultimate sequela is arterial compromise and amputation. These are most commonly seen in young children although it has also been reported in men who use penile rings or constrictors.

Management and Disposition

Immediate release of the constriction generally gives relief and restores any impaired circulation. Take care not to injure any underlying structures. If the edema is so great that the constricting band cannot be easily released, obtain emergent urological consultation.

Pearls

1. Edema may obscure the hair or filament and bury it subcutaneously.
2. Measures to decrease the swelling, such as direct pressure and ice packs, may facilitate visualization and incision of the tourniquet.
3. Clitoral tourniquets have been described.
4. Penile tourniquet should be considered when presented with a fussy infant.



FIGURE 8.28 ■ **Penile Tourniquet.** Penile engorgement due to self-inflicted tourniquet in a prepubescent boy. (Photo contributor: Lawrence B. Stack, MD.)

STRADDLE INJURY

Clinical Summary

In straddle injury, the patient has pain, swelling, contusion, and hematoma of the perineum or scrotum following direct blunt trauma. This injury is commonly caused by a fall onto a bicycle frame cross-tube, playground equipment, or a toilet seat. Swelling can be severe enough to interfere with urination. Scrotal contents can also be contused or crushed with this injury.

Management and Disposition

Treatment is supportive and includes cold packs, elevation, rest, and analgesics. If unable to void, catheterize the patient.

Pearls

1. Perineal laceration can be obscured by swelling if a careful examination is not performed.
2. Pelvic radiographs should be obtained in all perineal injuries.
3. Men and women are at high risk for urethral injuries with this type of injury.
4. Straddle injury is differentiated from abuse with a good history from a reliable caregiver that matches the injury.



FIGURE 8.29 ■ **Straddle Injury.** Marked ecchymosis, swelling, and contusion of the perineum in an adolescent girl. Examination under anesthesia was required to determine the extent of this patient's injuries. (Photo contributor: Lawrence Stack, MD.)



FIGURE 8.30 ■ **Straddle Injury.** Contusion of the scrotum and lower abdomen in a young boy consistent with a straddle injury. (Photo contributor: David W. Munter, MD.)



FIGURE 8.31 ■ **Straddle Injury.** This unfortunate patient suffered a vaginal tear as a result of slipping forward off the seat of her bicycle and landing on its top tube. (Photo contributor: R. Jason Thurman, MD.)

Clinical Summary

Balanoposthitis is an infection and inflammation of the glans penis that also involves the overlying foreskin (prepuce). Balanitis is isolated to the glans, whereas posthitis involves only the prepuce. Pain, erythema, and edema of the affected parts of the penis are typically present. Patients may refrain from urination secondary to dysuria, or the edema may induce meatal occlusion leading to urinary retention or obstruction. Common etiologies include overgrowth of normal bacterial flora secondary to poor hygiene (pediatric patients), sexually transmitted diseases (adolescents and adults), and candidal infections (the elderly or immunocompromised).

Management and Disposition

Treatment is directed at the suspected etiology. Warm soaks and topical antibiotics (bacitracin) are the mainstay of therapy for infectious etiologies owing to poor hygiene. Counsel parents about proper cleansing and handling of the prepuce. Oral or intravenous antibiotics may be indicated if there is an accompanying cellulitis. If urinary obstruction is present, attempt catheterization using a small catheter. If catheterization is unsuccessful, urologic consultation for emergent surgical correction of the prepuce is required. Candidal infections are treated with meticulous hygiene and topical antifungal agents. Routine urologic referral is indicated for suspected lichen sclerosus et atrophicus and squamous cell carcinoma.

Pearls

1. The inability to retract the foreskin completely is normal in young boys up to age 4 or 5. Attempting to do so could cause a paraphimosis, a true emergency.
2. Placing the child in a bathtub with warm water will help alleviate difficulty with micturition assuming that no obstruction is present.
3. Candidal balanitis or balanoposthitis may be indicative of an undiagnosed immunocompromised state.
4. Suspected sexually transmitted diseases require treatment for the partners as well.



FIGURE 8.32 ■ **Balanoposthitis.** Note the erythema, localized edema, and significantly constricted preputial orifice of the distal penis. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 8.33 ■ **Balanitis**. Candidal balanitis in an elderly patient with no other complaints. New-onset diabetes was diagnosed. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 8.34 ■ **Balanoposthitis.** This toddler presented with physiologic phimosis, inflamed distal foreskin, dysuria, and swelling of the glans. (Photo contributor: James Palma, MD.)



FIGURE 8.35 ■ **Balanoposthitis.** Candidal balanoposthitis in an uncircumcised man. (Photo contributor: Alan Storrow, MD.)

Clinical Summary

Zipper injury is usually seen in young children. The foreskin becomes entrapped in the teeth of the zipper or between the fastener and the zipper teeth as the zipper is being opened (downward). Accessibility to the median bar of the zipper facilitates removal.

Management and Disposition

Treatment is directed at removing the zipper and freeing the entrapped penile foreskin or prepuce while minimizing trauma and pain. A penile block with local injection at the base of the penis eases removal, but might not be necessary if the child is cooperative and removal can be effected atraumatically. Several methods are described depending on the mechanism.

Pearls

1. Zipper entrapment of the penis is one of the most common genital injuries in prepubertal boys.
2. When tissue is entrapped by the zipper teeth only, release can be effected by cutting the cloth of the zipper either between the teeth or below the point of entrapment.
3. Cutting the median bar with a bone cutter allows the whole zipper to fall apart and release the entrapped skin.
4. Lateral compression of the distal zipper fastener with pliers may immediately release the tissue without need for anesthesia. This method requires application of equal pressure to both the anterior and posterior fastener plates simultaneously.
5. After removal, ensure that the urethra is patent and the child can void.

FIGURE 8.36 ■ **Penile Zipper Injury.** Entrapment of the foreskin between the fastener and the zipper teeth is seen. The median bar is exposed and easily accessible for cutting. (Photo contributor: Kevin J. Knoop, MD, MS.)

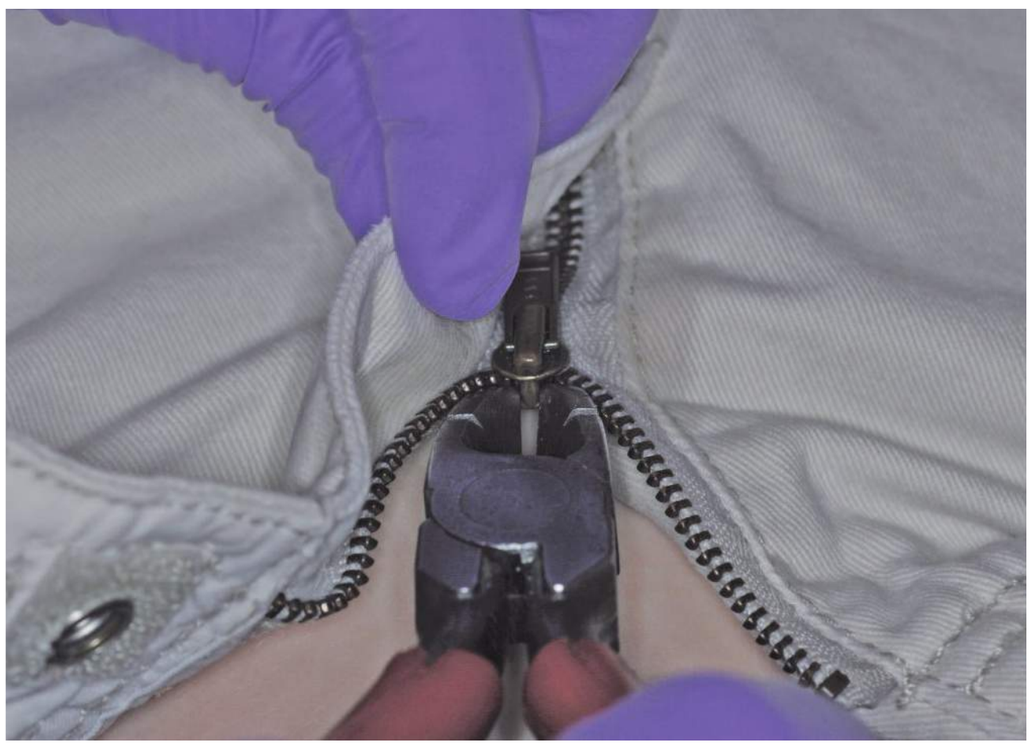


FIGURE 8.37 ■ **Penile Zipper Injury.** Separation of the zipper by cutting the median bar of the zipper with a bone cutter allows release. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 8.38 ■ **Penile Zipper Injury.** Crushed tissue after zipper removal did not obstruct urine flow in this patient. (Photo contributor: Kevin J. Knoop, MD, MS.)

VARICOCELE

Clinical Summary

A varicocele forms when the veins along the spermatic cord become engorged due to valve incompetence and is usually asymptomatic. Patients may complain of a dragging sensation or heaviness of the testicle, or notice the painless mass of veins itself (bag of worms). It usually develops slowly by the third decade of life and is on the left side 98% of the time.

Management and Disposition

Varicocele is generally harmless, but severe ones can affect fertility by impairing arterial flow to or temperature of the testicle causing atrophy. Obtain an ultrasound to assure diagnosis. Refer to a urologist and, if symptomatic, prescribe scrotal support.

Pearls

1. Rapid appearance or presentation on the right side or at an age greater than 40 years should raise concern for extrinsic compression from an abdominal/pelvic mass or renal tumor.
2. Superior mesenteric artery compression of the left renal vein against the aorta (nutcracker syndrome) can present as a varicocele.
3. Varicocele forms on the left side because the left spermatic vein anastomosis to the renal vein is at 90 degrees, while the right side anastomosis is considerably less acute.

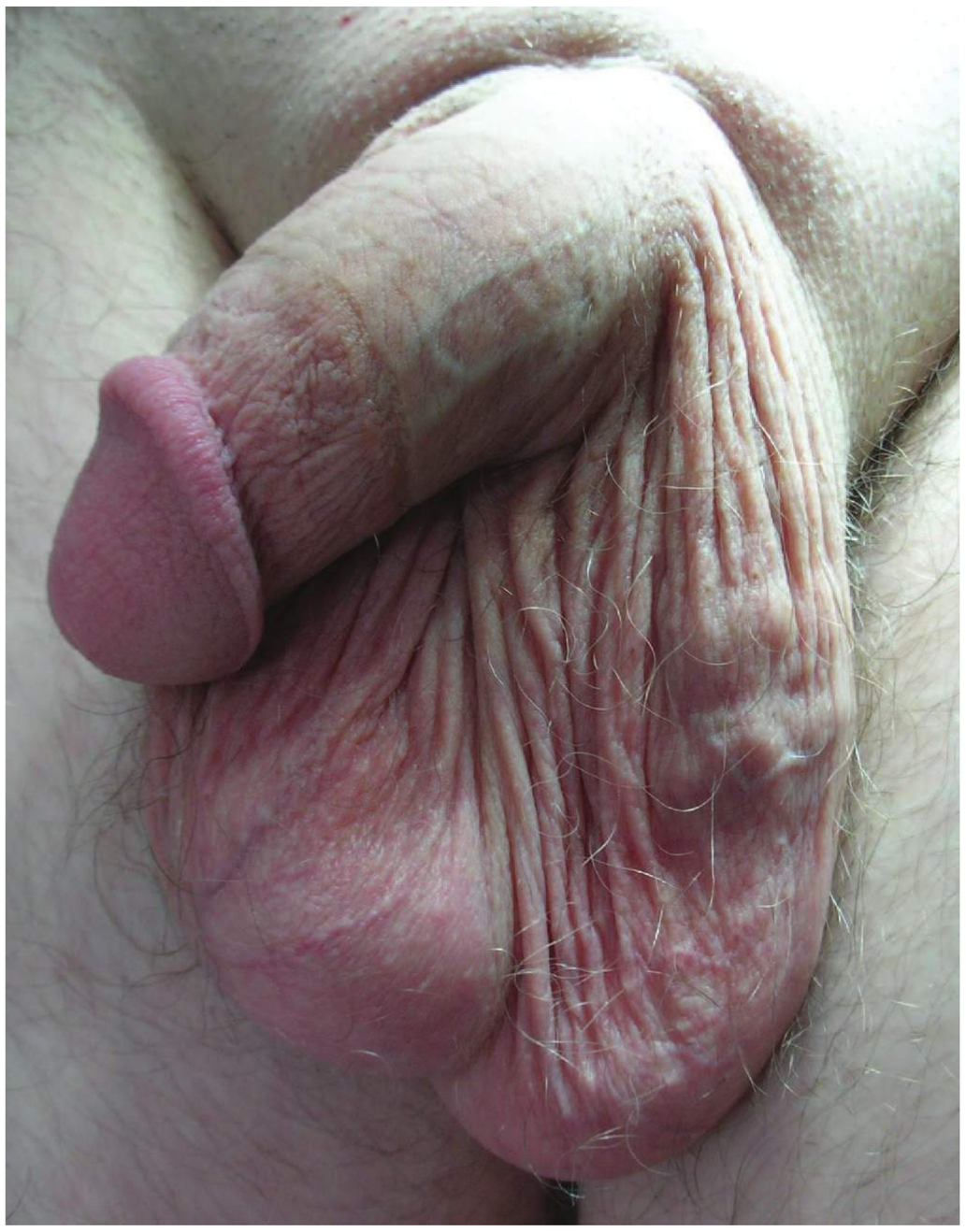


FIGURE 8.39 ■ **Varicocele.** Palpation of the nontender, twisted mass along the spermatic cord feels like a "bag of worms." (Photo contributor: Dr. Paul Turek (www.TheTurekClinic.com).)

Chapter 9

SEXUALLY TRANSMITTED INFECTIONS AND ANORECTAL CONDITIONS

Suzanne Dooley-Hash
Brian R. Sharp



Acute thrombosed external hemorrhoid. Young male suffered sudden onset of rectal pain and bleeding after a bowel movement. (Photo contributor: Lawrence B. Stack, MD.)

The authors wish to thank Diane M. Birnbaumer and Lynn K. Flowers for their contributions to prior editions.

SYPHILIS

Clinical Summary

Syphilis is a systemic illness caused by Treponema pallidum. The disease occurs in stages, with each stage having distinct physical findings. Primary syphilis is heralded by the development of a single lesion at the site of inoculation. This usually occurs within 2 to 6 weeks of exposure, but may take as long as 90 days. Patients often do not notice or seek care for the primary lesion. They may, however, present with a solitary, round, painless genital chancre or ulcer. The ulcer may also be slightly painful and multiple ulcers are sometimes seen. The base of the genital ulcer is typically dry in males and moist in females; purulent fluid in the base is uncommon. The borders of the ulcer are often indurated. Although genital ulcers are most common, ulcers may develop at any exposed site. Bilateral, nontender, nonfluctuant adenopathy is common. Without treatment, ulcers will resolve spontaneously within 3 to 12 weeks as the infection progresses to the secondary stage. Patients with primary syphilis are at risk for concurrent infection with other sexually transmitted infections.

Secondary syphilis involves systemic dissemination of T pallidum and manifests first with the development of diffuse, nonpruritic macules. The macules develop within 2 to 10 weeks after resolution of the primary ulcers. The macules later evolve into a maculopapular eruption with small, round or annular lesions and involve primarily the trunk, palms, and soles. Diffuse, painless lymphadenopathy is also seen at this stage. Mucous patches may develop on the tongue and buccal mucosa. Condyloma lata and patchy alopecia can also be seen during this stage. The manifestations of this stage resolve without treatment in several months. Despite resolution of the symptoms, untreated syphilis will persist in the body and may become latent or later manifest as tertiary syphilis, a discussion of which is beyond the scope of this text.

Management and Disposition

To confirm the diagnosis, check an RPR or VDRL. Also test the patient for HIV and other sexually transmitted infections such as Neisseria gonorrhea and Chlamydia. Parenteral penicillin G is the preferred treatment for every stage of syphilis. For primary or secondary syphilis, treat with a single dose of benzathine penicillin G, 2.4 million units, intramuscularly. Treat penicillin-allergic patients with either doxycycline 100 mg orally twice daily or tetracycline 500 mg orally four times daily for 14 days. Refer pregnant patients with penicillin allergy for desensitization and subsequent treatment with pen G as the alternative treatments are both contraindicated in pregnancy.



FIGURE 9.1 ■ **Primary Chancre—Male.** This dry-based, painless ulcer with indurated borders is typical for a primary chancre in a male patient. (Reproduced with permission from Wisdom A. A Colour Atlas of Sexually Transmitted Diseases. London: Mosby-Wolfe Ltd; 1992. Copyright © Elsevier.)



FIGURE 9.2 ■ **Primary Chancre—Male.** Painless, solitary penile ulcer in a sexually active male with multiple partners. (Photo contributor: Larry B. Mellick, MD.)



FIGURE 9.3 ■ **Primary Chancre—Female.** A solitary, painless genital chancre with a clean base in a patient with primary syphilis. (Photo contributor: Department of Dermatology, Naval Medical Center, Portsmouth, VA.)



FIGURE 9.4 ■ **Secondary Syphilis—Trunk.** Rash on trunk due to secondary syphilis. (Photo contributor: Andrew Wong, MD.)



FIGURE 9.6 ■ **Secondary Syphilis—Palms.** Resolving palmar eruption of secondary syphilis. (Photo contributor: Andrew Wong, MD.)



FIGURE 9.5 ■ **Secondary Syphilis—Palms.** Papulosquamous eruption of secondary syphilis. Note the annular appearance of the palmar scales. (Photo contributor: H. Hunter Handsfield, MD. From Handsfield HH (ed): Atlas of Sexually Transmitted Diseases, 3rd ed. New York: McGraw-Hill; 2011.)



FIGURE 9.7 ■ **Secondary Syphilis—Soles.** Hyperkeratotic plantar rash in a patient with secondary syphilis. (Photo contributor: H. Hunter Handsfield, MD. From Handsfield HH (ed): Atlas of Sexually Transmitted Diseases, 3rd ed. New York: McGraw-Hill; 2011.)

Serum titers need to be followed in 6 to 12 months to confirm adequate response to treatment. Presumptively treat any sexual partners the patient has had within 90 days and recommend serologic testing for any partners outside of 90 days with subsequent treatment based on the results. All cases of syphilis must be reported to the appropriate public health agency.

Warn patients about the potential development of the Jarisch-Herxheimer reaction which is caused by the massive release of endotoxins from the dying spirochetes following antibiotic treatment. This reaction usually occurs within hours of treatment. It is characterized by fever, headache, malaise, and myalgias and is self-limited.

Pearls

1. Consider dark-field microscopy examination of scrapings from the lesion of the primary stage or from the rash, mucocutaneous patches, or condyloma lata of the secondary stage to rapidly confirm the diagnosis.
2. Lesions of secondary syphilis are very infectious. Always wear gloves when examining a patient with a rash that may be due to secondary syphilis.
3. Pretreatment with nonsteroidal anti-inflammatory drugs may attenuate the Jarisch-Herxheimer reaction.

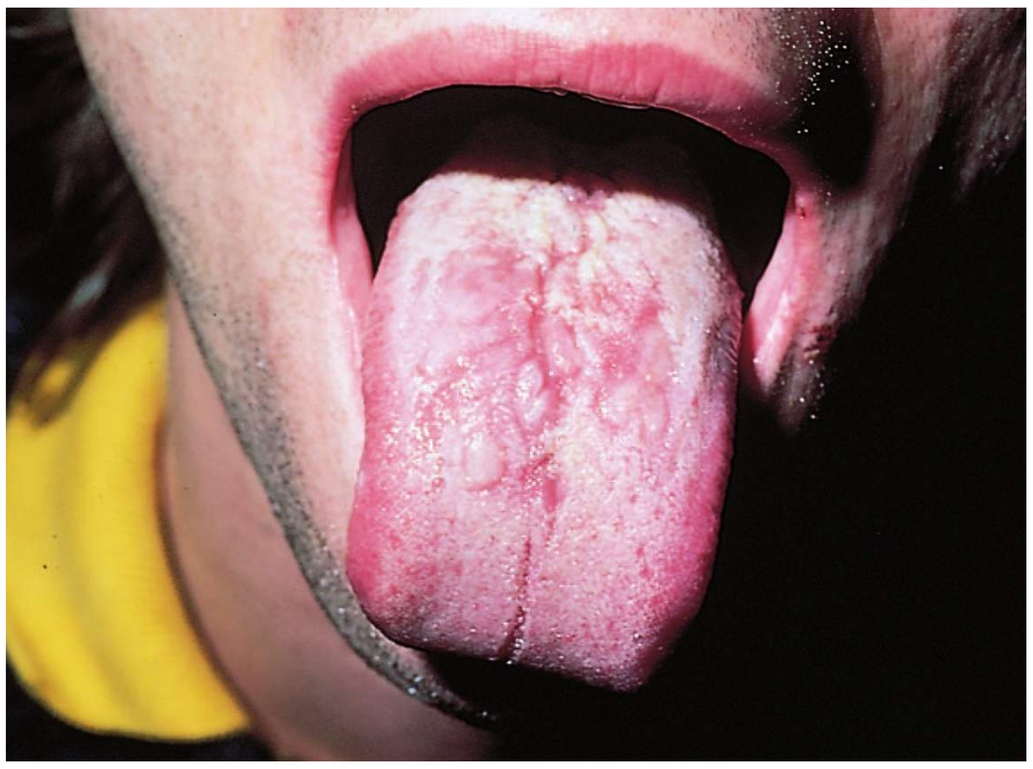


FIGURE 9.8 ■ **Mucous Patches.** Oral involvement in secondary syphilis manifested by mucous patches. These lesions are very infectious, and dark-field examination is often positive for spirochetes. (Reproduced with permission from Morse SA, Holmes KK, Ballard RC, Moreland AA (eds). Atlas of Sexually Transmitted Diseases and AIDS. 4th ed. Saunders Elsevier; 2010. Copyright © 2010 Elsevier, Ltd.)



FIGURE 9.9 ■ **Condyloma Lata.** Typical appearance of the smooth, broad-based, hypopigmented flat plaques of condyloma lata, a manifestation of secondary syphilis. (Photo contributor: H. Hunter Handsfield, MD. From Handsfield HH (ed): Atlas of Sexually Transmitted Diseases, 3rd ed. New York: McGraw-Hill; 2011.)

Clinical Summary

Infection with N gonorrhea may occur at any exposed site and has an incubation period of 2 to 7 days. Up to 10% of infections are asymptomatic in men as are as many as 80% in women. Urethritis is the most common manifestation of infection in men and is characterized by copious amounts of purulent urethral discharge and dysuria. Although women may also develop urethritis and dysuria, their infections more commonly manifest as cervicitis. If symptoms are present, they include vaginal irritation, discharge, or spotting, particularly with intercourse. On speculum examination, the cervix appears friable and a mucopurulent endocervical exudate may be present.

N gonorrhea infections can spread throughout the body. Disseminated gonococcal infections (DGI) occur more frequently in women. DGI most often present as a monoarticular septic arthritis of a knee, elbow, wrist, or ankle. Endocarditis and meningitis must be ruled out in any patient with DGI. A rash consisting of necrotic pustules on an erythematous base may also be present. The skin lesions may ulcerate, are more commonly found on the distal extremities, and may involve mucosal surfaces.

Ophthalmic gonococcal infections can occur in anyone but are seen most often in neonates exposed during birth. Gonococcal conjunctivitis develops within 2 to 7 days of birth and is characterized by chemosis and copious purulent exudate; untreated, these patients can develop endophthalmitis and perforation of the globe with permanent loss of vision.

Management and Disposition

Urethritis and cervicitis: Treat with a single dose of ceftriaxone 250 mg intramuscularly. Treatment with one dose of oral cefixime 400 mg orally is no longer recommended as an alternative and if used should be followed with a test of cure in 7 days.

Conjunctivitis: Treat adults with a single dose of ceftriaxone 1 g intramuscularly. Treat infants and children over 2 months of age with ceftriaxone 25 to 50 mg/kg once or use cefotaxime 100 mg/kg for those younger than 2 months; eye irrigation with normal saline as needed. All neonates should receive empiric treatment with ophthalmic erythromycin ointment at birth.

DGI: Preferred initial treatment is ceftriaxone 1 g intravenously or intramuscularly once daily for at least 7 to 10 days. Patients with suspected endocarditis or meningitis are treated with higher doses of ceftriaxone (1-2 g every 12 hours). Alternative agents for parenteral treatment are: cefotaxime 1 g intravenously every 8 hours, or ceftizoxime 1 g intravenously every 8 hours. Parenteral treatment is continued for 24 to 48 hours after symptoms improve and for a minimum of 7 to 10 days. Parenteral treatment is followed by a 7 to 10 course of oral antibiotics; cefixime 400 mg orally twice daily or cefpodoxime 400 mg orally twice daily. All sexual partners should be notified and treated empirically. Gonorrhea is a reportable disease.

Pearls

1. Treat patients with gonorrhea for presumed concurrent infection with Chlamydia. Coinfection with these organisms is seen in 30% of men with urethritis and 50% of women with cervicitis.



FIGURE 9.10 ■ **Male Gonococcal Urethritis.** Purulent urethral discharge from a patient with gonococcal urethritis. (Photo contributor: Larry B. Mellick, MD.)



FIGURE 9.11 ■ **Female GC Urethritis.** Gonococcal urethritis in a female patient. Note the purulent urethral discharge. (Reproduced with permission from Morse SA, Holmes KK, Ballard RC, Moreland AA (eds). Atlas of Sexually Transmitted Diseases and AIDS. 4th ed. Saunders Elsevier; 2010. Copyright © 2010 Elsevier, Ltd.)

2. Gonococcal arthritis is the most common cause of monoarticular arthritis in young, sexually active patients.
3. Suspect gonococcal conjunctivitis in patients with copious eye discharge and chemosis.
4. Cultures are the gold standard for confirming the diagnosis of DGI. Selective media should be used when specimens are obtained from the cervix, pharynx, urethra, or rectum. Nonselective medium (blood agar) should be used in culturing joint fluid, blood, or cerebrospinal fluid.

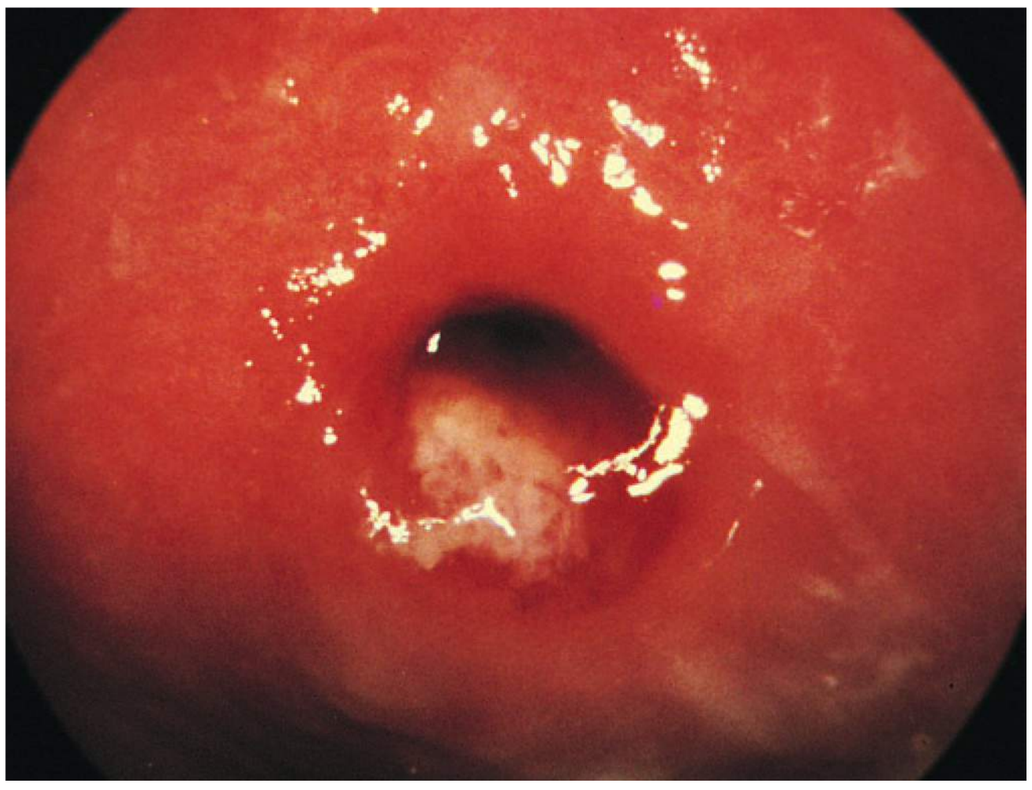


FIGURE 9.12 ■ **Cervicitis.** Endocervical purulent exudate in an asymptomatic patient with gonococcal cervicitis. The cervix is very friable. (Photo contributor: King K. Holmes, MD. From Handsfield HH (ed): Atlas of Sexually Transmitted Diseases, 3rd ed. New York: McGraw-Hill; 2011.)

FIGURE 9.13 ■ **Hyperacute Conjunctivitis.** Chemotic conjunctiva and copious purulent exudate in a patient with gonococcal conjunctivitis. (Photo contributor: Lawrence B. Stack, MD.)

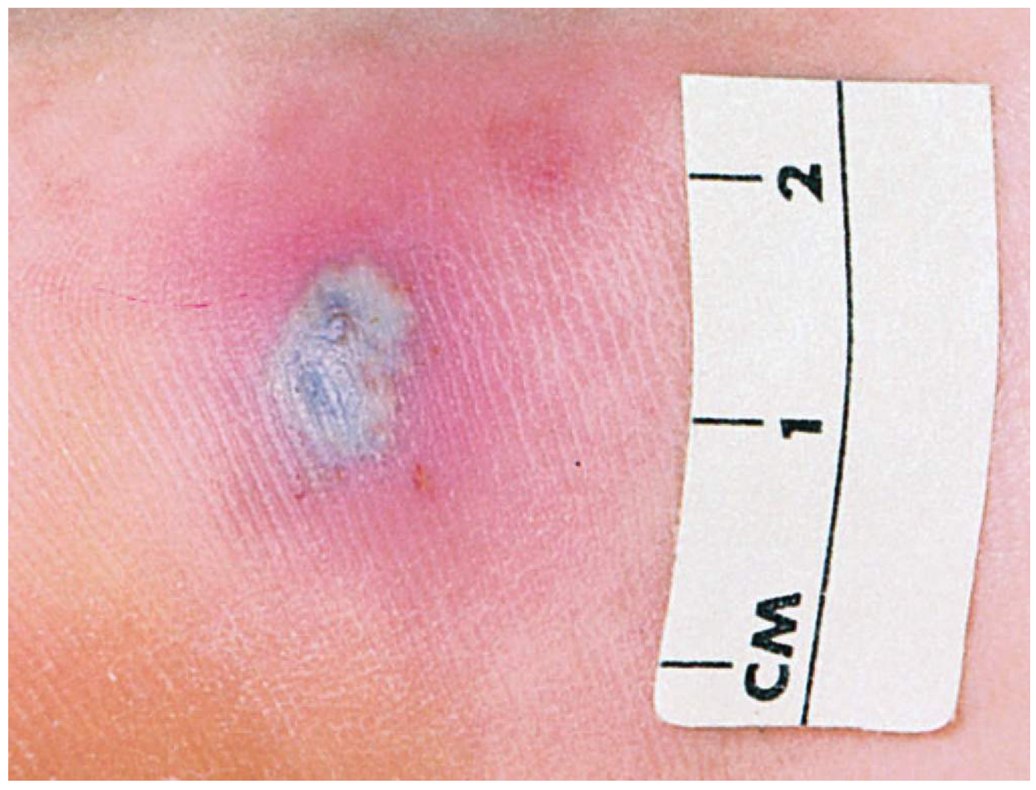


FIGURE 9.14 ■ **Skin Eruptions.** Hemorrhagic pustules suggestive of disseminated gonococcal infection. (Photo contributor: H. Hunter Handsfield, MD. From Handsfield HH (ed): Atlas of Sexually Transmitted Diseases, 3rd ed. New York: McGraw-Hill; 2011.)

FIGURE 9.15 ■ **Bartholin Abscess.** Enlarged, fluctuant, tender Bartholin abscess of the labia, usually but not always a result of gonorrhea. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Infections with Chlamydia trachomatis typically have an incubation period of 1 to 3 weeks. Men may develop urethritis and present with a thin, nonpurulent urethral discharge and/or dysuria. Up to 50% of men with urethritis may be asymptomatic. Men may also develop epididymitis which can cause sterility if untreated.

Women may also develop urethritis and present with dysuria. These infections can be misdiagnosed as urinary tract infection due to the presence of pyuria on urinalysis. Chlamydial cervicitis in women is usually asymptomatic and often goes undiagnosed. Chlamydial infections in women are more likely to spread to the upper genital tract and may result in pelvic inflammatory disease. This can lead to tubo-ovarian abscess formation with resultant fallopian tube scarring and other sequelae of infection that increase the woman's risk for subsequent ectopic pregnancy. Chlamydial infections are a major cause of female infertility.

Management and Disposition

Preferred treatment is a single oral dose of azithromycin 1 g given in the emergency department. A 7-day course of doxycycline 100 mg taken orally twice daily is equally effective, but should not be used if there are any concerns about medication compliance with the longer course. Alternative treatments include a 7-day course of either ofloxacin 300 mg orally twice daily, or levofloxacin 500 mg orally once daily, or erythromycin 500 mg orally four times daily, or erythromycin ethylsuccinate 800 mg four times daily. Advise patients that their sexual partners should be examined and treated appropriately and, to reduce partner transmission, recommend sexual abstinence for 7 days.

Pearls

1. Chlamydial infection often accompanies gonococcal infection. Empirically treat any patient being treated for gonorrhea for concurrent chlamydial infection and vice versa.
2. Women with chlamydial infections may be completely asymptomatic for long periods of time.
3. Consider syphilis serologic testing and HIV testing in patients presenting with any sexually transmitted infection.

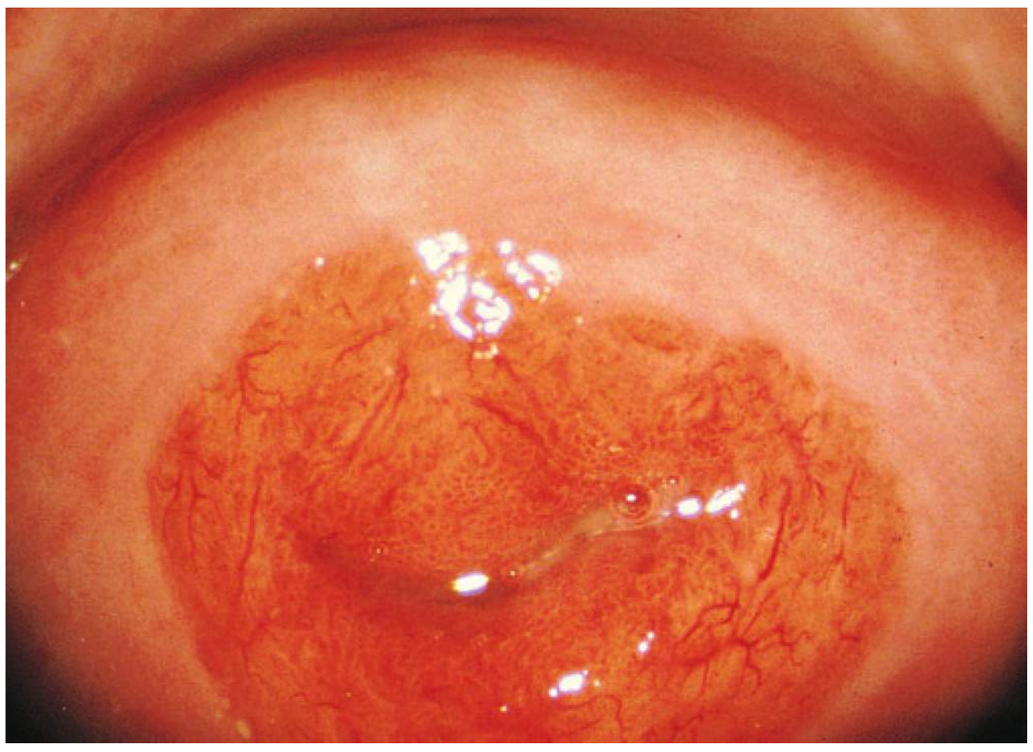


FIGURE 9.17 ■ **Chlamydial Cervicitis.** Mucopurulent cervicitis from chlamydial infection. (Photo contributor: Claire E. Stevens, PA. From Handsfield HH (ed): Atlas of Sexually Transmitted Diseases, 3rd ed. New York: McGraw-Hill; 2011.)



FIGURE 9.16 ■ **Male Chlamydial Urethritis.** Thin urethral discharge of chlamydial urethritis. (Photo contributor: Walter Stamm, MD. From Handsfield HH (ed): Atlas of Sexually Transmitted Diseases, 3rd ed. New York: McGraw-Hill; 2011.)



FIGURE 9.18 ■ **Chlamydia Conjunctivitis**. Follicles (focal areas of conjunctival inflammation) and a thin watery discharge are seen typical of chlamydia conjunctivitis. This is usually an indolent course lasting weeks to months. Follicles and inflammation are best seen on the upper tarsal conjunctiva. (Photo contributor: Jeffrey Goshe, MD.)

Clinical Summary

Herpes genitalis refers to infection with the herpes simplex virus (HSV)—most commonly HSV-2. The patient may present with a symptomatic primary infection (PI), a first-episode nonprimary infection, or a recurrent episode. The majority of individuals with HSV, however, are unaware of their infection.

PI refers to the initial transfer of the virus. The patient may remain asymptomatic at this time, and may later present with a first-episode nonprimary infection. Patients who develop symptoms with the PI may experience a prodromal viral syndrome consisting of a low-grade fever and myalgias that start 1 to 3 days prior to the onset of the characteristic rash of genital herpes. This rash consists of multiple groups of small vesicles that quickly ulcerate into shallow, painful ulcers on an erythematous base. The ulcers may coalesce and form larger ulcers, especially in women. The lesions are typically intensely painful, last up to 3 weeks, and heal without scarring. Women may also experience urinary retention due to sacral autonomic dysfunction. Although the majority of PIs are relatively mild and self-limited, more serious infections can occur, especially in the immunosuppressed. Up to 10% of patients develop aseptic meningitis. Disseminated infections can also result in a diffuse rash, pneumonitis, hepatitis, and encephalitis.

The clinical presentation of first-episode nonprimary genital herpes and recurrent genital herpes is less dramatic. Systemic symptoms are usually absent and patients typically have only a single to a few painful lesions. First-episode nonprimary outbreaks resolve within 1 to 2 weeks, whereas recurrent episodes generally last less than a week. Recurrences of genital herpes are often heralded by a warning prodrome of tingling or numbness in the perineal area.

Management and Disposition

Counseling of patients with genital herpes that this is a sexually transmitted infection with a chronic and recurrent course is imperative. Evaluate all patients for other possible infections and recommend evaluation and appropriate treatment for all sexual partners. Definitive diagnostic testing includes cell culture, HSV polymerase chain reaction, and serologic testing.

Advise patients that treatment with antiviral agents can decrease the length and severity of symptoms but cannot completely eradicate the infection from the body. Antivirals may also be used for suppressive treatment for patients with frequent recurrences or immunosuppression, and decrease episodes by 70% to 80%.

Treat primary genital herpes with acyclovir, 400 mg orally three times daily, or 200 mg orally five times daily, for 7 to 10 days or until symptoms resolve. Alternatives include famciclovir 250 mg orally three times daily or valacyclovir 1 g orally twice daily for 7 to 10 days.



FIGURE 9.19 ■ **Primary Genital Herpes—Female.** Multiple coalescing superficial ulcerations of primary genital herpes. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 9.20 ■ **Herpetic Ulcerations—Female.** Multiple coalescing vesicles and ulcers causing a deep ulcer. (Photo contributor: Lawrence B. Stack, MD.)

For recurrent genital herpes, treatment must be started within 1 day of the onset of symptoms in order to be effective. Multiple antiviral treatment regimens are appropriate. Five-day oral courses include acyclovir 400 mg three times daily, acyclovir 800 mg twice daily, famciclovir 125 mg twice daily, or valacyclovir 1 g once daily. Shorter courses include acyclovir 800 mg three times daily for 2 days, famciclovir 1000 mg PO twice daily for 1 day, or valacyclovir 500 mg twice daily for 3 days.

Admit patients with disseminated herpes infections and other complications for treatment with intravenous antiviral medications.



FIGURE 9.21 ■ **Primary Lesions—Male.** Multiple genital vesicles of primary genital herpes. (Photo contributor: H. Hunter Handsfield, MD. From Handsfield HH (ed): Atlas of Sexually Transmitted Diseases, 3rd ed. New York: McGraw-Hill; 2011.)



FIGURE 9.22 ■ **Primary Lesions—Male.** Confluence of ulcerations on an erythematous base in a patient with primary herpes simplex type II. (Photo contributor: David Effron, MD.)

Pearls

1. Perinatal transmission of genital herpes is associated with high rates of infant morbidity and mortality. Infected women must be counseled to inform their obstetrician of their history of herpes when they become pregnant.
2. Genital herpes is the most common cause of ulcerating genital lesions in the developed world.
3. Topical antiviral agents are not effective against genital herpes.

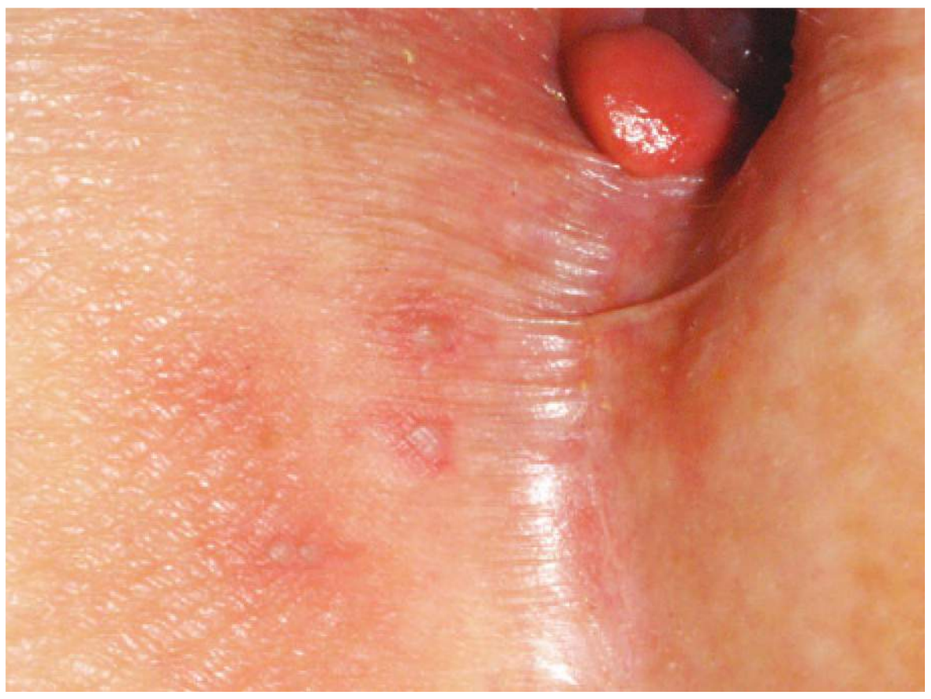


FIGURE 9.23 ■ **Recurrent Lesions—Female.** Few, small, minimally painful lesions of recurrent genital herpes. (Photo contributor: Hope Haefner, MD.)

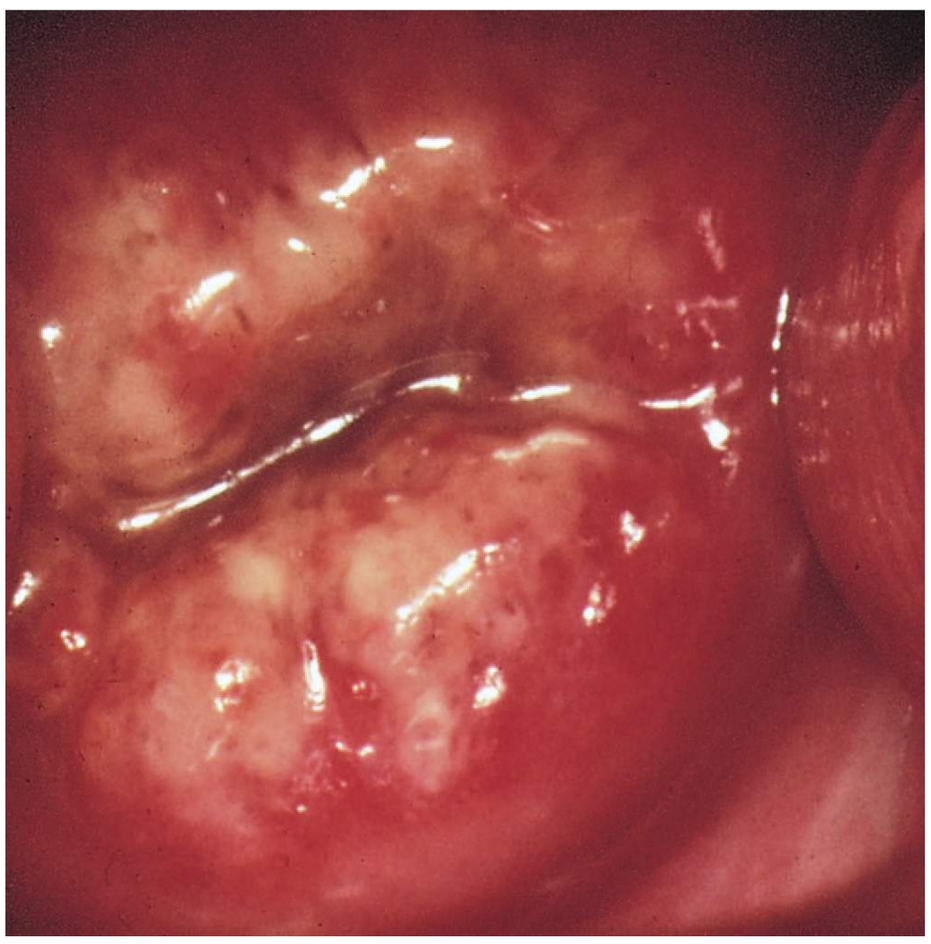


FIGURE 9.24 ■ **Herpes Simplex Virus—Cervix.** Erosive ulcerations of the cervix in a patient with genital herpes infection. This patient may be completely asymptomatic and may transmit the disease. (Reproduced with permission from Wisdom A. A Colour Atlas of Sexually Transmitted Diseases. London: Mosby-Wolfe Ltd; 1992. Copyright © Elsevier.)

LYMPHOGRANULOMA VENEREUM

Clinical Summary

Lymphogranuloma venereum (LGV) is caused by the L1, L2, or L3 serotypes of C trachomatis and is primarily a disease of lymphatic tissue. Initially, LGV causes a self-limited, painless genital ulceration that is rarely noticed by the patient. Patients typically present later with painful, firm femoral or inguinal adenopathy which is often but not always unilateral. Lymphadenopathy may occur both above and below the inguinal ligament, causing the "groove sign" suggestive of this diagnosis. The enlarged lymph nodes may spontaneously open into draining sinus tracts to the skin. Patients with rectal exposure may present with rectal pain, drainage, and tenesmus due to proctocolitis with possible development of fistulas or strictures.

Management and Disposition

Treat with doxycycline 100 mg orally twice daily for 21 days. Alternatives are erythromycin base 500 mg orally four times daily for 21 days or azithromycin 1g orally once weekly for 3 weeks. Needle aspiration of large fluctuant lymph nodes may be necessary. Avoid incision and drainage as larger openings increase the risk of sinus tract formation. Diagnosis is primarily based on clinical suspicion and findings, but serologic testing may be used to confirm the diagnosis.

Pearls

1. Evaluate patients with LGV for concurrent sexually transmitted diseases and treat accordingly. Advise treatment of any sexual partners within the past 60 days.
2. The lymphadenopathy of LGV progresses over several weeks.
3. Treatment for LGV requires completion of a full 3 weeks of therapy for a cure.

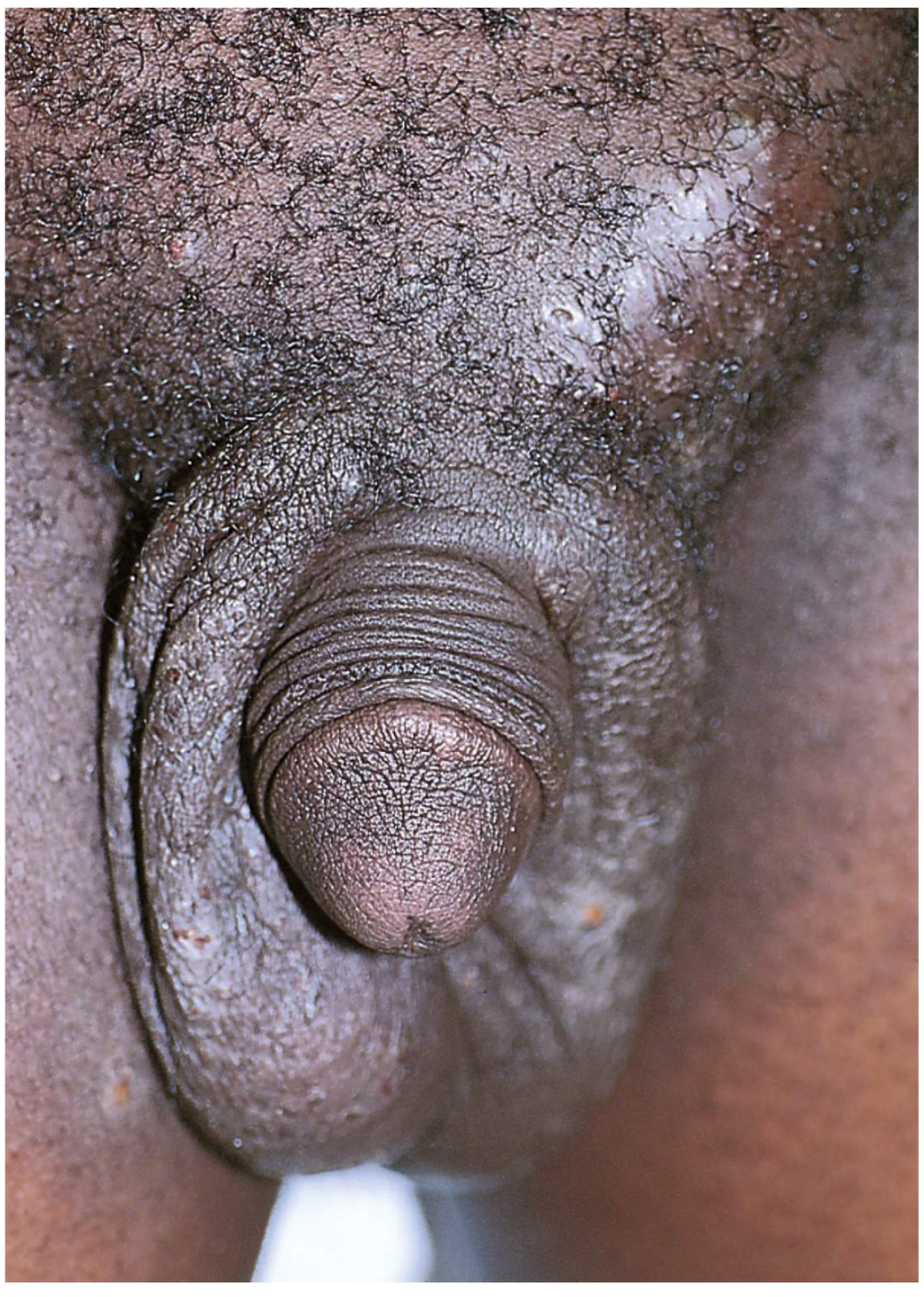


FIGURE 9.25 ■ **Lymphogranuloma Venereum.** Unilateral left lymphadenopathy in a patient with lymphogranuloma venereum. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Chancroid is caused by infection with Haemophilus ducreyi. After an incubation period of 2 to 10 days, patients develop multiple painful genital ulcerations that are not indurated. The ulcers are often deep and undermined and may have a purulent base. Painful suppurative inguinal lymphadenopathy may develop. Infected lymph nodes can become large and may spontaneously rupture. Systemic symptoms are uncommon.

Management and Disposition

Treat with a single dose of intramuscular ceftriaxone 250 mg or oral azithromycin 1 g. Alternative treatments include ciprofloxacin 500 mg orally twice daily for 3 days or erythromycin base 500 mg orally four times daily for 7 days; however, increasing resistance to both of these agents limits their usefulness. Refer for follow-up examination in 7 days to evaluate response to treatment. In advanced cases, scarring can result despite successful treatment. Aspirate large, fluctuant nodes to prevent rupture, but incision and drainage should be avoided to prevent development of chronically draining sinus tracts. Evaluate for other sexually transmitted infections and advise patients that all sexual partners should be notified of exposure to the disease and treated appropriately.

Pearls

1. Chancroid is a diagnosis of exclusion as culturing H ducreyi requires a special medium that is <80% sensitive and not readily available. Genital herpes and syphilis must be ruled out.
2. Incidence of chancroid is decreasing worldwide and it now occurs primarily in sporadic outbreaks among high-risk individuals.
3. The presence of chancroid increases the risk of HIV transmission.
4. The ulcerative lesions of chancroid are very tender and usually multiple.



FIGURE 9.26 ■ **Chancroid Lesions.** Multiple painful, deep ulcerations of chancroid. (Photo contributor: H. Hunter Handsfield, MD. From Handsfield HH (ed): Atlas of Sexually Transmitted Diseases, 3rd ed. New York: McGraw-Hill; 2011.)



FIGURE 9.27 ■ **Chancroid Lesions and Inguinal Nodes.** Chancroid lesions with an enlarged lymph node. On examination, this node is tender and fluctuant. (Photo contributor: H. Hunter Handsfield, MD. From Handsfield HH (ed): Atlas of Sexually Transmitted Diseases, 3rd ed. New York: McGraw-Hill; 2011.)

CONDYLOMA ACUMINATA (GENITAL WARTS)

Clinical Summary

Genital warts are caused by infection with human papillomavirus (HPV): most commonly strains 6 or 11. Typical lesions are flesh-colored with a cauliflower-like appearance but may be flat, sessile, or pedunculated. These warts are usually asymptomatic, but may be seen or felt by patients or their sexual partners. They range in size from 1 to 4 mm to several centimeters.

Management and Disposition

In general, genital warts do not require emergent treatment and patients may be referred for outpatient follow-up. Topical caustic agents can be prescribed if treatment must be initiated during the emergency department visit. Options include podofilox applied twice daily for 3 days, followed by 4 days of no therapy for up to four cycles, imiquimod cream applied once daily at bedtime, three times weekly for up to 16 weeks, and sinecatechin ointment applied three times daily for up to 16 weeks. Imiquimod should be washed off within 10 hours of application, while sinecatechin should not be washed off. The patient should be advised to avoid sexual contact while these agents are in use. Safety during pregnancy has not been established for any of these agents.



FIGURE 9.29 ■ **Genital Warts—Male.** Typical appearance of condyloma acuminata of the glans penis. (Reproduced with permission from Morse SA, Holmes KK, Ballard RC, Moreland AA (eds). Atlas of Sexually Transmitted Diseases and AIDS. 4th ed. Saunders Elsevier; 2010. Copyright © 2010 Elsevier, Ltd.)



FIGURE 9.28 ■ **Genital Warts—Female.** Verrucous lesions of the posterior fourchette in a patient with condyloma acuminata. (Photo contributor: H. Hunter Handsfield, MD. From Handsfield HH (ed): Atlas of Sexually Transmitted Diseases, 3rd ed. New York: McGraw-Hill; 2011.)



FIGURE 9.30 ■ **Genital Warts—Male.** Cauliflower-like appearance of condyloma acuminata of the foreskin of this uncircumcised male. (Photo contributor: Lawrence B. Stack, MD.)

Advise patients that recurrence is common and multiple treatments may be needed to completely eradicate the condyloma. Refer for appropriate follow-up care and consideration of other therapies which include cryotherapy, electrocautery, and trichloracetic acid. Laser therapy or surgery may be needed in cases of giant warts. Patients should be evaluated for other sexually transmitted infections. HPV infection is also associated with increased risk of cervical cancer. Refer women with genital warts for gynecological evaluation including Pap smear to evaluate for coexisting carcinoma in situ.

Pearls

1. Immunization of young women against HPV is decreasing its incidence in the United States.
2. Genital warts are considered a sexually transmitted condition. Their presence in a child should raise suspicion for child sexual abuse.
3. Large lesions should be biopsied to rule out cancer.
4. In cases where the diagnosis is not obvious, perform serologic testing (VDRL or RPR) for syphilis to rule out condyloma lata.



FIGURE 9.31 ■ **Perianal Condyloma Acuminata.** Multiple perianal pedunculated warts are seen in this patient. (Photo contributor: Larry B. Mellick, MD.)



FIGURE 9.32 ■ **Giant Warts—Female.** Extensive condyloma acuminata in a female. (Photo contributor: Hope Haefner, MD.)



FIGURE 9.33 ■ **Genital Warts—Child.** Condyloma in female child should raise suspicion for sexual abuse. (Photo contributor: Hope Haefner, MD.)



FIGURE 9.34 ■ **Giant Warts—Male.** Giant warts in a male patient with extensive condyloma acuminata. (Reproduced with permission from Wisdom A. A Colour Atlas of Sexually Transmitted Diseases. London: Mosby-Wolfe Ltd; 1992. Copyright © Elsevier)

PEDICULOSIS

Clinical Summary

Pediculosis can be caused by either the body louse or the crab louse. Body lice are not sexually transmitted and tend to cluster around the waist, shoulders, axillae, neck, and head. Eyelash infestations can also occur. Hypersensitivity reactions to louse saliva cause intense pruritus and patients may present with complaints of intense itchiness and excoriations. The lice are very small and may not be easily seen. The larval form of the louse, the nit, may be mistaken for dandruff in the hair. Unlike dandruff, however, the nits are extremely adherent to the hair shaft and cannot be brushed out of the hair. The adult lice and their eggs are often found in the seams of clothing.

Pubic infestation is caused by Phthirus pubis, the crab louse. Patients may present with intense pruritis in the pubic area; however, as many as half of patients with this infestation may be asymptomatic. Patients may notice the lice or may note tiny rust-colored spots on their underwear, which represent bleeding from the sites of louse bites. Nits may be found at the base of pubic hair and hatch in 5 to 10 days.

Management and Disposition

Treat pubic lice with topical permethrin 1% cream rinse or pyrethrins with piperonyl butoxide. Either agent should be washed off after 10 minutes. Repeat treatment in 1 week to treat any nits that may have hatched. Alternative regimens include topical malathion 0.5% lotion washed off after 8 to12 hours or oral ivermectin 250 μg/kg once and repeated in 2 weeks. Treat eyelash infestations with twice daily application of petroleum jelly or other occlusive ophthalmic ointment for 10 days. Mechanical removal of nits attached to hair may also be attempted.

Advise patients to wash all clothing or linens used in the preceding 24 hours. Also advise avoiding sexual contact until cured and examination of all sexual partners.

Pearls

1. Nits are easier to find on examination than are mature lice; the average number of lice in an infestation is only 10.
2. Patients with pediculosis pubis should be evaluated for other sexually transmitted infections.
3. Although an effective treatment, lindane is no longer recommended as first-line therapy of pubic lice due to concerns regarding toxicity.



FIGURE 9.35 ■ Phthirus pubis—**On Pubic Hair.** Phthirus pubis, or the crab louse, in the pubic hair (pediculosis pubis) of a patient complaining of itching. Note the nits attached to the hair. (Reproduced with permission from Morse SA, Holmes KK, Ballard RC, Moreland AA (eds). Atlas of Sexually Transmitted Diseases and AIDS. 4th ed. Saunders Elsevier; 2010. Copyright © 2010 Elsevier, Ltd.)

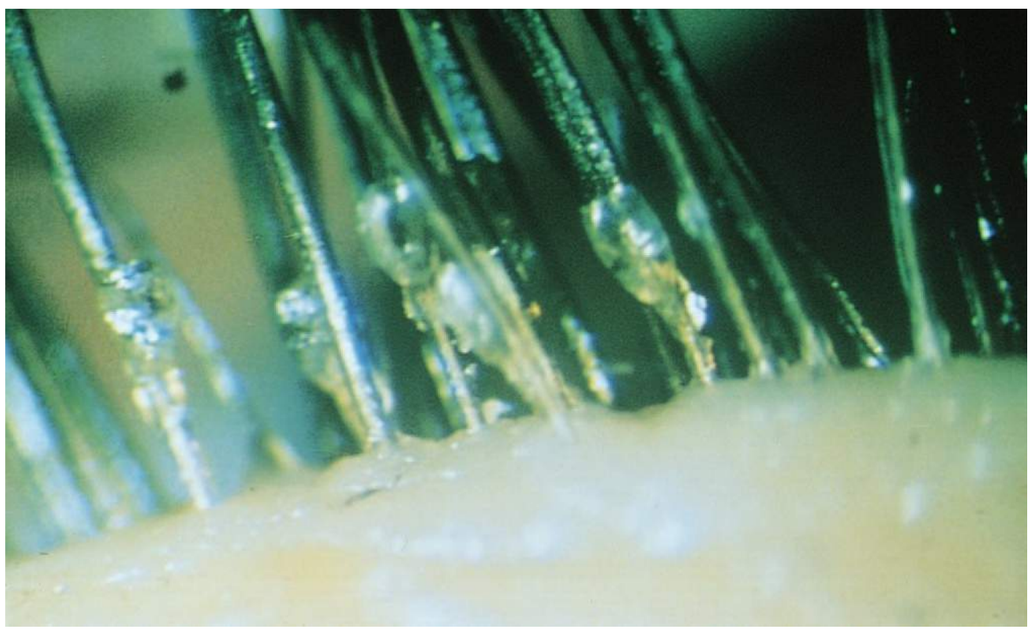


FIGURE 9.36 ■ Phthirus pubis—**On Eyelashes.** Phthirus pubis lice noted in the eyelashes. (Reproduced with permission from Spalton DJ, Hitchings RA, Hunter PA (eds). Atlas of Clinical Ophthalmology, 2nd ed. London: Mosby-Wolfe Ltd; 1994. Copyright © Elsevier.)

Clinical Summary

An anal fissure is a longitudinal tear of the skin of the anal canal and extends from the dentate line to the anal verge. It is the most common cause of painful rectal bleeding. Fissures are thought to be caused by the passage of hard or large stools with constipation, but may also be seen with diarrhea. Although seen in infants, this condition is found mostly in young and middle-aged adults (30-50 years old). Patients present with intense sharp, burning pain during and up to several hours after bowel movements. They may see bright red blood at the time or shortly after the passage of stool. Gentle examination with separation of the buttocks usually provides good visualization. Anal fissures are typically a few millimeters wide and most commonly occur in the posterior midline, where the skeletal muscle fibers encircling the anus are the weakest. An anal fissure that is off the midline should raise suspicion of a secondary cause, such as inflammatory bowel disease including ulcerative colitis, or Crohn disease, or sexually transmitted infection such as chlamydia, gonorrhea, herpes, and syphilis. Other atypical causes include tuberculosis, anal neoplasms, leukemia, and sickle cell disease.

Management and Disposition

Acute treatment of anal fissures consists of anal hygiene, bulk fiber diet supplements to soften stools, warm sitz baths, topical nitroglycerin or nifedipine gel, and topical anesthetics (lidocaine). Oral pain medication and muscle relaxants such as diazepam may be required for some certain patients. Most simple anal fissures resolve in 2 to 4 weeks, but refractory cases may require surgical repair.

Pearls

1. Pain and involuntary sphincter spasm may preclude routine digital or anoscopic examination and anesthesia may be required for adequate evaluation.
2. A proctoscopic examination should be done at some point to rule out secondary causes—particularly if location is not in the posterior midline.



FIGURE 9.38 ■ **Anal Fissure.** An anal fissure is seen at the superior midline in a patient with 2 weeks of constipation. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 9.37 ■ **Anal Fissure.** A typical anal fissure located in the posterior midline. (Photo contributor: Paul J. Kovalcik, MD.)



FIGURE 9.39 ■ **Anal fissure.** This adolescent patient complains of bloody, painful, hard stools and has an anal fissure in the midline toward the perineum. (Photo contributor: Lawrence B. Stack, MD.)

ANORECTAL ABSCESSES

Clinical Summary

Anorectal abscesses include perianal and perirectal abscesses which are named according to the involved space: ischiorectal, intersphincteric, or supralevator. These are most commonly found in males aged 30 to 50 years old and are thought to be due to occlusion of the mucus-producing anal glands. The perianal abscess is most common. Symptoms include pain in the anal area worsened by bowel movements, straining, coughing, or palpation. Examination findings include a fairly superficial, fluctuant, and possibly erythematous mass found at the perianal region. Perirectal abscesses present as fluctuant masses that are palpable along the rectal wall. Patients may complain of pain, fever, and mucous or bloody discharge with bowel movement. Crohn disease is a common cause of anorectal abscess and should be considered. Other predisposing conditions may include chronic steroid use, diabetes mellitus, leukemia or other malignancy, radiation fibrosis, trauma, or infections such as Chlamydia, herpes, TB, LGV, or actinomyces.



FIGURE 9.40 ■ **Perianal Abscess.** Swelling and erythema around the anus consistent with a perianal abscess. (Photo contributor: The American Society of Colon and Rectal Surgeons.)

Management and Disposition

Incise and drain perianal abscesses using a small radial or cruciate incision lateral to the external sphincter. For an uncomplicated abscess, this can be accomplished under local anesthesia. Clear the cavity of loculations and loosely pack with iodoform gauze. Gauze should be removed in 24 to 48 hours. All patients require outpatient follow-up. Antibiotic therapy is indicated in immunosuppressed patients and those with systemic symptoms consistent with sepsis. Consider computed tomography or magnetic resonance imaging for deep perirectal abscesses that are not palpable on rectal examination. Obtain surgical consultation for large or complicated abscesses or those requiring examination and treatment under anesthesia.



FIGURE 9.41 ■ **Perirectal Abscess—CT.** Hypodense fluid collection surrounded by enhancing ring in the right perirectal tissue suggests an abscess. (Photo contributor: Brett Bechtel, MD.)

Pearls

1. Consider admission for debilitated, elderly, febrile, obese, or toxic-appearing patients.
2. All patients warrant follow-up referral due to the high incidence of fistulae formation associated with anorectal abscesses.



FIGURE 9.42 ■ **Draining Perianal Abscess.** A draining, left perianal abscess in a febrile infant. (Photo contributor: Lily Yu, MS4.)



FIGURE 9.43 ■ **Perineal Abscess and Cellulitis.** A large perineal abscess is seen extending from the perianal tissues to the right labia in a female infant. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 9.44 ■ **Perirectal Abscess.** This large, spontaneously draining perirectal abscess required further surgical debridement in the operating room. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Hemorrhoids are classified according to their location and severity. External hemorrhoids result from the dilatation of the venules of the inferior hemorrhoidal plexus distal to the dentate line. They have a covering of skin, or anoderm. Internal hemorrhoids originate above the dentate line from the superior hemorrhoidal plexus and have a mucosal covering consistent with the surrounding internal anal canal. Internal hemorrhoids can be classified from first to fourth degree by their amount of prolapse and ease of reduction. Factors that contribute to hemorrhoid formation include constipation, family history, obesity, and increased intra-abdominal pressure from pregnancy or ascites. Hemorrhoids are commonly found at three anatomic locations: right anterior, right posterior, and left lateral positions (2-, 5-, and 9-o'clock positions). Internal hemorrhoids commonly present with painless, bright red rectal bleeding after defecation. This results from the passage of the fecal mass over the thin-walled venules, causing abrasions and bleeding. Patients may also complain of a sensation of anal fullness from prolapse of the vessel. Symptoms from external hemorrhoids can include swelling, burning, pruritus, and anal fullness. External hemorrhoids can also thrombose and result in exquisite pain in the acute phase. Other diagnoses to consider include infection, perianal or perirectal abscess, inflammatory bowel disease, malignancy,



FIGURE 9.45 ■ **External Hemorrhoids.** Multiple engorged, thrombosed (dark blue hue) external hemorrhoids are seen in this patient with anal pain and bleeding. (Photo contributor: Lawrence B. Stack, MD.)

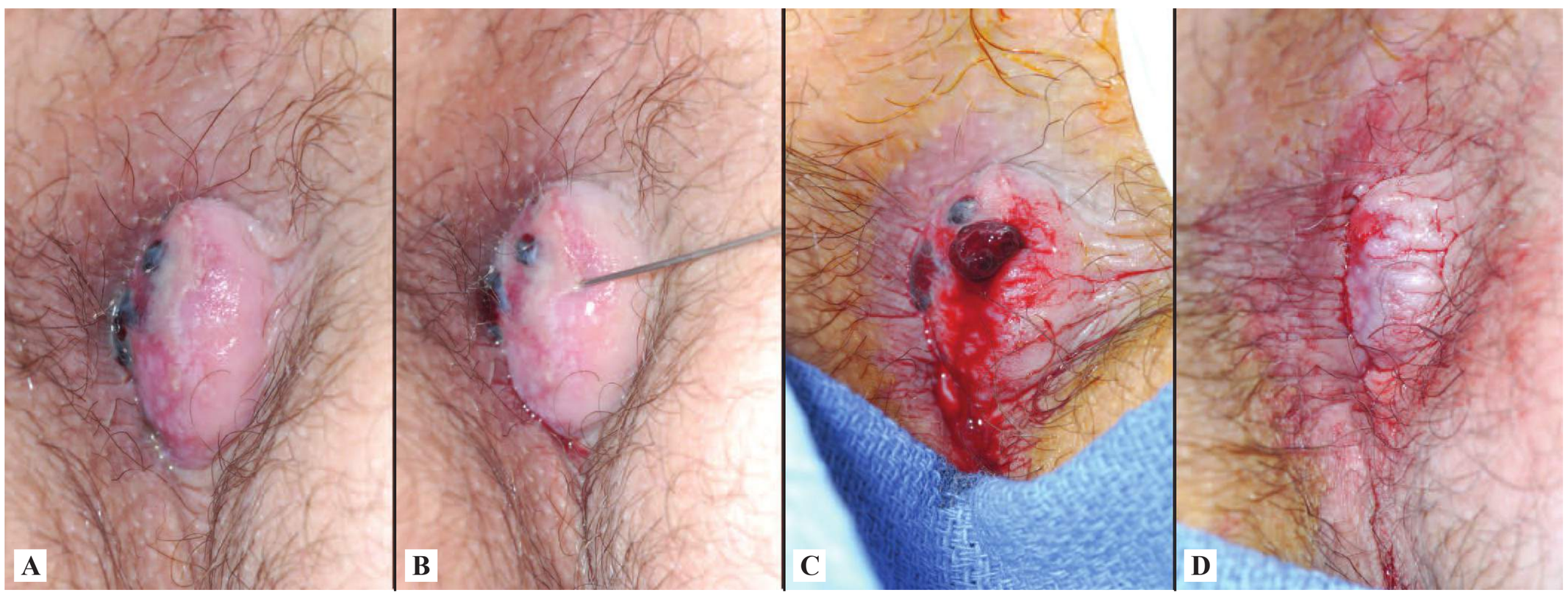


FIGURE 9.46 ■ **Evacuation of Thrombosed Hemorrhoid.** (**A**) Painful 1.5-cm thrombosed hemorrhoid. (**B**) Infiltration with marcaine till blanches. (**C**) Clot delivery after incision. (**D**) Collapsed hemorrhoid after clot evacuation. (Photo contributor: Lawrence B. Stack, MD.)

local trauma, herpes or other sexually transmitted infection, rectal polyp, or rectal prolapse.

Management and Disposition

In the rare case of severe, life-threatening bleeding, initiate fluid resuscitation and locate and clamp or ligate the bleeding vessel. Treatment for less severe cases includes increased dietary fiber, increased fluid intake, hot sitz baths, bed rest, and nonnarcotic pain medication. Advanced cases may require surgical consultation and treatment. Emergency department treatment of thrombosed external hemorrhoids includes an elliptical excision and extrusion of the clot under local anesthesia.

Pearls

1. Patients with other serious anorectal problems may complain of "hemorrhoids." Therefore, careful examination and consideration of the differential diagnosis should be undertaken with each patient.
2. Having the patient strain during the examination may reveal bleeding or prolapse of an internal hemorrhoid that might otherwise go unnoticed. Anoscopy can also be helpful in examination.
3. Thrombosed hemorrhoids should not be excised in pregnant, immunocompromised, or pediatric patients.



FIGURE 9.47 ■ **Bleeding External Thrombosed Hemorrhoid.** Engorged painful bleeding thrombosed hemorrhoids in a patient with chronic constipation. (Photo contributor: Department of Emergency Medicine, Naval Medical Center, Portsmouth, VA.)

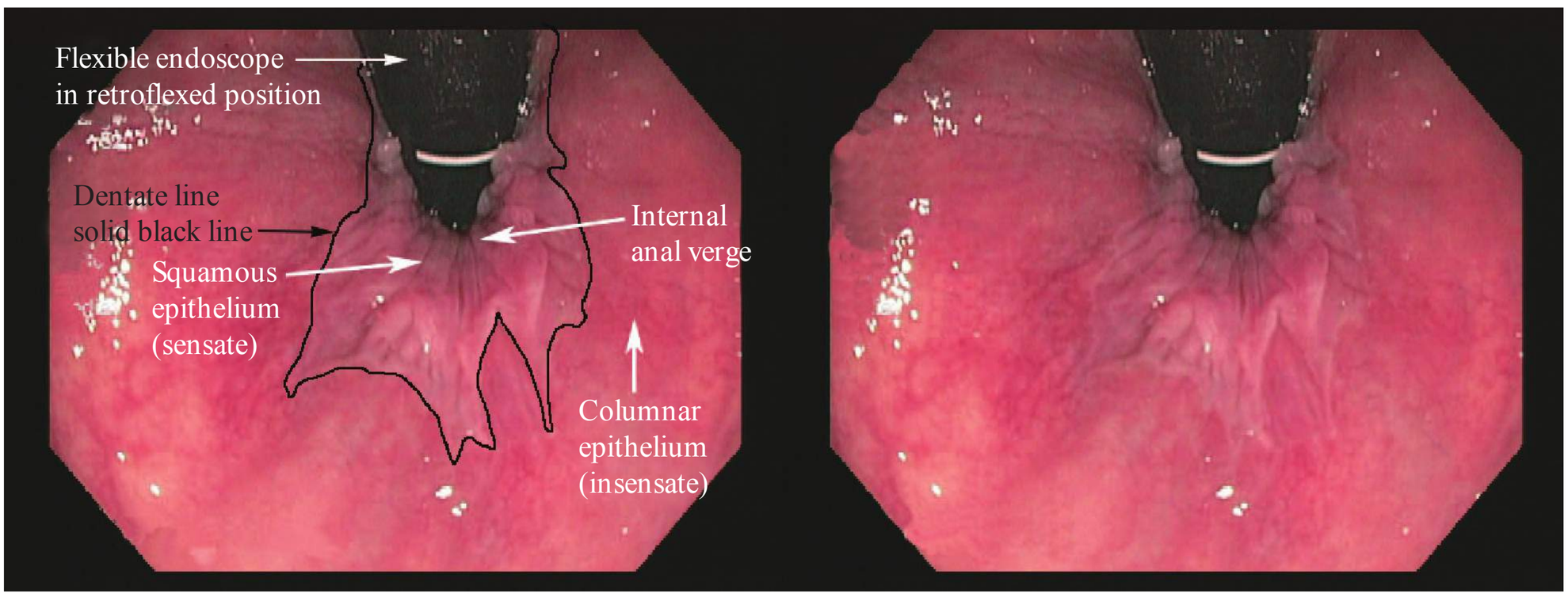


FIGURE 9.48 ■ **Normal Distal Rectum Surface Anatomy.** Columnar epithelium lines the colon (rectum), while squamous epithelium lines the anal canal and extends into the distal rectum. The columnar-squamous junction (interface) is termed the dentate line. The columnar epithelium is insensate, while the squamous epithelium is sensate. (Photo contributor: Philip E. Stack, MD.)

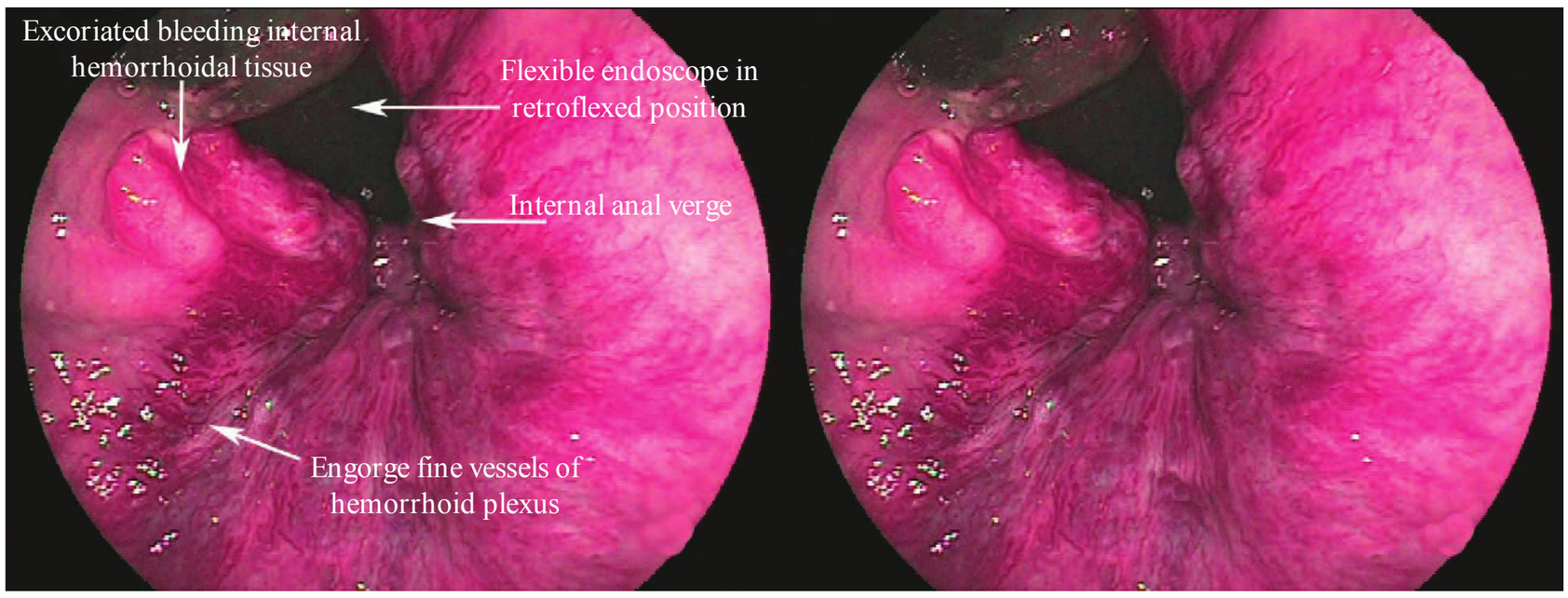


FIGURE 9.49 ■ **Bleeding Internal Hemorrhoids.** Middle-aged male with painless hematochezia. Internal hemorrhoidal bleeding is usually painless with a bleeding character that is described as "dripping or like a spray." Moderate hematochezia outside of anticoagulation rarely results in any hemodynamic compromise. Treatment of predisposing underlying condition(s)—constipation, diarrhea, straining, poor dietary fiber intake, prolonged sitting and lifting, etc—is paramount, otherwise hemorrhoids are likely to recur or become chronic. (Photo contributor: Philip E. Stack, MD.)

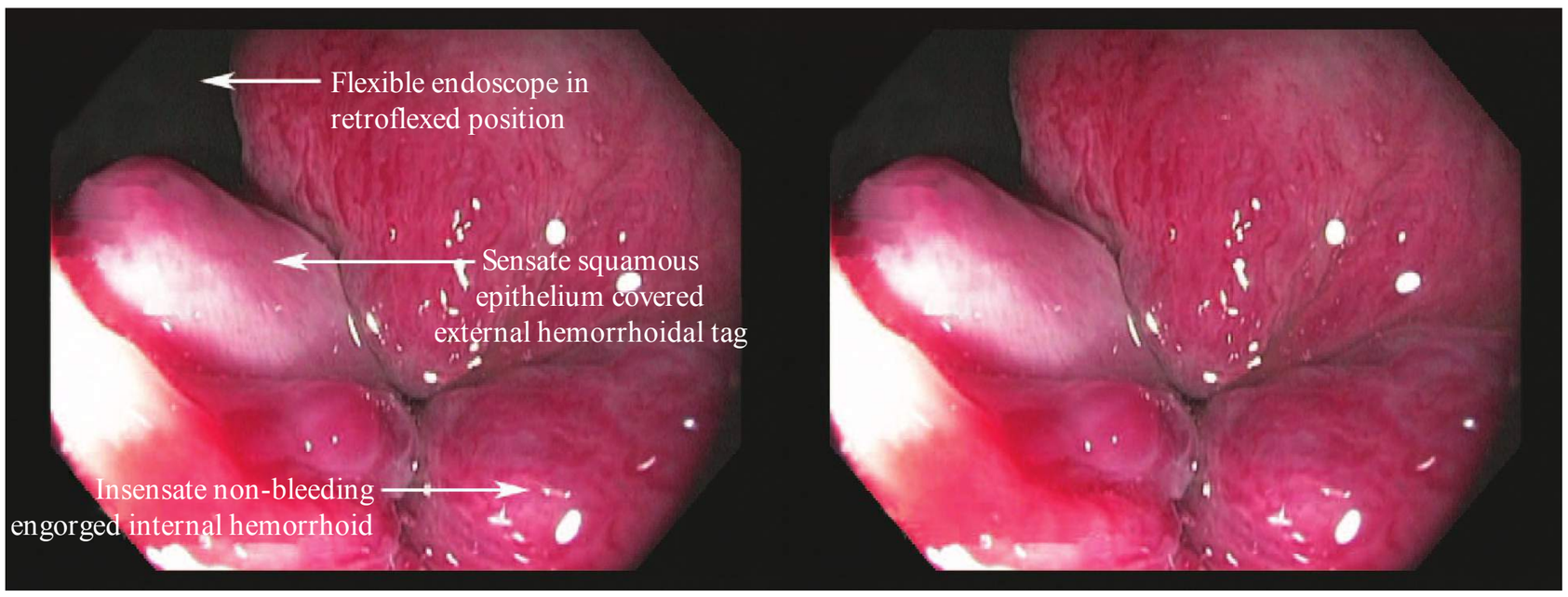


FIGURE 9.50 ■ **External and Internal Hemorrhoids.** Middle-aged woman with hemorrhoids and intermittent bloody stools and tender prolapsing small mass after bowel movements which she manually reduces. Acute engorgement of external hemorrhoids can result in residual redundant tissue that is prone to re-engorgement. This sequence can result in the formation of a "tag or tissue" of squamous epithelium which can be tender. This tissue, once formed, is not amenable to medical therapies and would require endoscopic or surgical removal if deemed necessary. (Photo contributor: Philip E. Stack, MD.)

PROLAPSED RECTUM

Clinical Summary

Rectal prolapse (or procidentia) occurs when anorectal tissue slides through the anal orifice. Prolapse may be partial, involving only the mucosa (prolapse is <2 cm), or complete, involving a full-thickness extrusion of the rectal wall. Prolapse may result from laxity of the pelvic floor, weak anal sphincters, and/or lack of mesorectal fixation. Patients are often at extremes of age and complain of bleeding, mucous discharge, rectal pressure, or a mass. Associated fecal incontinence, constipation, and rectal ulceration are common as well. Family history, cystic fibrosis, chronic cough, dysentery, or parasitic infections are conditions associated with increased risk of procidentia. Differential diagnoses to consider include foreign body, tumor, perianal or perirectal abscess, rectal polyp, or engorged external hemorrhoids.

Management and Disposition

Gentle manual pressure will reduce most rectal prolapses. If manual reductions fail, surgical consultation and operative reduction are needed. Surgical treatment is also indicated with a complete prolapse. Refer all patients for anoscopic and sigmoidoscopic examination to evaluate for tumor, polyps, or inflammatory bowel disease. If rectal bleeding is a problem, full colonic evaluation should be completed.

Pearls

1. This is commonly seen in children with cystic fibrosis (22%); therefore, all children with rectal prolapse should have a sweat chloride test.
2. Examination of rectal prolapse reveals concentric mucosal rings and a sulcus between the anal canal and the rectum. Prolapsed hemorrhoids are separated by radial grooves and the sulcus is absent.
3. To confirm the diagnosis, prolapse may be reproduced by having the patient bear down.

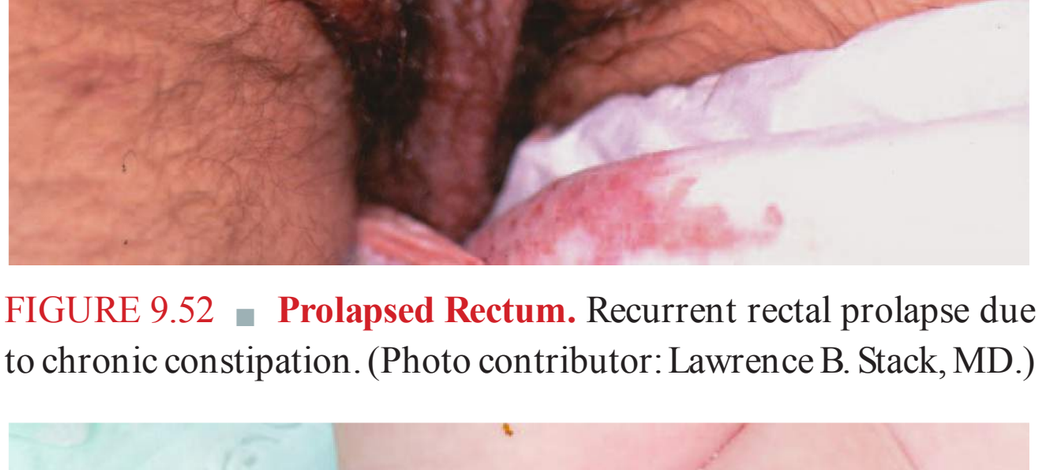


FIGURE 9.52 ■ **Prolapsed Rectum.** Recurrent rectal prolapse due to chronic constipation. (Photo contributor: Lawrence B. Stack, MD.)

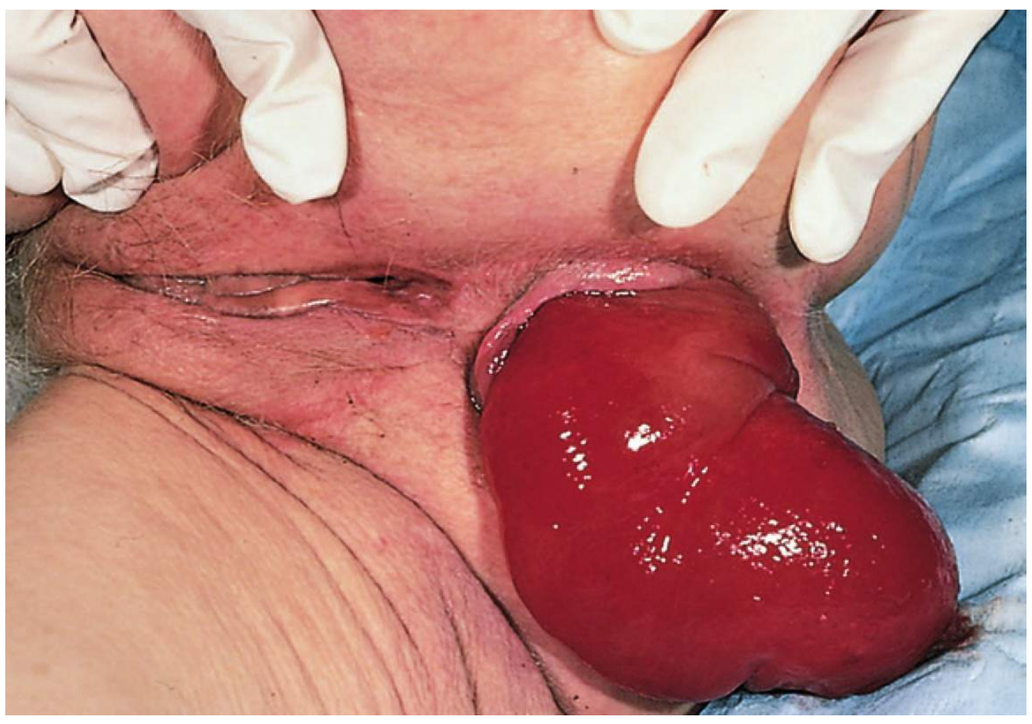


FIGURE 9.51 ■ **Prolapsed Rectum.** The rectum is completely prolapsed in this elderly patient. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 9.53 ■ **Prolapsed Rectum—Infant.** Two-month-old with rectal prolapse after repeated episodes of straining at stool. Rectal biopsy performed because of abnormal barium enema. Biopsy results negative for Hirschsprung disease. (Photo contributor: Lawrence B. Stack, MD.)

PILONIDAL ABSCESS

Clinical Summary

Pilonidal abscesses are typically seen at or just superior to the gluteal fold and are more common in teenage and young adult males. Patients complain of localized pain, swelling, and drainage but usually do not have systemic symptoms. The abscesses are thought to occur when bacteria enter the hair follicle and cause edema that obstructs the opening to the skin surface. Eventually the follicle may rupture which allows spread into the subcutaneous fatty tissue and abscess formation. The acute abscess contains mixed organisms including Staphylococcus aureus and Streptococcus spp., but anaerobes and gram-negative organisms may also be present. Evidence of cellulitis in the sacrococcygeal area may result from a simple abscess or furuncle. Differential diagnosis includes anal fistulae, hidradenitis, inflammatory bowel disease, or tuberculosis.

Management and Disposition

An acutely fluctuant abscess requires incision and drainage under local anesthesia with removal of pus and debris. Instruct the patient on meticulous wound care and sitz baths. Antibiotic therapy is indicated only in immunocompromised patients or those with significant surrounding cellulitis or systemic symptoms. Pilonidal abscesses have a 40% recurrence rate. Refer chronic or recurrent cases to a surgeon for consideration of definitive treatment by surgical excision and closure.

Pearls

1. Pilonidal abscesses almost always occur in the midline but can have sinus tracts extending off the midline.
2. Pilonidal disease is three times more common in men than in women.



FIGURE 9.54 ■ **Pilonidal Abscess.** Redness, fluctuance, and tenderness in the gluteal cleft seen with a pilonidal abscess. (Photo contributor: Louis La Vopa, MD.)



FIGURE 9.55 ■ **Pilonidal Abscess.** Close-up of the cutaneous manifestations of a large pilonidal abscess. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

The diagnosis of rectal foreign body is usually made by history and confirmed by digital examination. Most often the foreign body is inserted, but it is possible to have an ingested foreign body trapped in the rectum. The most common and serious complication of a rectal foreign body is perforation of the rectum or distal colon. The patient must be carefully evaluated for evidence of perforation which may include physical examination findings consistent with an acute abdomen or x-rays demonstrating free air. Perforation above the peritoneal reflection is associated with free air in the abdominal cavity and peritoneal signs. Perforation below the peritoneal reflection often causes retroperitoneal injuries and presents with more insidious signs of pain and infection in the perianal or perineal region. It is important to determine the size, shape, and number of objects to assess the risk of perforation. In children, rectal foreign bodies usually present as rectal bleeding. Depending on the clinical scenario, the diagnoses of sexual assault or child abuse should be considered.

Management and Disposition

Removal can often take place in the emergency department with sedation of the patient and local anesthesia of the anal sphincter. If the risk of perforation appears high or adequate relaxation and anesthesia cannot be obtained, then removal should occur under general anesthesia. After removal, x-ray studies and proctoscopic or sigmoidoscopic examination are recommended to rule out perforation or laceration.

Pearls

1. A Foley catheter or an endotracheal tube may be passed proximal to some foreign bodies. This may release the vacuum effect of the rectum or the balloon may be inflated to aid in removal.
2. A rectal foreign body in a child should raise the suspicion of abuse.



FIGURE 9.56 ■ **Rectal Foreign Body.** This foreign body (a 7-oz beer bottle) required removal in the operating room. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 9.57 ■ **Rectal Foreign Body.** This victim of assault had a stick forced into his rectum. After removal, sigmoidoscopy revealed a retained small broken stick. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 9.58 ■ **Rectal Foreign Body.** Ventriculo-peritoneal shunt tubing seen protruding from the anus after eroding through the transverse colon. (Photo contributor: Lawrence B. Stack, MD.)

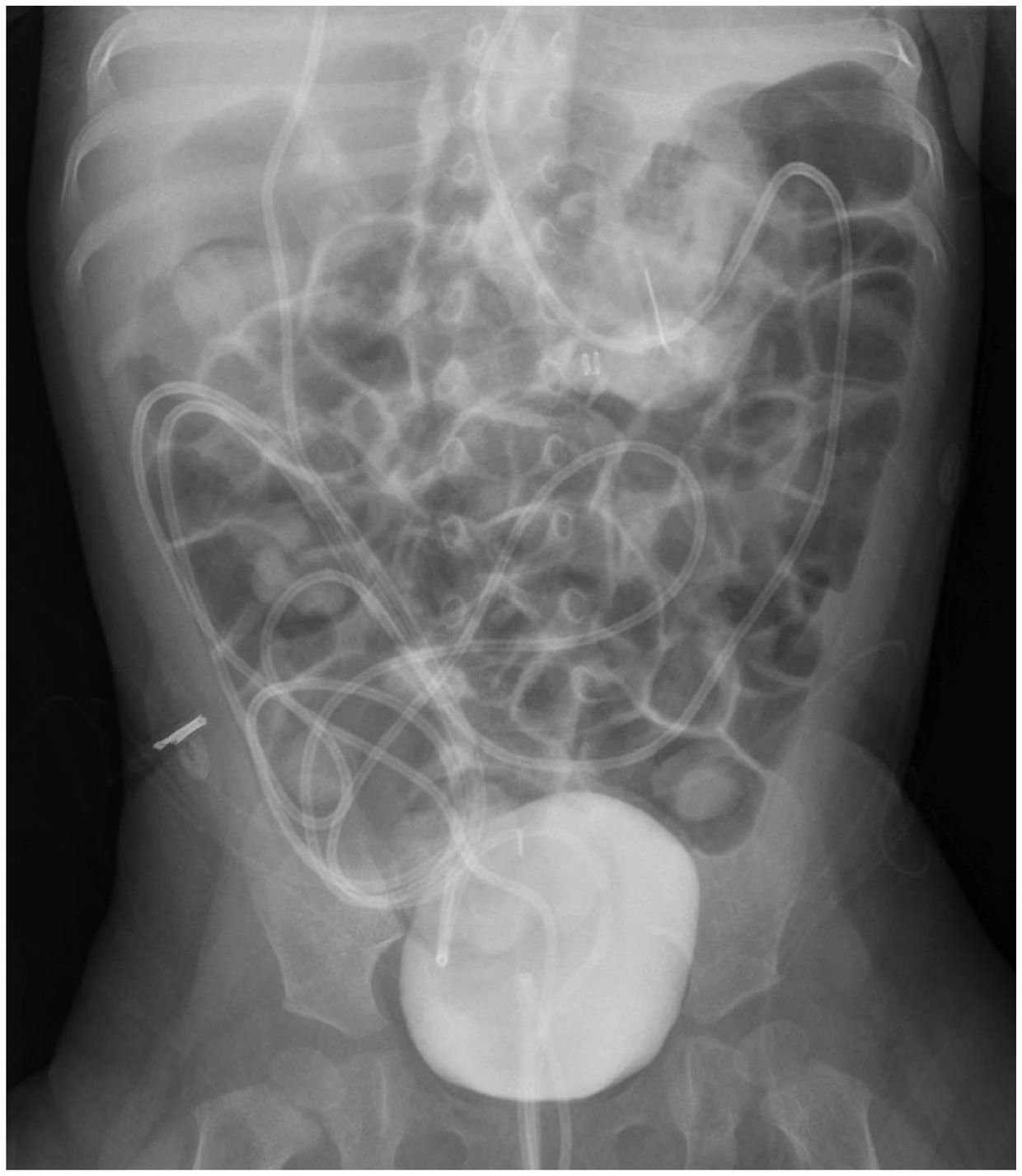


FIGURE 9.59 ■ **Ventriculo-Peritoneal Shunt Tubing in Colon.** Ventriculo-peritoneal shunt tubing seen in the transverse colon and proceeding down the distal colon and exiting the anus. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Gastrointestinal bleeding commonly presents with the alteration of stool color. Melena is defined as the passage of dark, pitchlike stools stained with blood pigments. Melena typically results from bleeding into the upper gastrointestinal tract proximal to the ligament of Treitz. Black stools have been seen with as little as 60 mL of blood in the upper gastrointestinal tract, but melena typically does not develop until 100 to 200 mL is present. Melena can be found in lower bleeds with decreased transit time, as with an obstruction distal to the site of bleeding. Melenic stools may occur from swallowed blood, as from epistaxis or other oropharyngeal bleeding. Dark or black stools can also be seen with the ingestion of bismuth salicylate, food coloring, and iron supplements.

Management and Disposition

Patients with true melena should be initially treated as unstable until orthostatic vitals signs, stool guaiac, and a hematocrit have been obtained. One or more large-bore intravenous line should be placed and saline infused. Depending on the patient's stability, type-specific packed red blood cells or other blood products may be required. Stable patients who present with melena may be admitted to the ward. Patients with unstable vital signs, continued bleeding, severe anemia, or comorbid disease are admitted to the intensive care unit with gastroenterology consultation. Obtain surgical consultation in patients with abdominal free air or those who remain unstable after receiving blood products.

Pearls

1. Melena is the most common presenting symptom of bleeding from peptic ulcer disease.
2. Melena represents approximately 200 mL of blood loss in the gastrointestinal tract.



FIGURE 9.61 ■ **Melena.** Guaiac-positive stool specimen in a patient with melena due to a duodenal ulcer. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 9.60 ■ **Melena.** The black tarry appearance of melena in a patient with a duodenal ulcer. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 9.62 ■ **Melena and Maroon Stool.** The maroon watery stool with more solid melena suggests a briskly bleeding upper gastrointestinal bleed. Esophagogastroduodenoscopy confirmed a bleeding gastric ulcer. (Photo contributor: Maria Ladino.)

Chapter 10

GYNECOLOGIC AND OBSTETRIC CONDITIONS

Scan QR code or go to www.ematlas.com for video related to chapter

Suzanne Dooley-Hash
Sean A. Lisse
Kevin J. Knoop



Nuchal Cord. Hand placement prior to applying downward traction to deliver the anterior shoulder. A loose nuchal cord is seen. (Photo contributor: William Leininger, MD.)

The authors acknowledge the special contributions of Robert G. Buckley, MD, for contributions to prior editions.

Clinical Summary

Candidal vaginitis is characterized by a thick, clumping, white discharge and vulvar discomfort. Intense vulvar erythema, pruritus, and/or burning are often present. Predisposing factors may include oral contraceptive, antibiotic, or corticosteroid use; pregnancy; and diabetes. A microscopic slide prepared with 10% potassium hydroxide yielding characteristic branched chain hyphae and spores establishes the diagnosis. Sexually transmitted diseases are not usually associated with isolated Candidal vaginitis.

Trichomonas vaginitis presents as a persistent, thin, copious discharge that is often frothy, green, or foul-smelling. The amount of vaginal and cervical erythema and inflammation varies considerably. Diagnosis may be made by the presence of motile flagellates on normal saline wet-mount microscopy, but this is only 60% to 70% sensitive. New nucleic acid tests are emerging with ~95% specificity. Multiple petechiae on the vaginal wall or cervix (strawberry spots/strawberry cervix) are pathognomonic but only occasionally seen.

Bacterial vaginosis is characterized by a malodorous, homogeneous discharge with an amine (fishy) odor that can be briefly accentuated by mixing with a drop of KOH solution. The presence of clue cells on normal saline wet mount establishes the diagnosis. Other associated vaginal or abdominal complaints are minimal and, if significant, suggest the presence of another disease process.

Although the above infectious causes are responsible for most cases of vaginitis, other possible etiologies including local chemical irritants or allergens, vaginal foreign bodies, and atrophic vaginitis should be considered in the differential diagnosis.

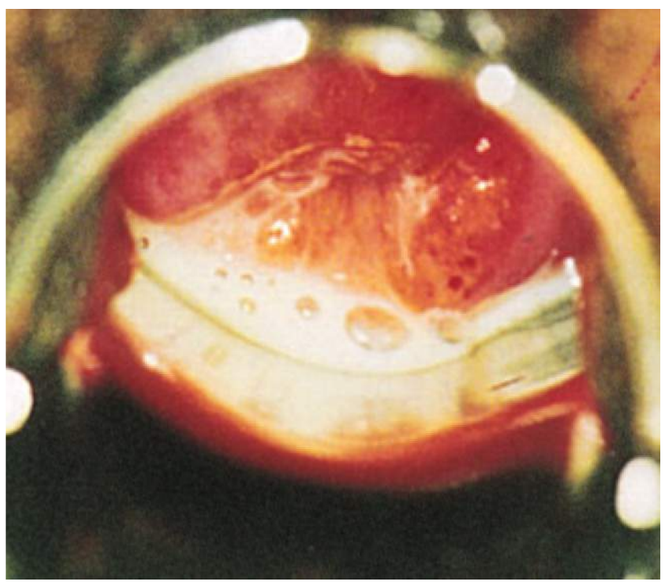


FIGURE 10.2 ■ Trichomonas **Vaginitis.** Thin vaginal discharge suggestive of trichomonas vaginitis. (Photo contributor: H. Hunter Handsfield. Atlas of Sexually Transmitted Diseases. New York: McGraw-Hill; 1992.)

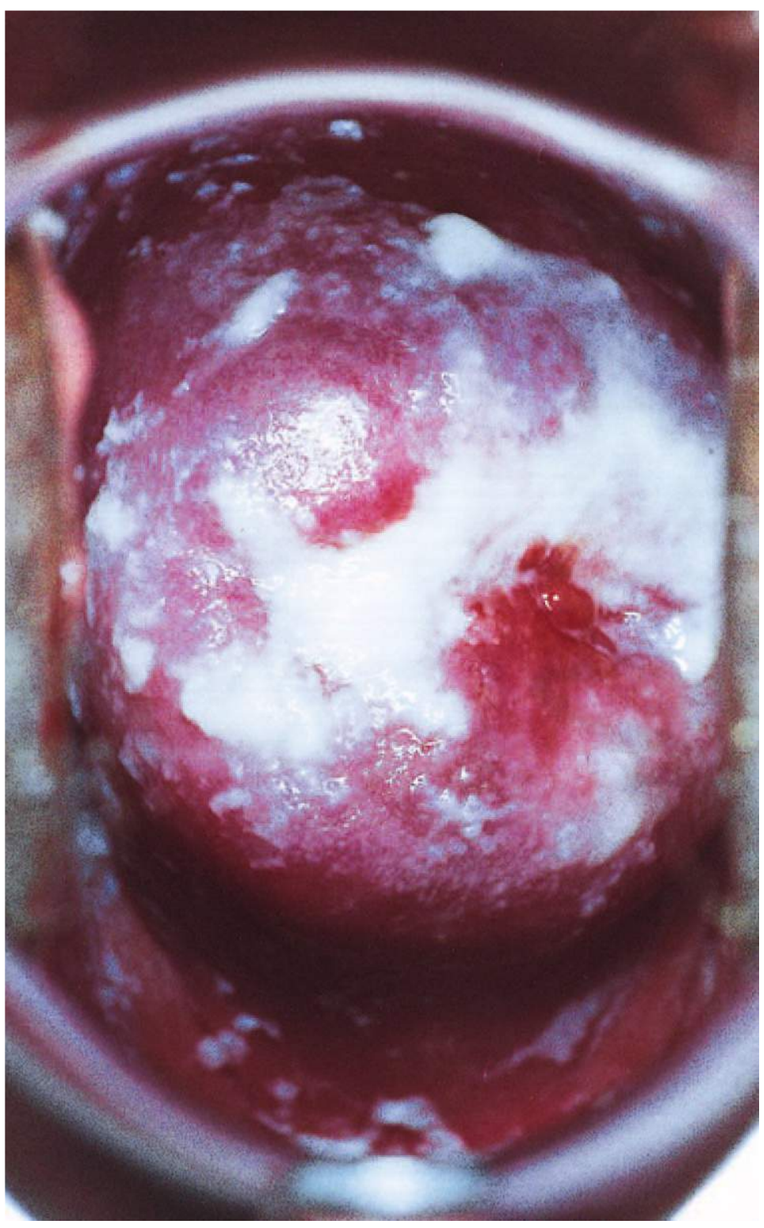


FIGURE 10.1 ■ **Candidal Vaginitis.** Thick, curdy white discharge secondary to candidal vaginitis. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 10.3 ■ Gardnerella **Vaginosis.** Thin, milky white discharge suggestive of Gardnerella vaginosis. (Photo contributor: Curatek Pharmaceuticals.)

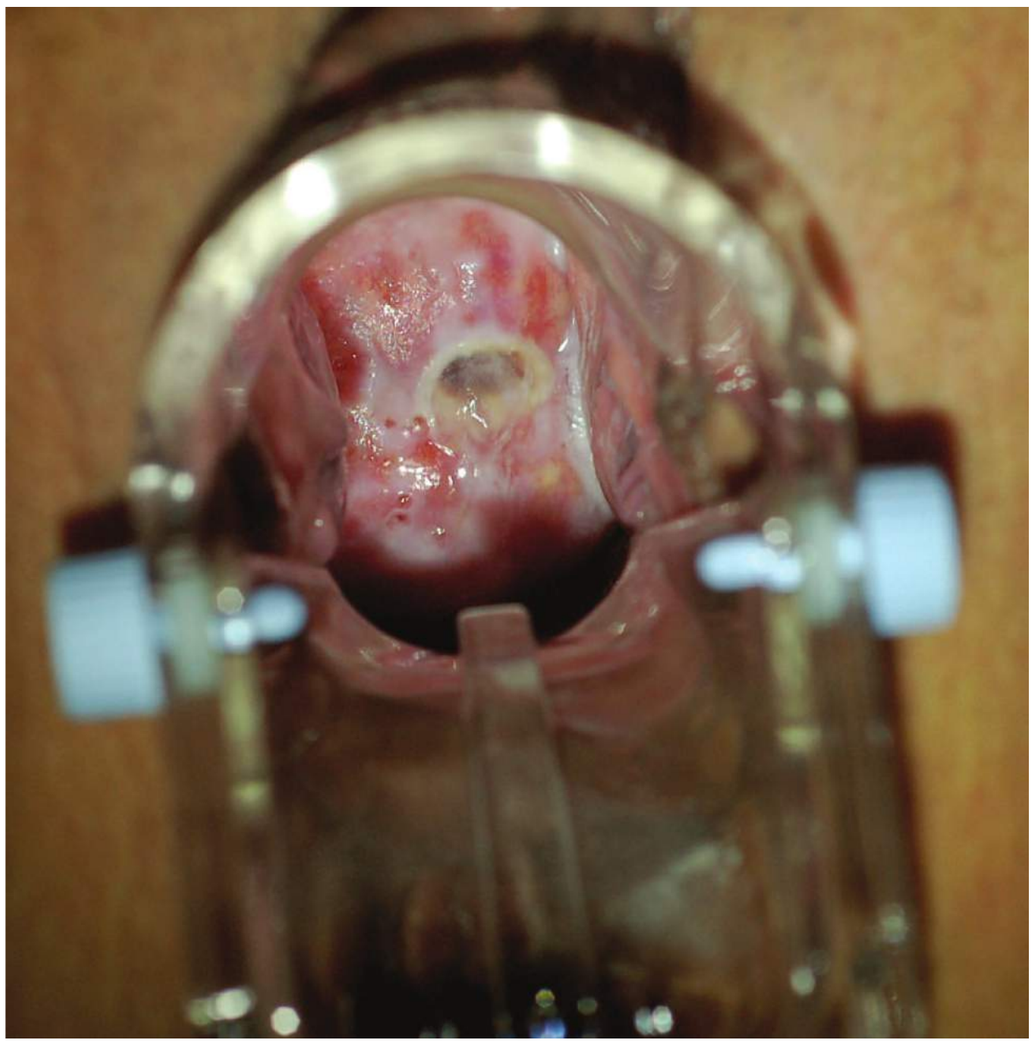


FIGURE 10.4 ■ **Bacterial Vaginosis.** Thin, milky white discharge suggestive of bacterial vaginosis. (Photo contributor: David Effron, MD.)

Management and Disposition

For Candidal vaginitis, treat with topical antifungals (eg, clotrimazole: 1% cream, 5 g for 7 to 14 days or two 100-mg vaginal tablets for 3 nights). Oral fluconazole (150 mg as a single dose) is also effective, but has a higher risk of adverse effects.

For Trichomonas vaginitis, a single dose of metronidazole (2 g orally) is generally curative but is associated with a disulfiram-like reaction when taken with alcohol. Although metronidazole was previously considered contraindicated in pregnancy, the Centers for Disease Control and Prevention (CDC) now recommends its use due to the association of Trichomonas infections with preterm rupture of membranes, low birth weight, and increased risk of prematurity.

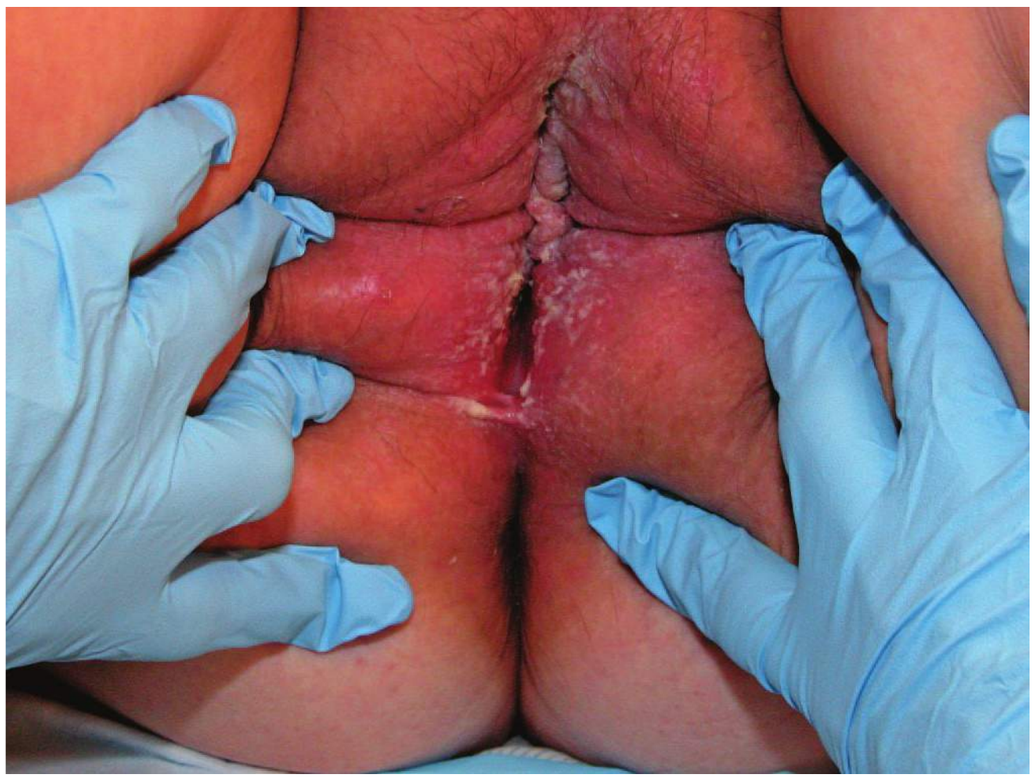


FIGURE 10.5 ■ **Candida Vulvovaginitis.** Intense vulvar erythema with thick white clumping discharge consistent with candida vulvovaginitis. (Photo contributor: David Effron, MD.)

For bacterial vaginosis, metronidazole (500 mg orally twice daily for 7 days) is recommended. Treatment for asymptomatic infection or for male sexual partners is not generally recommended. The equivocal risk of metronidazole teratogenicity must be weighed against the likelihood of alleviating symptoms and discomfort.

Pearls

1. Diabetes mellitus or immunosuppression should be considered in refractory or recurrent cases of candidal vaginitis.
2. A history of balanitis in the sexual partner should be sought and treated if present.
3. Trichomonas should be considered a sexually transmitted infection. Therefore, it is generally recommended that concomitant culturing for Neisseria gonorrhoeae and Chlamydia be performed. Serologic testing for syphilis, HIV, and hepatitis B should also be considered for patient and partner.

TABLE 10.1 ■ CHARACTERISTICS OF INFECTIOUS VAGINITIS

Disease	Discharge Qualities	Vaginal pH	Microscopic Findings
Candida spp.	White, thick, clumping	<4.5	Hyphae and spores in KOH
Trichomonas vaginitis	Green, thin, frothy, foul	>4.5	Flagellates in wet mount
Bacterial vaginosis	Thin, white, “fishy” odor in KOH	>4.5	Clue cells

CERVICAL POLYPS

Clinical Summary

Cervical polyps are friable, fleshy, fingerlike growths that emanate from the cervical os or endocervical canal. They are typically asymptomatic, but may bleed with minimal trauma such as intercourse or douching. Single polyps are more common, but multiple polyps can occur. The etiology of polyps varies and may be related to infection, chronic inflammation, or excess estrogen.

Management and Disposition

No treatment is necessary in the emergency department (ED). Consider alternative causes of vaginal bleeding such as infection and pregnancy. Refer all patients with incidentally discovered polyps for outpatient gynecological evaluation.

Pearls

1. The majority of cervical polyps are benign, but all should be referred for ligation and histologic evaluation.
2. Cervical polyps are the most common benign neoplasm of the cervix.

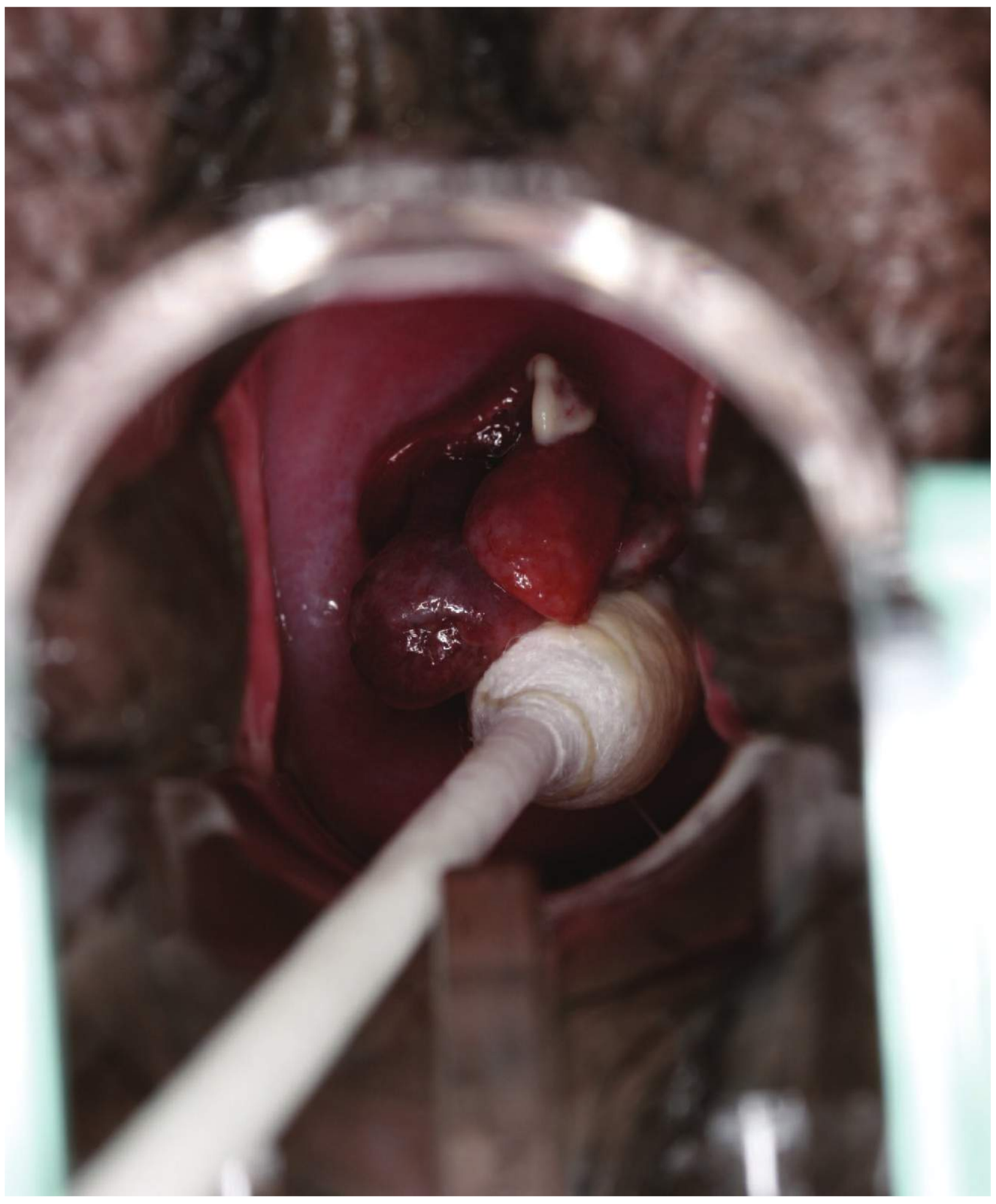


FIGURE 10.6 ■ **Cervical Polyps.** Several fleshy fingerlike growths are seen protruding from the cervical os. (Photo contributor: Kevin J. Knoop, MD, MS.)

BARTHOLIN GLAND ABSCESS

Clinical Summary

Bartholin glands are located on either side of the lower third of the vaginal introitus near the labia minora. A cyst or abscess may result from an obstructed duct, often secondary to trauma or inflammation. Infection of the cyst is usually with mixed vaginal or fecal flora (Escherichia coli) but may also contain N gonorrhoeae and Chlamydia trachomatis. Cysts or abscesses may be asymptomatic or may lead to increasing pain, swelling, and dyspareunia. A tender, fluctuant cystic mass with surrounding labial edema is easily appreciated on examination. Differential should include epidermal inclusion cysts and sebaceous cysts of the labia majora, hidradenitis suppurativa, vulvar hematomas, leiomyomas, lipomas, and fibromas.

Management and Disposition

Simple incision and drainage followed by sitz baths is the most effective immediate treatment, but recurrence of cysts is common with this method. Placement of a Word catheter into the cyst cavity decreases the incidence of reocclusion. The catheter, however, must remain in place up to 6 weeks to ensure epithelialization.

Pearls

1. Antibiotics are usually not required.
2. Incise the internal (medial) surface of the cyst or abscess rather than the external (lateral) aspect, as the incision site may become the new drainage tract.
3. Refer recurrent abscesses to gynecologist for definitive treatment which may involve marsupialization of the gland.



FIGURE 10.7 ■ **Bartholin Gland Cyst.** Bartholin gland cyst that is not infected. The labia minoris lays over the middle of the cyst. (Photo contributor: Hope Haefner, MD.)



FIGURE 10.8 ■ **Bartholin Gland Abscess.** Bartholin gland abscess with the labial fluctuance pointing medially. (Photo contributor: Medical Photography Department, Naval Medical Center, San Diego, CA.)

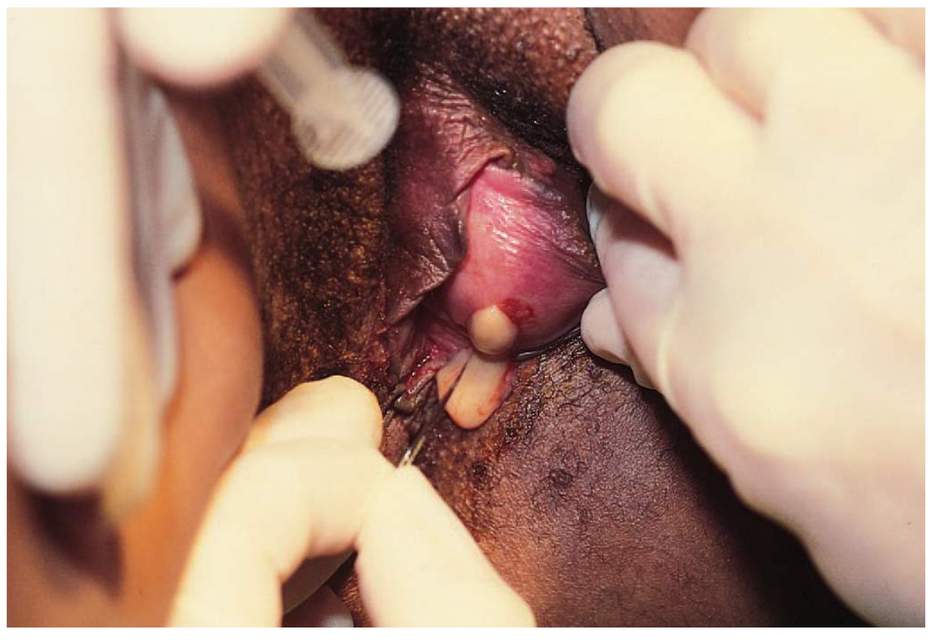


FIGURE 10.9 ■ **Bartholin Gland Abscess.** Medial incision of the cyst yielding purulent fluid, consistent with a Bartholin gland abscess. (Photo contributor: Medical Photography Department, Naval Medical Center, San Diego, CA.)

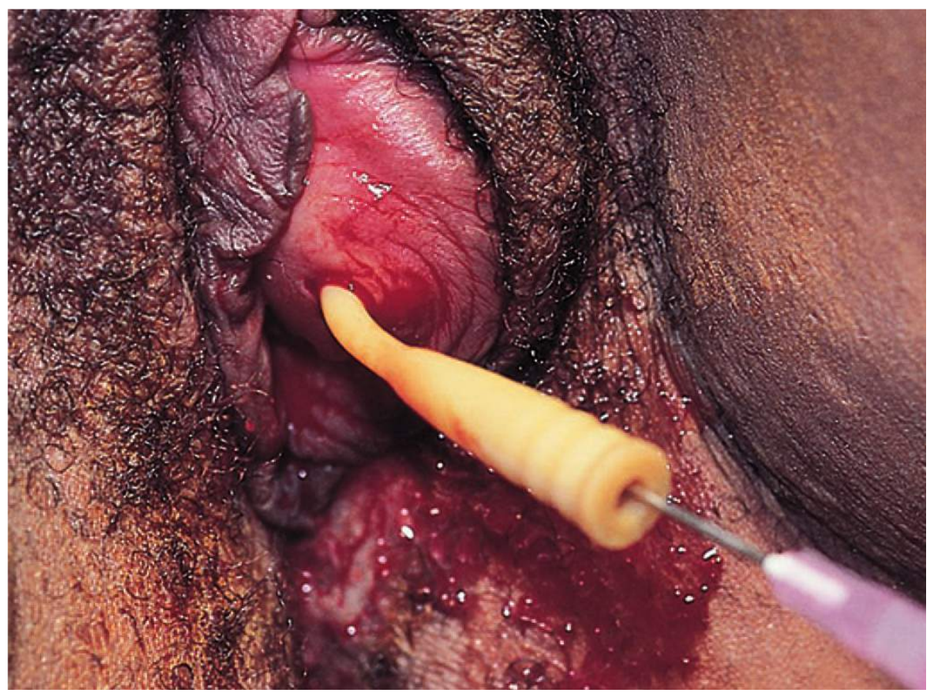


FIGURE 10.10 ■ **Bartholin Gland Abscess.** Insertion and inflation of a Word catheter into the cyst cavity. The free end of the catheter can be tucked into the vagina for long-term placement, allowing for epithelialization of the incision site. (Photo contributor: Medical Photography Department, Naval Medical Center, San Diego, CA.)

SPONTANEOUS ABORTION

Clinical Summary

Spontaneous abortion is associated with vaginal bleeding and abdominal discomfort. Severe pain, heavy bleeding, passage of clots or tissue, and hypotension may also be present. Mild cramping and vaginal bleeding that are not accompanied by passage of tissue or cervical dilation constitutes a threatened abortion. Uterine cramping with progressive cervical dilation indicates an inevitable abortion. An incomplete abortion is diagnosed when some products of conception (POC) have passed, while other retained intrauterine tissue leads to ongoing symptoms. Fever, leukocytosis, pelvic tenderness, and malodorous cervical discharge suggest a septic abortion. Completed abortion is characterized by the passage of confirmed POC, followed by resolution of bleeding and closure of the cervical os. Large blood clots or intrauterine decidual casts may be mistaken for POC and their presence cannot be used to rule out ectopic pregnancy.

Management and Disposition

Immediately obtain large-bore intravenous access and institute aggressive fluid resuscitation for any patient with severe pain, heavy bleeding, or hypovolemia. Also request cross-matched blood and urgent gynecologic consultation. Stable patients can undergo routine ED evaluation with complete blood count (CBC), human chorionic gonadotrophin (hCG), pelvic exam, and ultrasound (US). Send all identified tissue to pathology for definitive identification. Ectopic pregnancy must be ruled out by US, close clinical follow-up, and serial hCG testing.

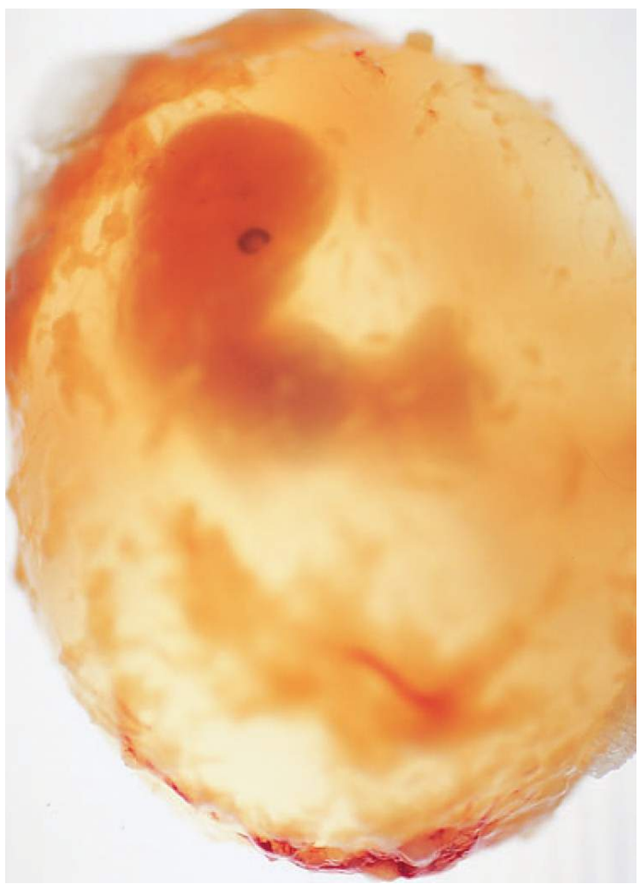


FIGURE 10.11 ■ **Spontaneous Abortion.** Passage of tissue in a spontaneous abortion at 4 weeks. (Photo contributor: Lawrence B. Stack, MD.)

Administer anti-Rh immunoglobulin (RhoGAM) in all cases of vaginal bleeding where the mother is Rh-negative.

Pearls

1. The passage of large clots usually indicates rapid heavy bleeding.
2. Heterotopic pregnancy is rare, but should be considered in patients with significant ongoing symptoms despite identification of a viable early intrauterine pregnancy (IUP).
3. Septic abortion may extend to the surrounding tissues or blood, with the potential for septic shock, ARDS, DIC, and group A strep induced toxic shock syndrome.

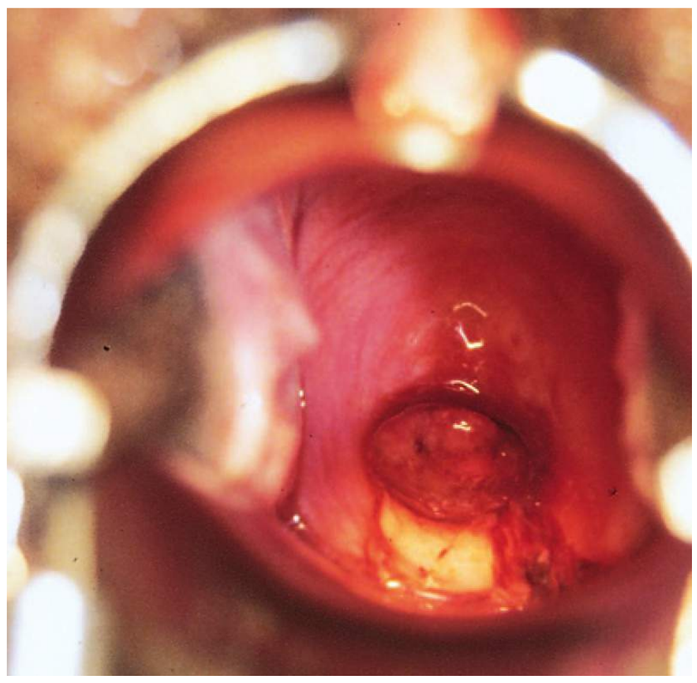


FIGURE 10.12 ■ **Spontaneous Abortion.** Dilation of the cervical os with partial extrusion of tissue in the setting of an inevitable abortion. (Photo contributor: Robert Buckley, MD.)



FIGURE 10.13 ■ **Decidual Cast.** A decidual cast or organized clot may occasionally be mistaken for products of conception. (Photo contributor: Medical Photography Department, Naval Medical Center, San Diego, CA.)

Clinical Summary

In cases of sexual assault, immediate care for the acute medical and psychosocial needs of the patient must happen in concert with forensic and legal requirements. Ideally, this care is provided by a specially trained sexual assault examiner. Regardless of who performs the examination, the provider must be very thorough in collecting and preserving all potential evidence and carefully documenting any physical findings. Chain of custody must be established and maintained for all samples collected in order for them to be used in later legal proceedings. Meticulous inspection of the genital structures in addition to a thorough general physical examination may reveal injuries to the perineum, rectum, vaginal fornices, vagina, and cervix as well as associated injuries to other areas of the body. Toluidine staining and colposcopy are useful in enhancing less apparent injuries such as those to the posterior fourchette and perianal area. Tears to the posterior fourchette are most commonly found in the distribution between the 3- and 9-o'clock positions when the patient is examined in dorsal lithotomy. Perianal lacerations are more evident with toluidine staining and appear as linear tears. Perineal injuries from accidental trauma may be indistinguishable from those of sexual assault and should be interpreted in the context of the history.

Management and Disposition

Treatment other than pain relief is preceded by forensic evidence gathering, consisting of a Wood lamp examination to identify semen for collection, pubic hair sampling and combing, vaginal and cervical smears (air-dried), a cervical and

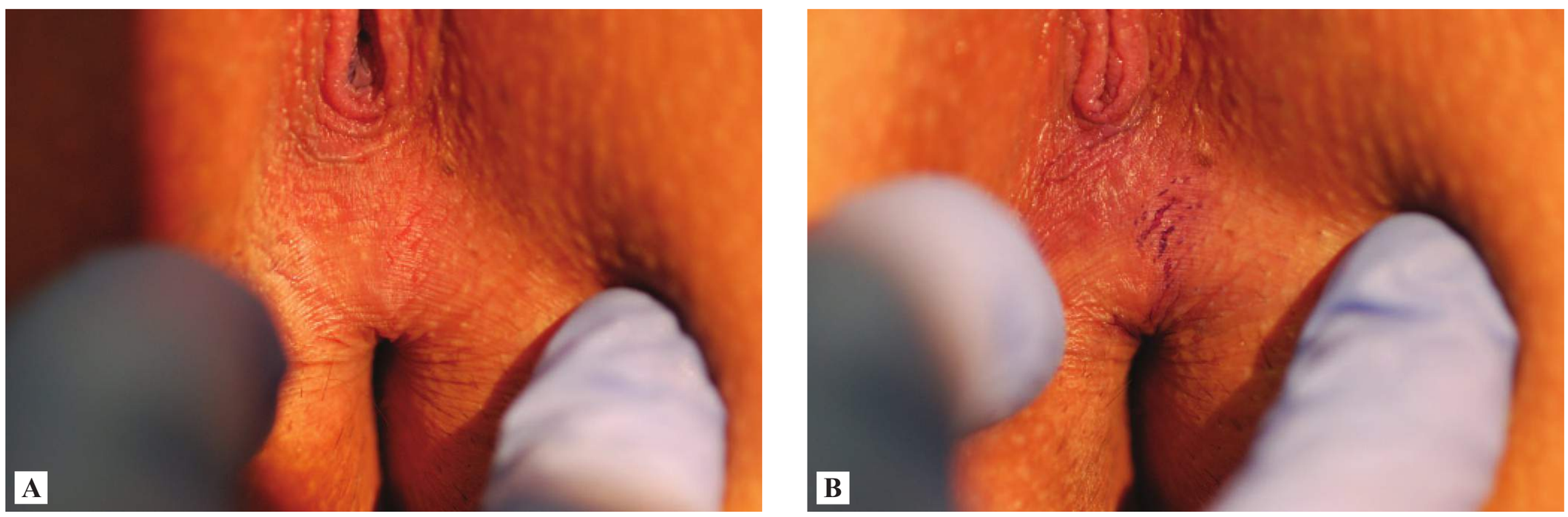


FIGURE 10.14 ■ **Genital Trauma (Posterior Fourchette).** (**A**) Linear tears of the posterior fourchette consistent with sexual assault. (**B**) Findings enhanced by toluidine blue staining. (Photo contributors: Hillary J. Larkin, PA-C, and Josua Luftig, PA-C.)

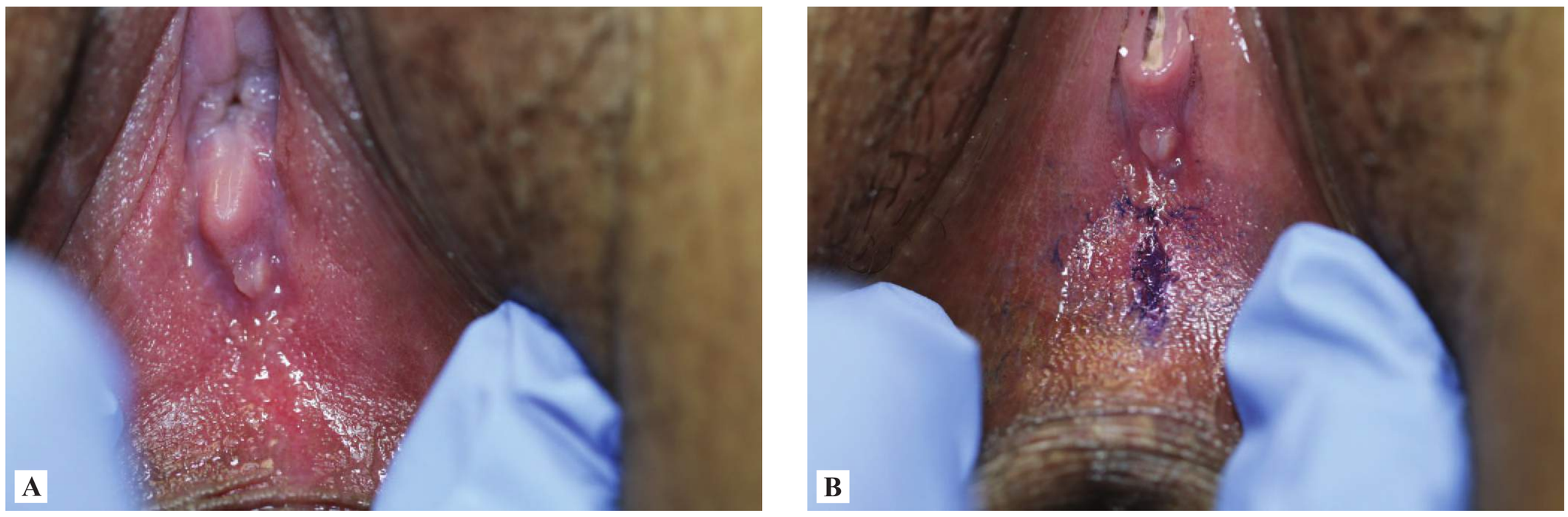


FIGURE 10.15 ■ **Genital Trauma (Posterior Fourchette).** (**A**) Linear midline tear of posterior fourchette. (**B**) Findings enhanced by toluidine blue staining. (Photo contributors: Hillary J. Larkin, PA-C, and Josua Luftig, PA-C.)

vaginal wet mount to identify sperm, vaginal aspirate to test for acid phosphatase, and rectal or buccal swabs for sperm. A prepackaged kit with directions may be available to facilitate the collection of evidence. Consult your state's laws for requirements.

Obtain cervical cultures for Chlamydia and N gonorrhoeae as well as serum testing for syphilis, hepatitis, and HIV. Provide empiric antibiotic coverage against sexually transmitted diseases and offer an oral contraceptive to prevent unwanted pregnancy after confirming a nonpregnant state.

Pearls

1. Victims of sexual assault may initially be reluctant to disclose their history and often present with an unrelated chief complaint. A thorough sexual history is necessary in order to properly identify and treat them.
2. The medical care of the patient who has been sexually assaulted is ideally performed by experienced supportive staff familiar with the details of forensic evidence gathering and colposcopic photography.
3. Normal physical examination and lack of sperm on wet preparation do not exclude the possibility of assault.

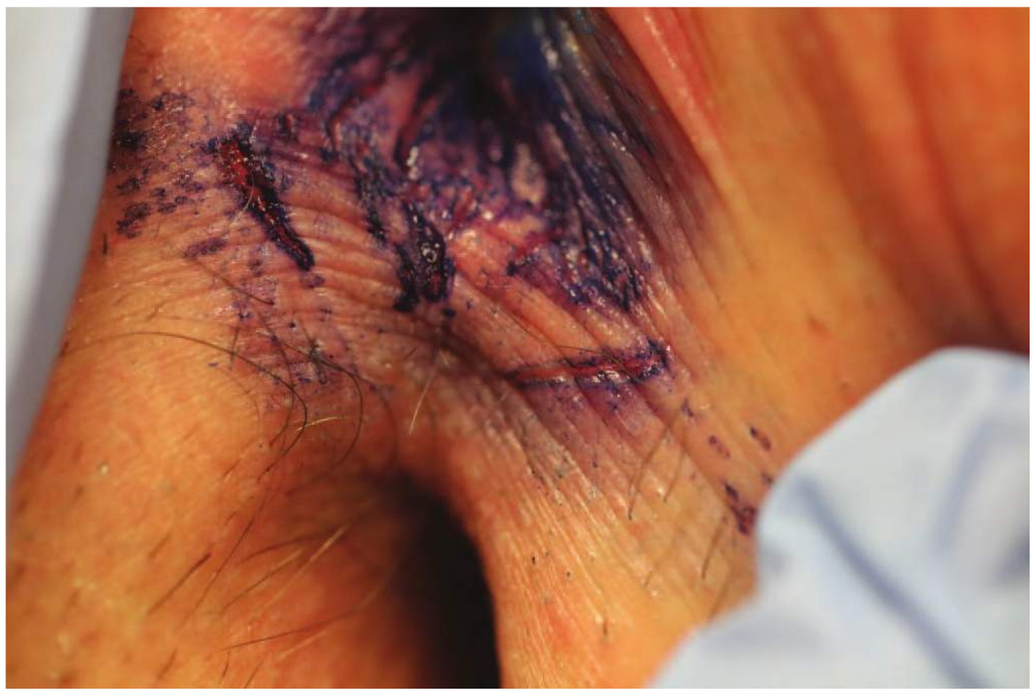


FIGURE 10.16 ■ **Genital Trauma (Posterior Fourchette and Perineum).** Multiple linear tears of posterior fourchette and perineum enhanced with toluidine blue stain. (Photo contributors: Hillary J. Larkin, PA-C, and Josua Luftig, PA-C.)

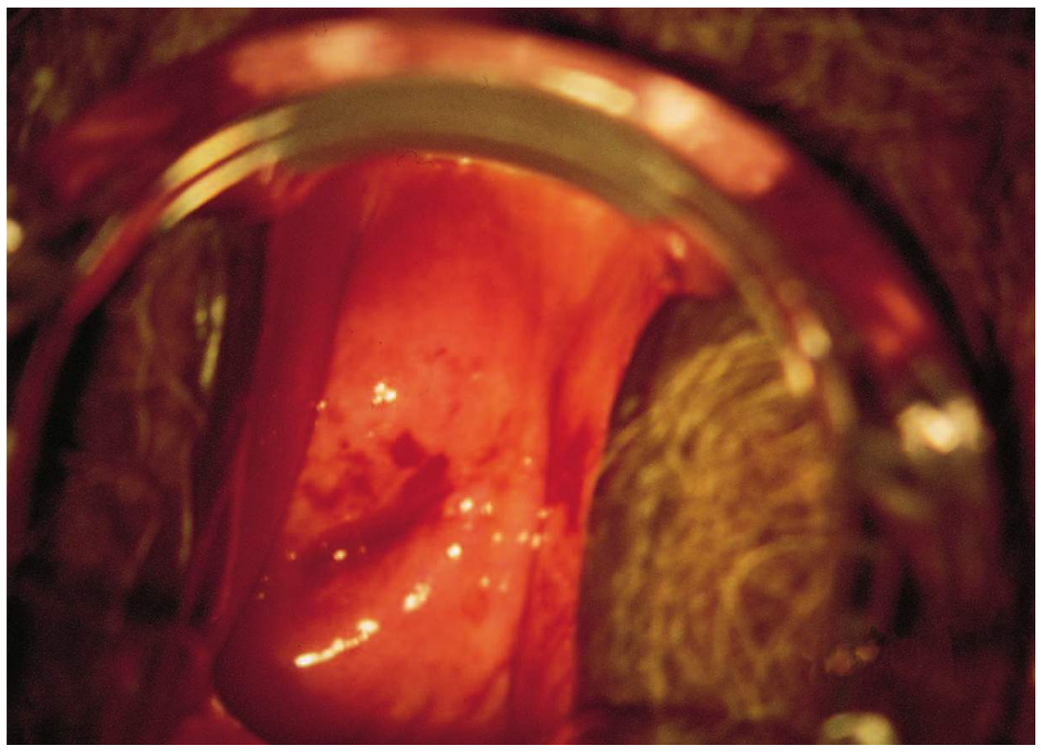


FIGURE 10.17 ■ **Genital Trauma (Cervix).** Cervical trauma in an elderly victim of sexual assault. Petechiae and freshly bleeding abrasions are noted from 10- to 3-o'clock position. (Photo contributors: Hillary J. Larkin, PA-C, and Lauri A. Paolinetti, PA-C.)

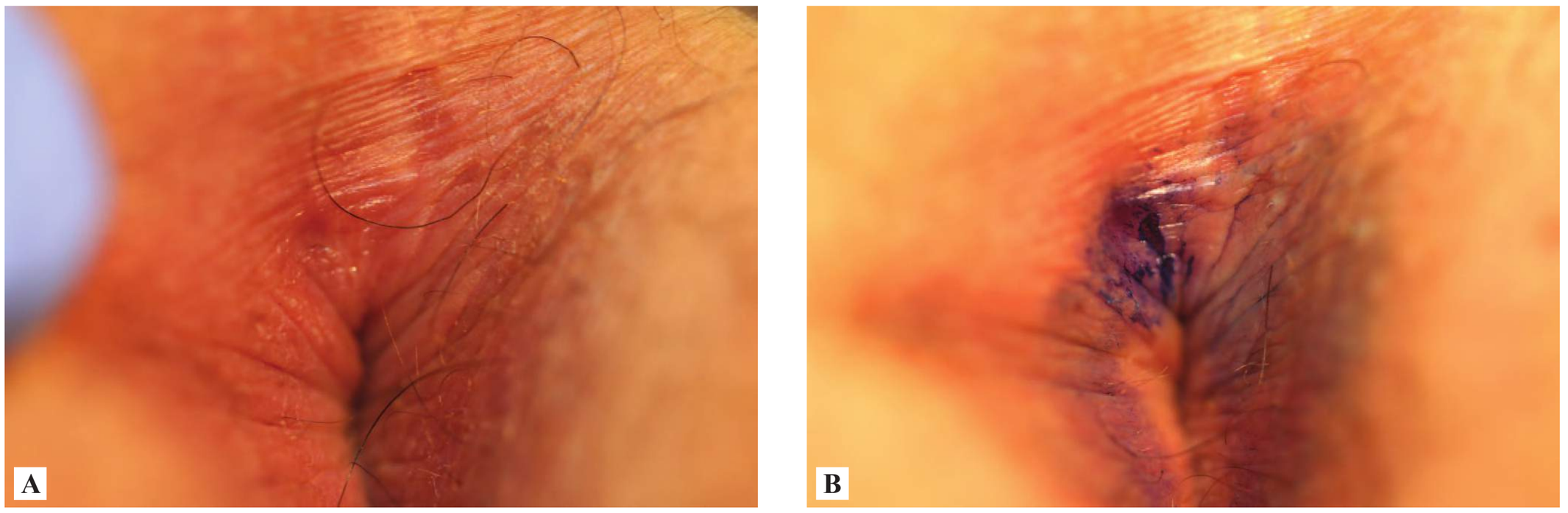


FIGURE 10.18 ■ **Anal Trauma (Perianal, Toluidine Blue).** Multiple perianal tears without toluidine blue stain (**A**) and with stain (**B**). Note specific stain uptake enhancing two tears at 11-o'clock position in contrast to nonspecific uptake elsewhere. (Photo contributors: Hillary J. Larkin, PA-C, and Josua Luftig, PA-C.)

Clinical Summary

Lichen sclerosus (LS) is a benign, chronic, inflammatory condition that results in epithelial atrophy or hyperplasia, and scarring. The condition typically occurs in the anogenital region (85-98%), but can develop on any skin surface. Women are affected 10 times more often than men and onset is often perimenopausal. The etiology is unknown, but it is seen more commonly in individuals with autoimmune conditions such as diabetes mellitus type 1, vitiligo, and thyroid disorders in women in a low estrogen state, suggesting that these may be contributing factors.

Patients may be asymptomatic or may have intense pruritis and pain in the affected areas. Other symptoms may include bleeding, dyspareunia, or dysuria. Lesions appear as white, atrophic papules and patches and most often affect the labia minora and/or labia majora. Excoriations may be present and scratching may lead to secondary lichenification (thickening of the epidermis with exaggeration of normal skin lines). As the disease progresses, scarring may lead to loss of the vulvar architecture with fusion of the labia. The vagina and cervix are not involved. Extragenital lesions may occur on the thighs, breasts, wrists, shoulders, neck, and, rarely, the oral cavity. These lesions also consist of white papules or plaques and are not as pruritic as genital lesions.

Management and Disposition

No treatment is necessary. Potent topical steroids, hormonal treatments, and/or immunosuppressants may benefit. Advise patients on good vulvar hygiene cessation of scratching to prevent secondary infections. Consider oral antihistamines for pruritis. Refer patients for outpatient gynecological evaluation. Definitive diagnosis is made by biopsy.

Pearls

1. LS is associated with an increased risk of vulvar squamous cell cancer. Refer all patients for biopsy and histologic evaluation.
2. Although LS is not an infectious condition, the presence of open excoriations and fissures can increase risk of acquiring sexually transmitted infections.



FIGURE 10.19 ■ **Lichen Sclerosus.** Early lichen sclerosus appearing as white, atrophic papules and patches of the labia minora and labia majora. (Photo contributor: Hope Haefner, MD.)

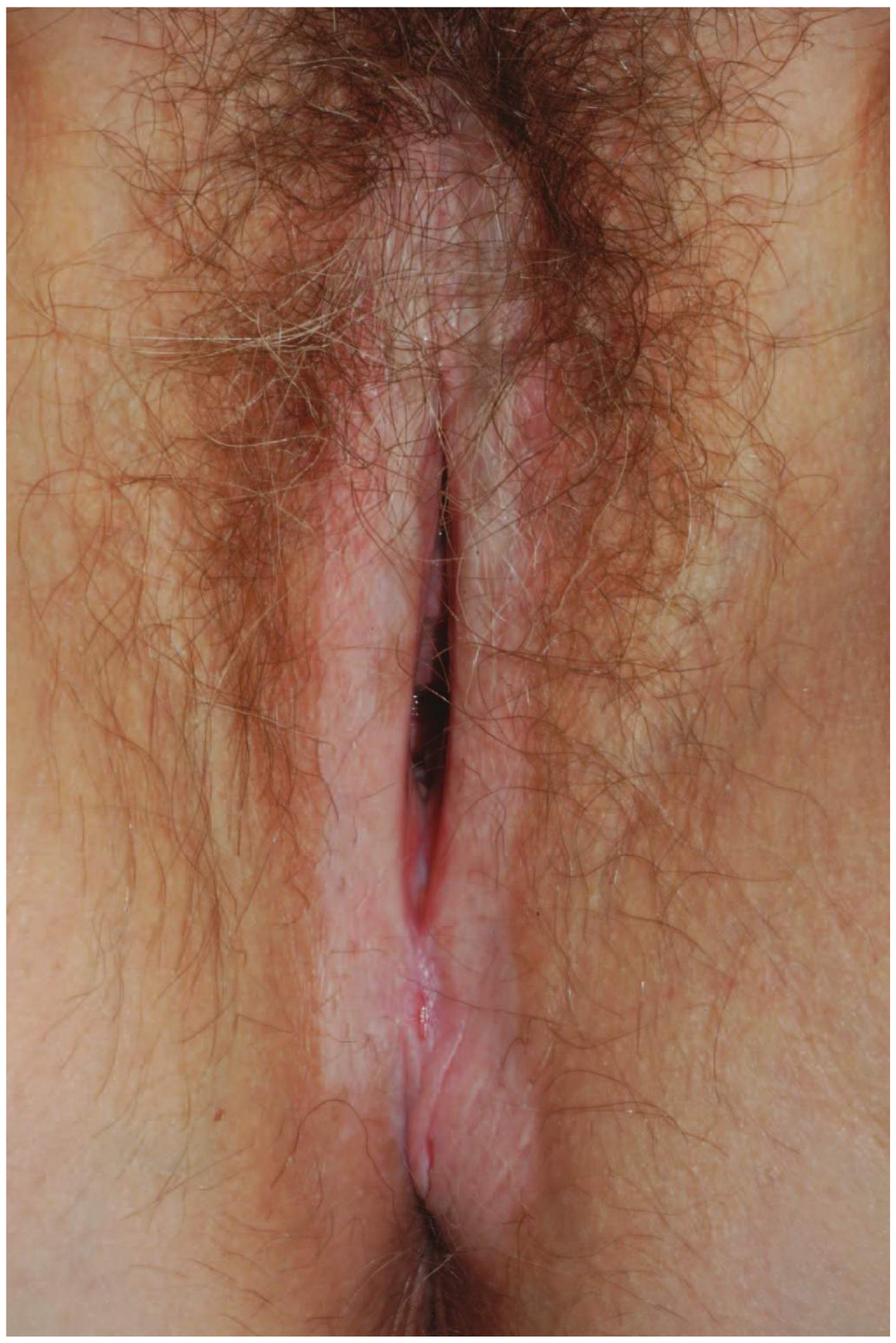


FIGURE 10.20 ■ **Lichen Sclerosus—Chronic.** Atrophy and scarring of chronic lichen sclerosus with early fusion of the anterior labia. (Photo contributor: Hope Haefner, MD.)



FIGURE 10.21 ■ **Lichen Sclerosus—Advanced.** Extensive fusion of the labia minora in advanced lichen sclerosus over the vaginal opening. (Photo contributor: Hope Haefner, MD.)

Clinical Summary

Lichen planus is a chronic inflammatory condition that can affect any area of skin, mucosa, nails, and scalp. Vulvar involvement is found in 50% of women with lichen planus. The etiology is unknown, but may be autoimmune as the disorder is often associated with other autoimmune diseases.

Affected women may complain of intense vulvar pruritus, pain, and/or dysuria. Vaginal involvement is seen in 70% of cases and may cause vaginal discharge, dyspareunia, and occasionally postcoital bleeding. Coexisting oral or other cutaneous lesions are common. On examination, vulvar lichen planus is characterized by glassy, erosive, or papular lesions. Desquamation may be present. Recurrent lesions may lead to extensive scarring with resultant loss of the vulvar architecture including stenosis of the vaginal introitus and urethral obstruction.

Lichenoid drug eruptions can be clinically indistinguishable from lichen planus. Use of specific medications including β-blockers, methyldopa, penicillamine, quinidine, nonsteroidal anti-inflammatory drugs (NSAIDs), angiotensin-converting-enzyme (ACE) inhibitors, sulfonylurea agents, carbamazepine, gold, lithium, quinidine, or hydrochlorothiazide (HCTZ) has been associated with lichenoid drug eruptions.

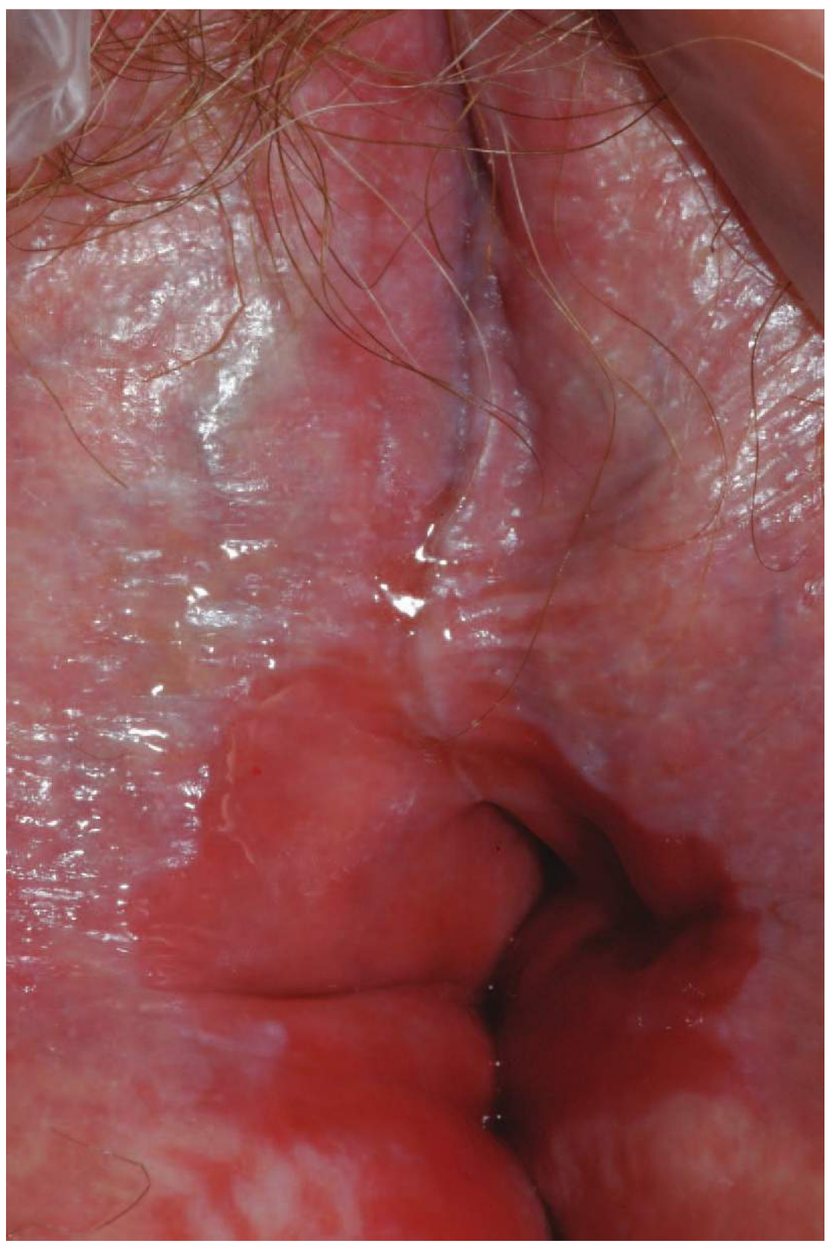


FIGURE 10.22 ■ **Erosive Lichen Planus.** Glassy erosive lesions of lichen planus with "lichenification," or thickening of the epidermis, in peripheral areas. (Photo contributor: Hope Haefner, MD.)

Management and Disposition

No treatment is necessary in the ED. Potent topical steroids are first-line treatment. Sitz baths may help alleviate symptoms. Advise patients on good vulvar hygiene including cessation of scratching to prevent secondary infections. Consider oral antihistamines for pruritis. Discontinue any medications that may be associated with a lichenoid drug eruption. Refer all patients for outpatient gynecological evaluation. Definitive diagnosis is made by biopsy.

Pearls

1. HCTZ and NSAIDs are common triggers for recurrent lichen planus. Discontinuation of these medications may improve symptoms.
2. Lichen planus does not involve the perianal region.



FIGURE 10.23 ■ **Erosive Lichen Planus.** Glassy erosive lesions of lichen planus. (Photo contributor: Hope Haefner, MD.)



FIGURE 10.24 ■ **Erosive Lichen Planus—Oral.** Lichen planus can affect any area of skin, mucosa, nails, and scalp. (Photo contributor: Hope Haefner, MD.)

Clinical Summary

Vulvar cancer accounts for 5% of gynecological malignancies and occurs most commonly in postmenopausal women. Many vulvar neoplasms are asymptomatic and are discovered incidentally during gynecologic examination. Patients with symptoms most commonly complain of vulvar pruritis. They may also have noticed a vulvar mass. In advanced cases, vulvar bleeding or discharge, dysuria, or inguinal lymphadenopathy may be present. On examination, there may be a single lesion, most commonly on the labia majora, that appears as a plaque, ulcer, or mass (fleshy, nodular, or warty). The labia minora, perineum, clitoris, and mons are less frequently involved and less than 5% of cases have multifocal lesions.

Squamous cell carcinomas account for 90% of vulvar cancers, while melanoma is the second most common type. Smoking, chronic inflammatory conditions such as LS, and infection with HPV 16, 18, or 33 are risk factors for vulvar cancer.

Management and Disposition

No treatment is necessary in the ED. Refer all patients with suspicious vulvar lesions for outpatient gynecological evaluation. Definitive diagnosis is made by biopsy.

Pearl

1. Coexisting cervical neoplasia are found in more than 20% of patients with a vulvar malignancy.



FIGURE 10.25 ■ **Squamous Cell Carcinoma—Vulvar.** Lichen sclerosus, seen surrounding the erythematous squamous cell carcinoma, is a risk factor for this neoplasm. (Photo contributor: Hope Haefner, MD.)



FIGURE 10.26 ■ **Squamous Cell Carcinoma—Vulvar.** Vulvar neoplasms may be asymptomatic and identified incidentally during examination for other complaints. (Photo contributor: Hope Haefner, MD.)

Clinical Summary

Urethral prolapse is a rare condition presenting as a protrusion of the distal urethra through the distal meatus, typically seen as a donut-shaped mass at the anterior vaginal wall. It occurs most commonly in prepubertal black females and postmenopausal white women. Presenting signs (vaginal bleeding and urinary complaints) are uncommon in children but common in the postmenopausal woman. Diagnosis is made when the central opening in the prolapsed tissue is confirmed as the urethral meatus. Urethral catheterization may be required if direct observation during voiding is not possible. A large mucosa prolapse may become strangulated, resulting in pain, bleeding, and urinary symptoms.

Management and Disposition

Confirm the diagnosis and rule out urinary tract infection. Consult urology emergently for urethral strangulation. Medical therapy consists of sitz baths and topical estrogen cream applied two to three times to the urethra for 2 weeks. Surgical therapy is reserved for strangulation or failed medical therapy. Refer all patients with confirmed or suspected urethral prolapse to a urologist for follow-up as urethral masses may be misdiagnosed as urethral prolapse.

Pearls

1. Urethral prolapse is often associated with urinary tract infection.
2. Topical estrogen cream may be contraindicated in patients with a history of breast cancer.
3. Urethral prolapse in children may be mistaken for sexual abuse (see Chapter 15).



FIGURE 10.27 ■ **Urethral Prolapse.** Note the protrusion of the distal urethra, seen as a donut-shaped mass at the anterior vaginal wall. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 10.28 ■ **Urethral Prolapse.** Protrusion of the distal urethra is seen as a donut-shaped mass just superior to the vaginal introitus in a patient complaining of "vaginal bleeding." (Photo contributor: Lawrence B. Stack, MD.)

UTERINE PROLAPSE

Clinical Summary

Uterine prolapse is defined as the extrusion of the uterus through the pelvic floor or vaginal introitus. Prolapse is caused by weakened pelvic floor muscles and is common in older, multigravid women. Degrees of prolapse are identified by level of descent of uterine tissues. In first-degree prolapse, the cervix descends into the lower third of the vagina, while in second-degree the cervix usually protrudes through the introitus. Third-degree prolapse, or procidentia, is characterized by externalization of the entire uterus with eversion of the vagina. Symptoms include a sensation of inguinal traction, low back pain, urinary incontinence, and the presence of a vaginal mass. Uterine prolapse can occasionally be confused with a cystocele (discussed below), enterocele, or soft tissue tumor.

Management and Disposition

Refer patients with first- or second-degree prolapse to a gynecologist for pessary placement or surgical correction. With procidentia, manually reduce the uterus into the vaginal vault, place the patient on complete bed rest, and obtain urgent gynecologic consultation.

Pearls

1. With procidentia, the exposed uterus is prone to abrasion and possible secondary infection.
2. Prolapse, cystocele, and enterocele can often be enhanced or reproduced by having the patient perform a Valsalva maneuver.



FIGURE 10.29 ■ **Third-Degree Uterine Prolapse.** Note the protrusion of the entire uterus with the cervix visible through the vaginal introitus. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

A cystocele is a herniation of the posterior bladder wall and trigone through the anterior wall of the vagina caused by weakening of the pelvic floor during childbirth. Patients complain of vaginal bulging or fullness that is worsened with Valsalva and improved with recumbency. Cystoceles are often associated with urinary incontinence and incomplete emptying of the bladder. Rectoceles, uterine prolapse, and soft tissue tumors should also be considered.

Management and Disposition

Generally, ED care consists of reassurance and outpatient referral to a gynecologist for further evaluation and treatment. Advise the patient to avoid heavy lifting or straining. Definitive treatment for larger cystoceles or those associated with significant urinary symptoms, pain, or bothersome bulging may require pessary placement or surgery.

Pearl

1. Most cystoceles are asymptomatic and are detected incidentally at the time of pelvic examination.



FIGURE 10.30 ■ **Cystocele.** Cystocele with bulging of the posterior bladder wall into the vagina. (Photo contributor: Matthew Backer, Jr., MD.)



FIGURE 10.31 ■ **Cystocele.** Cystocele worsening with Valsalva. (Photo contributor: Matthew Backer, Jr., MD.)

RECTOCELE

Clinical Summary

A rectocele is the herniation of the rectum through the posterior vaginal wall. Most small rectoceles are completely asymptomatic, though symptoms of introital bulging, constipation, and incomplete rectal evacuation may occur. Bulging of the posterior vaginal wall through the introitus may be seen grossly on physical examination and will be enhanced with Valsalva. Vaginal examination reveals a thin-walled protrusion of the rectovaginal septum into the lower part of the vagina.

Management and Disposition

Supportive measures with hydration, laxatives, and stool softeners are generally all that is needed to relieve the patient's symptoms. Refer patients with large or symptomatic rectoceles to a gynecologist for possible surgical repair.



FIGURE 10.32 ■ **Rectocele.** This is characterized by bulging of the posterior vaginal wall at the introitus. (Photo contributor: Matthew Backer, Jr., MD.)

Pearl

1. Patients may complain of the need for manual reduction of the rectocele when defecating.

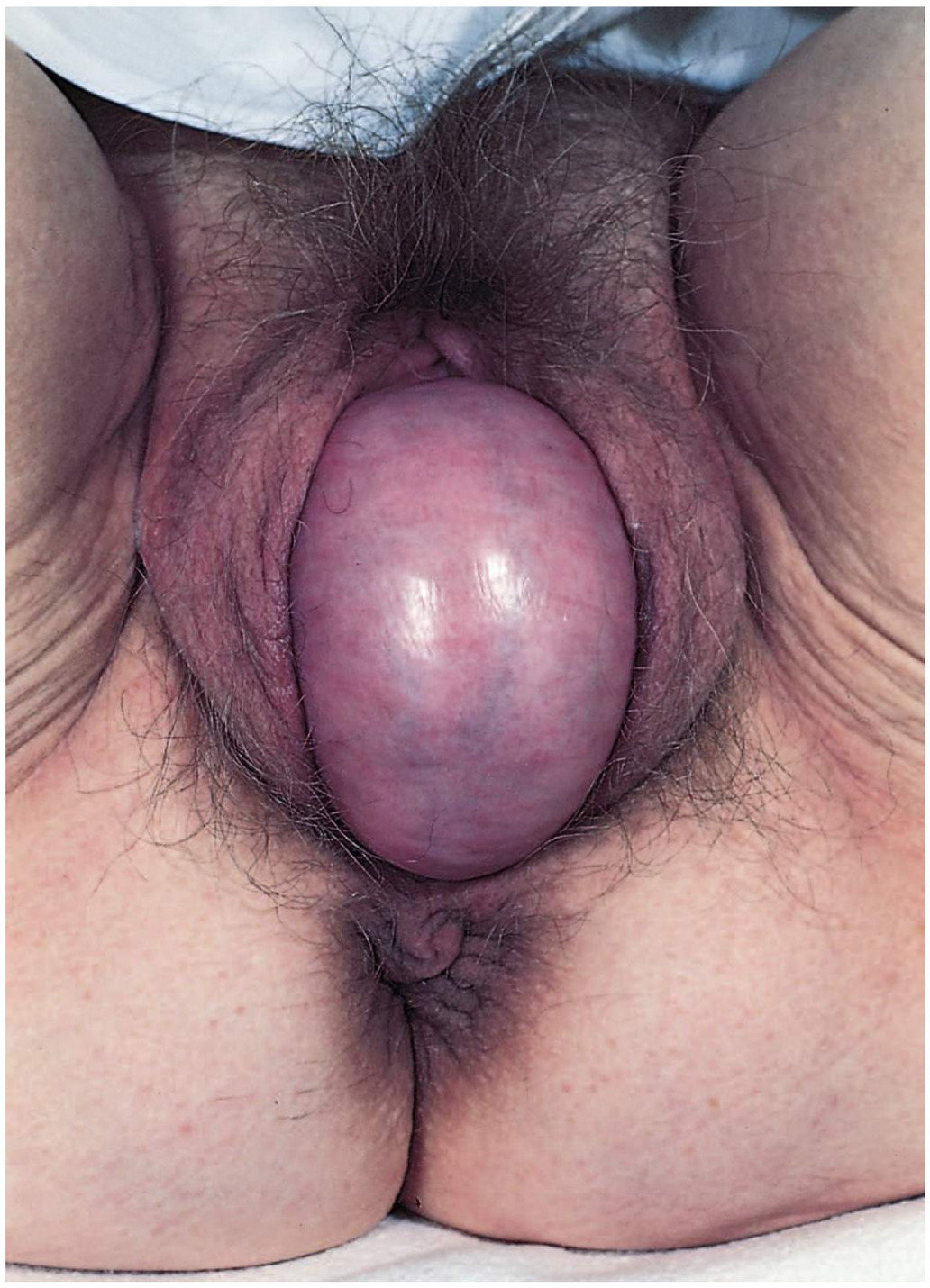


FIGURE 10.33 ■ **Rectocele.** Worsening of the rectocele with Valsalva. (Photo contributor: Matthew Backer, Jr., MD.)

Clinical Summary

The hymen is a membrane visible at the vaginal introitus that separates the vestibule externally from the vagina internally. Normally there is an opening in the hymen that allows for the passage of fluids. The congenital absence of a hymenal orifice is called an imperforate hymen. This condition may present in infants or young children as a smooth, glistening, membrane protruding from the introitus due to the buildup of vaginal secretions known as a mucocolpos. More commonly, it becomes evident in adolescent girls when it may present as a hematocolpos. Hematocolpos is due to the accumulation of menstrual blood and secretions behind the hymen which on physical examination is evident as a smooth, dome-shaped, bluish-red bulging membrane at the introitus. A large, smooth, cystic mass can often be palpated anteriorly on digital rectal examination. The fluid collection can become large enough to cause urinary retention by compressing the bladder neck. Occasionally the accumulated blood may spill over through the fallopian tubes into the peritoneal cavity, resulting in free pelvic fluid and signs of peritonitis.

Management and Disposition

Imperforate hymen as well as other abnormalities of the vaginal outlet should be referred to a gynecologist for definitive treatment. This includes incision of the hymen to allow drainage of the hematocolpos. Those instances detected in preadolescence should ideally be referred to a practitioner who specializes in pediatric cases.

Pearl

1. An imperforate hymen commonly presents in adolescent girls with primary amenorrhea and recurrent abdominal pain.



FIGURE 10.34 ■ **Imperforate Hymen.** A bulging mass at the introitus is seen in this patient with abdominal distention and amenorrhea. The imperforate hymen was diagnosed, with subsequent incision and drainage of the hematocolpos. (Photo contributor: Mark Eich, MD.)

Clinical Summary

Ectopic pregnancy is the leading cause of first-trimester maternal obstetric morbidity. Presentations commonly include mild vaginal bleeding and lower abdominal pain, but patients can present in shock secondary to massive hemorrhage. Menstrual history may reveal a missed or recent abnormal menses. Pelvic examination may be normal or may include frank cervical motion tenderness or adnexal tenderness. Ectopic pregnancy is more common in women with a history of pelvic inflammatory disease or other causes of fallopian tube scarring.

The visualization of an IUP by US essentially excludes the diagnosis of ectopic pregnancy, the exception being a rare heterotopic pregnancy (both intrauterine and ectopic). The appearance of a gestational sac (GS) at about 5 weeks is the first significant finding on US suggestive of an IUP; however, definitive diagnosis of IUP should be deferred until a yolk sac is present. A fetal pole develops next and can be seen on part of the yolk sac. The double decidual sac sign is evidence of a true GS and should be differentiated from the single pseudogestational sac formed from a decidual cast in ectopic pregnancy. When no GS is visualized ("empty uterus"), ectopic pregnancy cannot be distinguished from an early IUP or from recently completed spontaneous abortion. Free fluid on US is nonspecific, but concerning if present with a clinical suspicion for ectopic pregnancy.

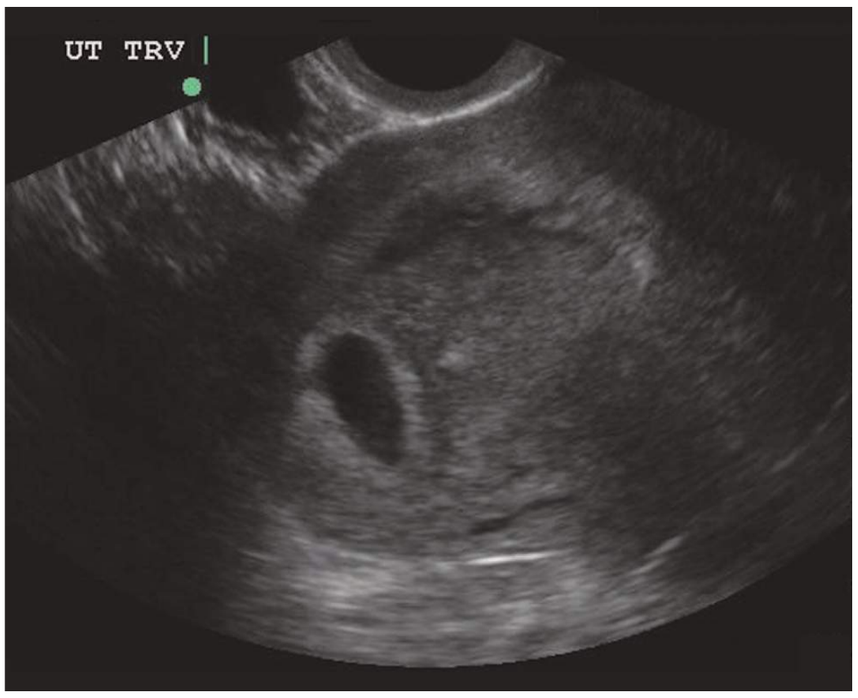


FIGURE 10.36 ■ **Intrauterine Gestational Sac.** Discrete ring of an intrauterine gestational sac seen on transvaginal ultrasound. No yolk sac is visualized. A double decidual sac sign is seen, lending evidence of a true gestational sac versus a pseudogestational sac formed from a decidual cast in ectopic pregnancy. A thorough look in the adnexa is important in diagnosing ectopic pregnancy when a gestational sac is the only finding. (Photo contributor: Lauren Oliveira, DO.)

Management and Disposition

Unstable patients require aggressive resuscitation with fluid and blood followed by surgery. Stable patients with a US

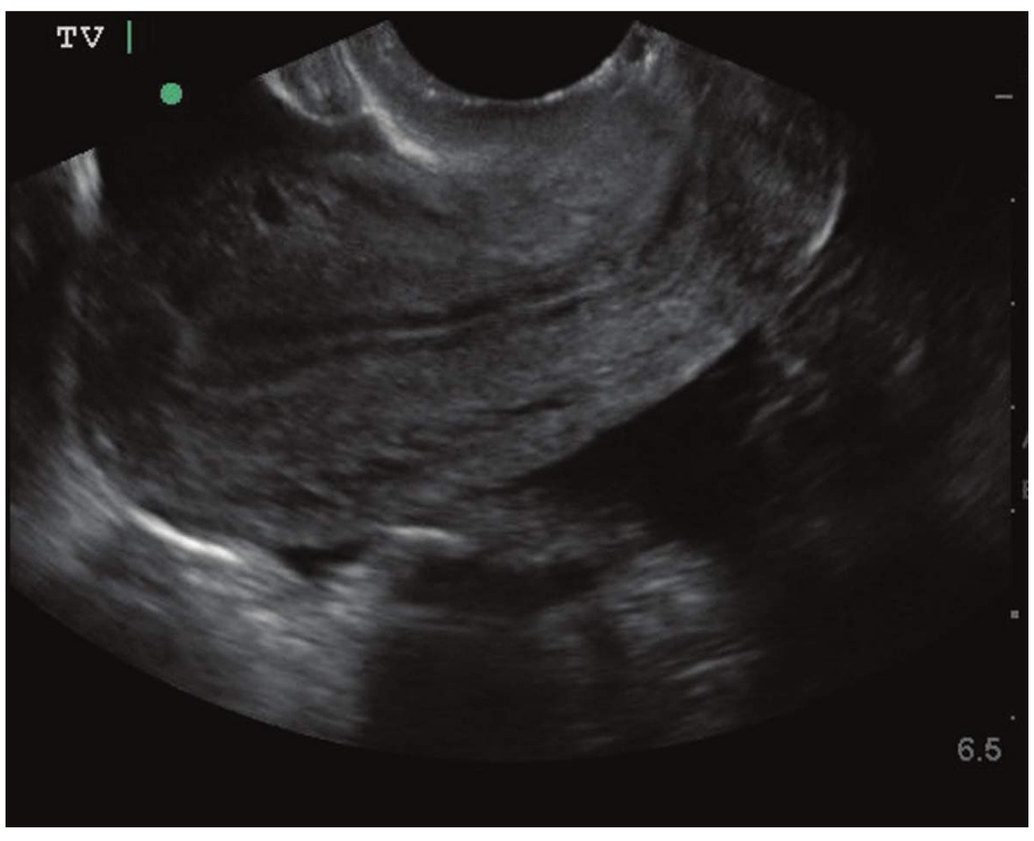


FIGURE 10.35 ■ **Empty Uterus.** Free fluid seen in the pelvis in this patient with an empty uterus is concerning if ectopic pregnancy is suspected. Ectopic pregnancy should be strongly suspected if a transvaginal ultrasound reveals an empty uterus in the setting of a serum quantitative hCG level above the institution's discriminatory zone. (Photo contributor: Lauren Oliveira, DO.)

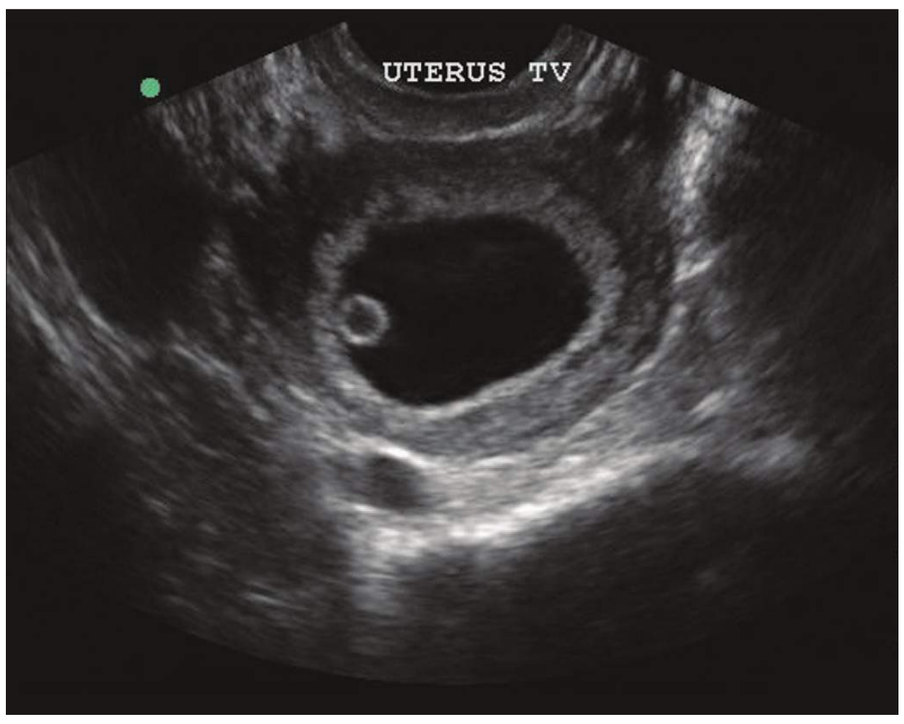


FIGURE 10.37 ■ **Intrauterine Yolk Sac.** Discrete ring of an intrauterine yolk sac within the gestational sac seen on transvaginal ultrasound. Definitive diagnosis of IUP can be made once a yolk sac is seen. A double decidual sac sign is also seen. (Photo contributor: Lauren Oliveira, DO.)

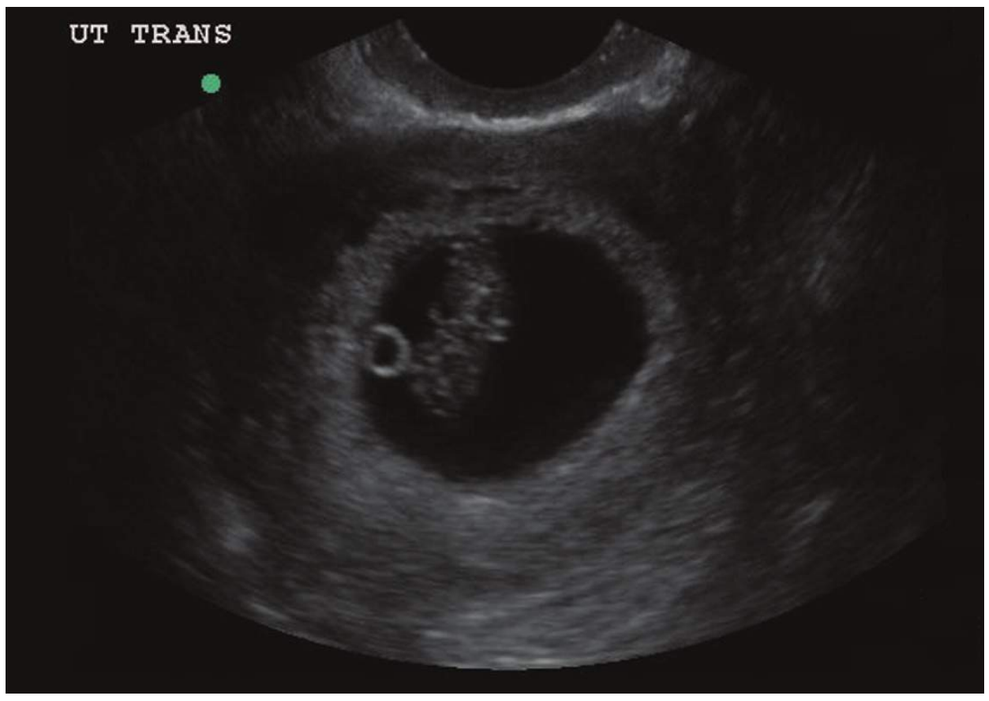


FIGURE 10.38 ■ **Intrauterine Fetal Pole.** Ultrasound image of an intrauterine pregnancy with a fetal pole seen adjacent to a yolk sac. (Photo contributor: Lauren Oliveira, DO.)

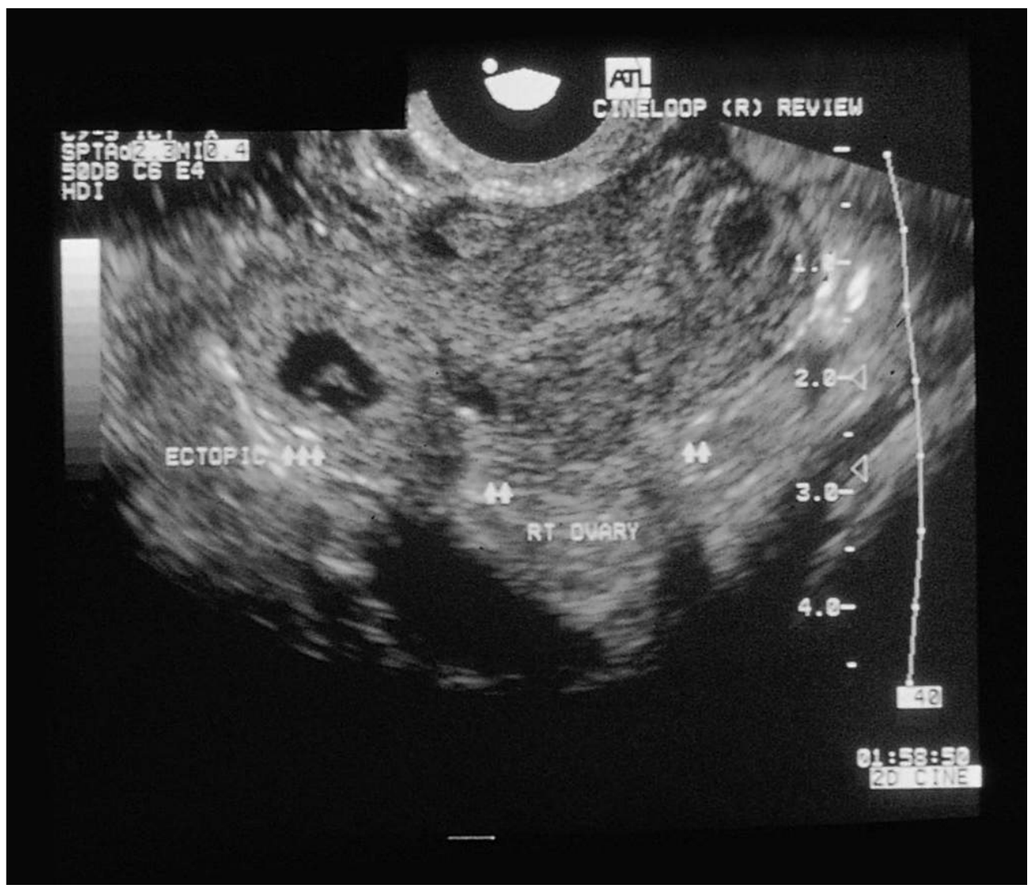


FIGURE 10.39 ■ **Ectopic Pregnancy.** A large ectopic pregnancy is seen adjacent to the uterus. (Photo contributor: Lauren Oliveira, DO.)

diagnosis consistent with ectopic pregnancy warrant immediate gynecologic consultation. Definitive therapeutic options range from observation (in an asymptomatic patient with declining hCG levels), to pharmacologic therapy with methotrexate, to surgery. US has diminished diagnostic accuracy at lower hCG levels (up to half of ectopic pregnancies have a serum hCG level <2000 mIU/mL, and ectopic pregnancy has been observed at hCG level less than 100). Therefore, obtain gynecologic consultation if there is a clinical suspicion for ectopic pregnancy regardless of the US findings or hCG level. Those patients in whom a normal IUP is visualized can be safely discharged with early outpatient follow-up.

Pearls

1. Ectopic pregnancy should be considered in all women of reproductive age presenting with syncope, vaginal bleeding, abdominal pain or tenderness, and/or a missed menstrual period.



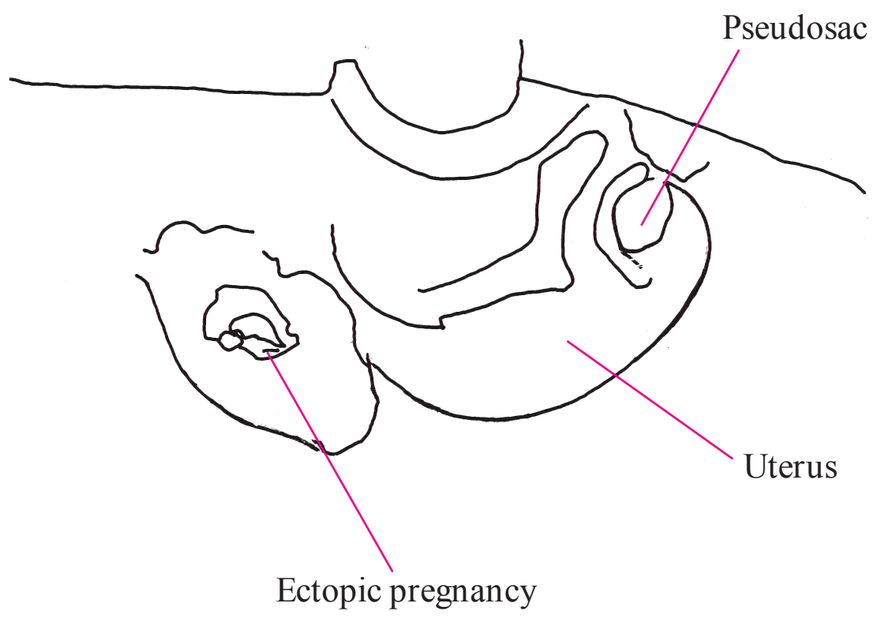


FIGURE 10.40 ■ **Ectopic Pregnancy.** Transvaginal ultrasound image of a right ectopic pregnancy with a decidual reaction in the uterus resembling a gestational sac, or "pseudosac." Visualization of a pseudogestational, or "single," sac sign could be consistent with an early gestational sac or an ectopic pregnancy with a uterine decidual cast. (Photo contributor: Janice Underwood.)

2. Failure to visualize an intrauterine pregnancy by transvaginal ultrasonography when the serum hCG level is above the discriminatory zone of the machine and examiner (typically 1500-2000 mIU/mL) is highly suggestive of ectopic pregnancy.
3. The double decidual sac sign is not considered reliable to confirm an IUP. A yolk sac must be present.
4. Visualization of the adnexa on US may reveal tubal ectopic pregnancy, but failure to show this does not necessarily rule out the diagnosis.

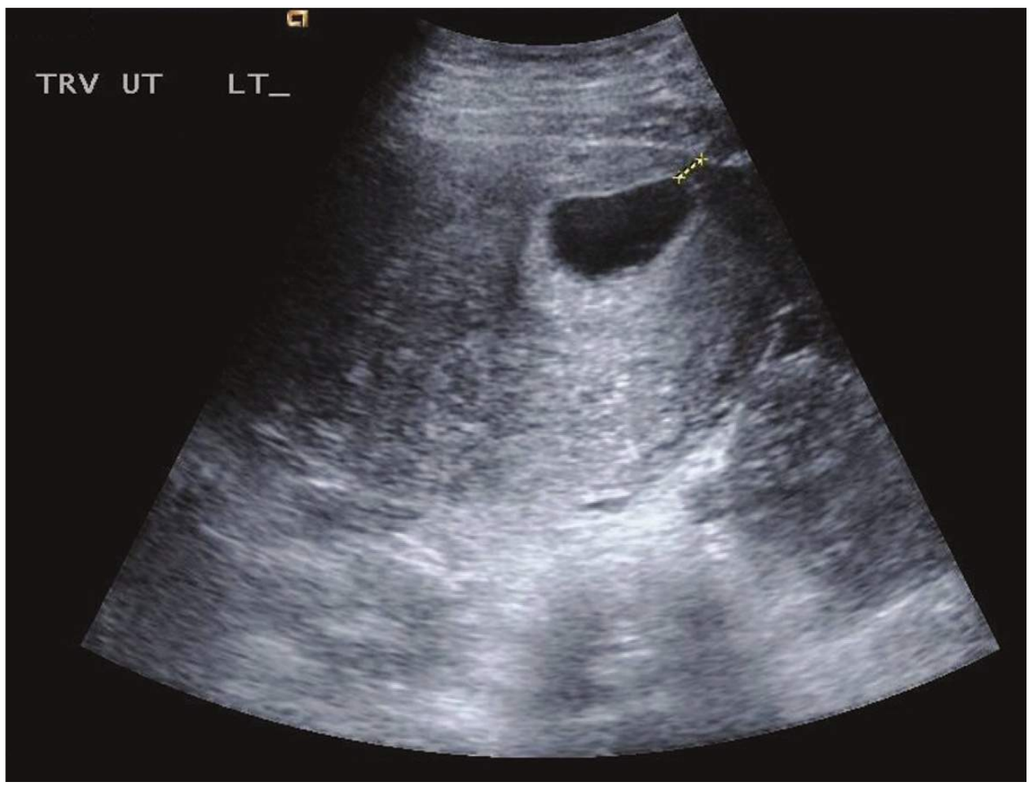


FIGURE 10.41 ■ **Interstitial Ectopic.** An interstitial ectopic exists when the gestational sac is less than 5 mm away from the uterine wall. This formal ultrasound confirmed a bicornate uterus and viable pregnancy with a distance 6.1 mm, enough endometrium to support normal pregnancy. (Photo contributor: Lauren Oliveira, DO.)

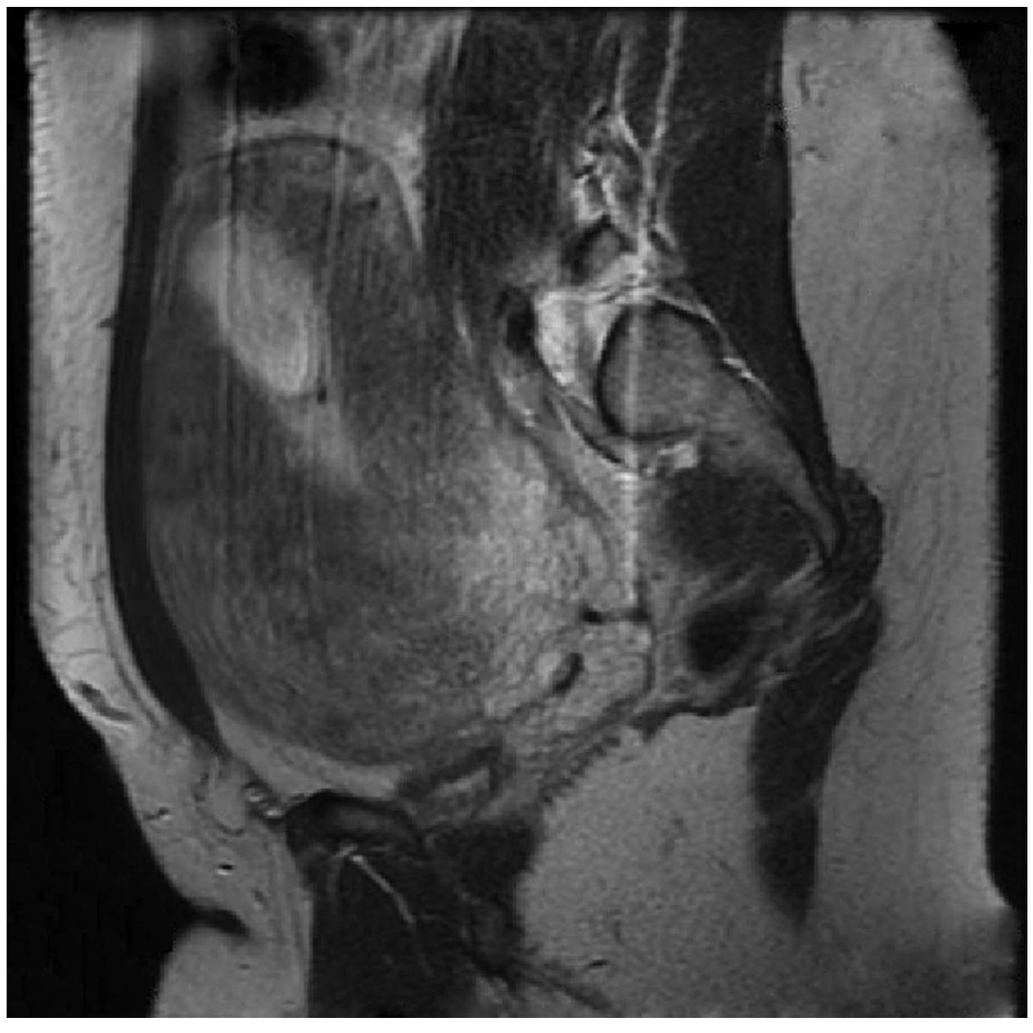


FIGURE 10.42 ■ **Interstitial Ectopic.** This MRI confirmed a formal ultrasound of an interstitial ectopic. (Photo contributor: Lauren Oliveira, DO.)

MOLAR PREGNANCY (HYDATIDIFORM MOLE)

Clinical Summary

Molar pregnancy is part of a spectrum of gestational trophoblastic tumors that include benign hydatidiform moles, locally invasive moles, and choriocarcinoma. The classic clinical presentation is painless first-trimester or early second-trimester vaginal bleeding with a uterine size larger than the estimated gestational age based on the last menstrual period. Signs of preeclampsia (hypertension, headache, proteinuria, and edema) in the first trimester or early second trimester are highly suggestive of this diagnosis as well. Hyperthyroidism can be found in roughly 5% of cases. Acute respiratory distress may occur due to embolization of trophoblastic tissue into the pulmonary vasculature, thyrotoxicosis, or simple fluid overload.

Management and Disposition

Obtain gynecologic consultation for dilatation and curettage in all cases. Close outpatient monitoring of serum hCG levels is required to rule out the presence of malignant gestational trophoblastic disease.

Pearls

1. All patients with pregnancies of less than 20 weeks' gestation with clinical findings of preeclampsia should have gestational trophoblastic disease ruled out.
2. A "snowstorm" pattern on ultrasonography (demonstrating multiple intrauterine echoes with no fetus) coupled with a high hCG level is typical of molar pregnancy.
3. "Moles" commonly produce serum hCG levels greater than 100,000 mIU/mL.



FIGURE 10.43 ■ **Molar Pregnancy.** "Snowstorm" pattern demonstrating multiple intrauterine echoes with no fetus seen on transvaginal ultrasonography in a patient with a molar pregnancy. Serum β-hCG was greater than 180,000 mIU/mL. (Photo contributor: Robin Marshall, MD.)



FIGURE 10.44 ■ **Molar Pregnancy.** Transabdominal study showing molar pregnancy. These are exceedingly difficult to identify and diagnose by ultrasound. They can be easily mistaken for nonspecific intrauterine findings (eg, missed abortion, fibroid). (Photo contributor: Lauren Oliveira, DO.)

FAILED INTRAUTERINE PREGNANCY

Clinical Summary

Fetal viability may be in question for some pregnant patients presenting to the ED. Conservative diagnostic criteria for nonviable pregnancy are based upon transvaginal US imaging and include: a crown-rump length of 7 mm with no cardiac activity; a mean GS diameter of ≥25 mm and no visible embryo; no heartbeat ≥2 weeks after a visualized GS without a yolk sac; and no heartbeat ≥11 days after a prior documented GS with a yolk sac. Findings that approximate these criteria may be suspicious for, but are not diagnostic, of a failed pregnancy.

Management and Disposition

The emergency physician must ensure that any criteria used to diagnose failed IUP minimize the possibility of a false positive finding (ie, incorrectly diagnosing nonviability). Consider obtaining a formal US to confirm ED besides US findings. Arrange a follow-up US in 7 to 10 days for patients with findings suspicious for but not meeting criteria for a failed IUP.

Pearls

1. Obtain gynecologic consultation and follow-up to confirm ED findings for patients whose fetal viability is in question.

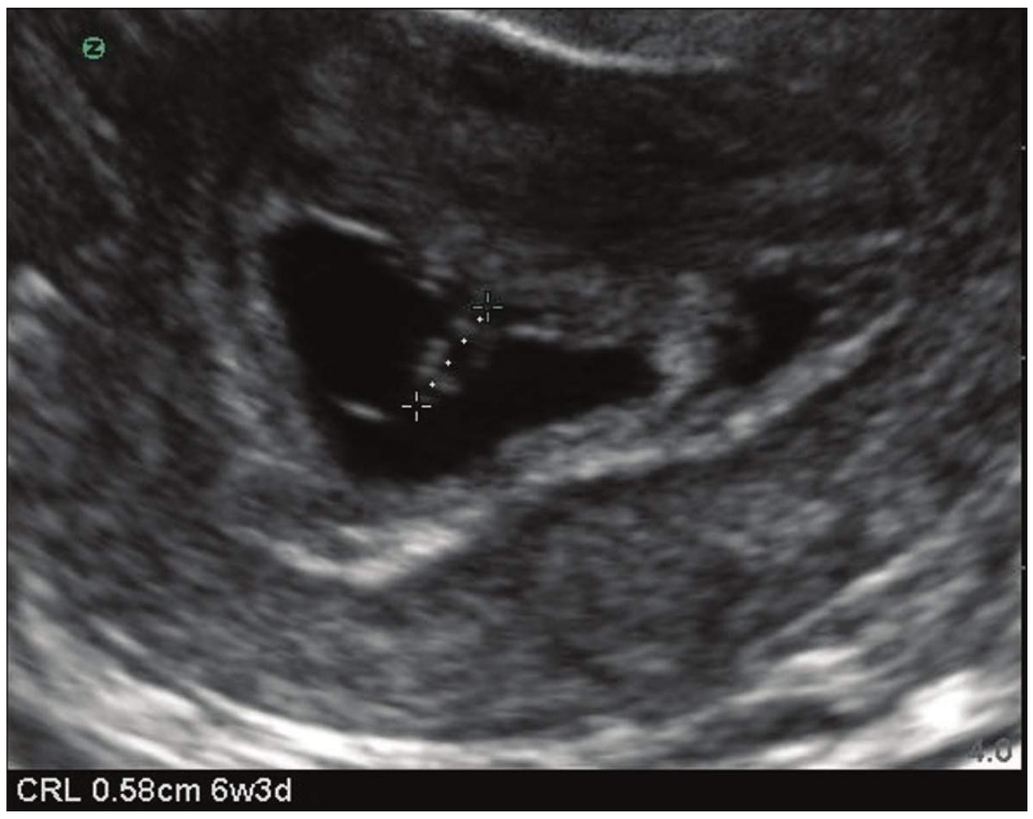


FIGURE 10.45 ■ **Indeterminate Viability.** This fetal pole with a crown-rump length of 5.8 mm and no cardiac activity is consistent with indeterminate viability versus a failed IUP which is diagnosed if crown-rump length is ≥7 mm and no cardiac activity. (Photo contributor: Kevin J. Knoop, MD.)

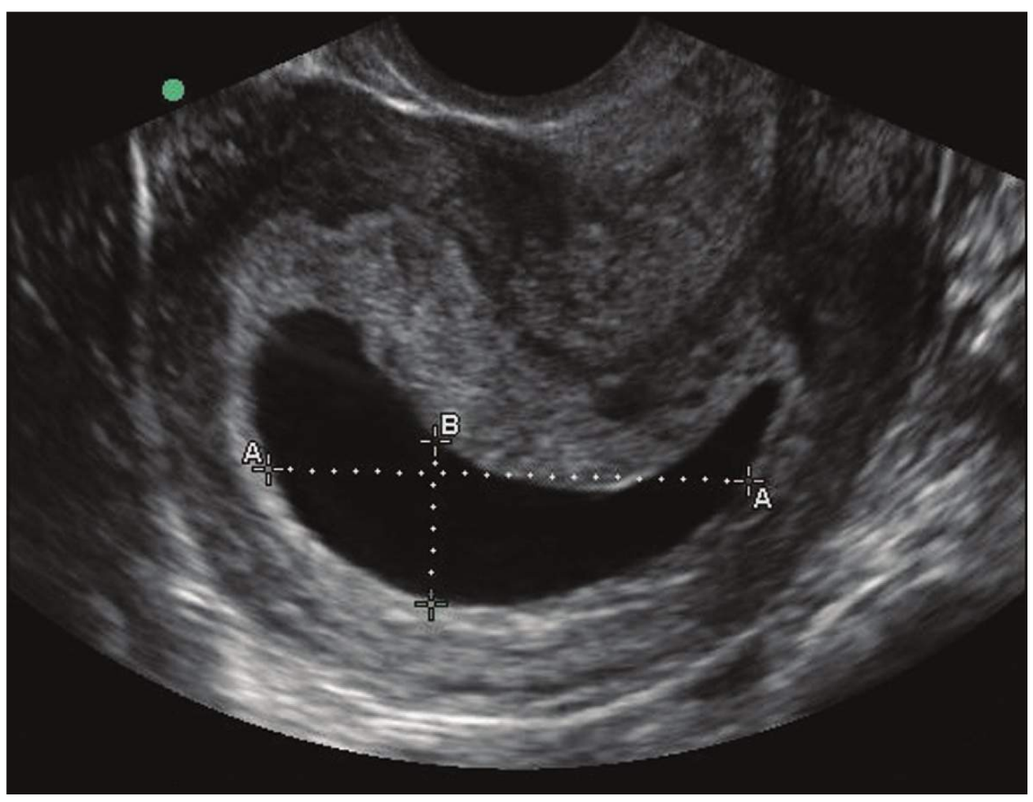


FIGURE 10.46 ■ **Failed Intrauterine Pregnancy.** This enlarged gestational sac with no visible cardiac activity is consistent with a failed IUP if the mean gestational sac diameter is ≥25 mm. (Photo contributor: Lauren Oliveira, DO.)

THIRD-TRIMESTER BLUNT ABDOMINAL TRAUMA

Clinical Summary

Trauma is a major cause of maternal and fetal mortality. In addition to the common injuries to solid organs and/or hollow viscus associated with blunt abdominal trauma, special consideration should be given to the possibility of preterm labor, fetal-maternal hemorrhage, uterine rupture, and, most importantly, abruptio placentae. Abruptio placentae is defined as the premature separation of the placenta from the site of uterine implantation. It is found in up to 50% of major blunt trauma patients and up to 5% of those with apparent minor injuries. There are generally signs of uterine hyperactivity and fetal distress when significant placental detachment occurs. Most patients have vaginal bleeding, but up to 20% will present with little or no external bleeding when the margins of detachment are above the cervical os.

Electronic fetal monitoring for a minimum of 4 hours is indicated in all cases of significant trauma in patients beyond 20 weeks' gestation. As the pregnancy progresses toward term, a normal fetal heart rate averages between 120 and 160 bpm. Rapid, frequent fluctuations in the baseline are characteristic of normal "reactivity." The loss of this reactivity can occur during a normal fetal sleep cycle, following narcotic administration, or, most importantly, in the setting of fetal hypoxia or distress. Significant transient decelerations in fetal heart rate are classified as early, late, or variable depending on how they correlate with uterine contractions (via tocometry).

Management and Disposition

In addition to performing a standard trauma evaluation, immediately consult an obstetrician in all pregnant trauma patients beyond 20 weeks' gestation. Obtain blood for type- and cross-matching, CBC, prothrombin time (PT), partial thromboplastin time (PTT), fibrinogen, and fibrin degradation products or d-dimer. It is generally recommended that patients undergo continuous tocometric and fetal heart rate monitoring for a minimum of 4 hours to rule out preterm labor or fetal distress. US is essential in visualizing placental abruption, and differentiates this from vaginal bleeding due to placenta previa. Indications for emergency cesarean section include placental abruption, signs of ongoing fetal distress, or uncontrolled maternal hemorrhage.

Pearls

1. Ecchymotic markings from blunt force are not always present on a gravid abdomen; thus, a careful history of the mechanism of trauma and complaints is essential.
2. Anti-Rh immunoglobulin should be administered for all cases of significant third-trimester blunt abdominal trauma if the mother is Rh-negative.
3. Laboratory evidence of a consumptive coagulopathy may be seen with significant abruption.

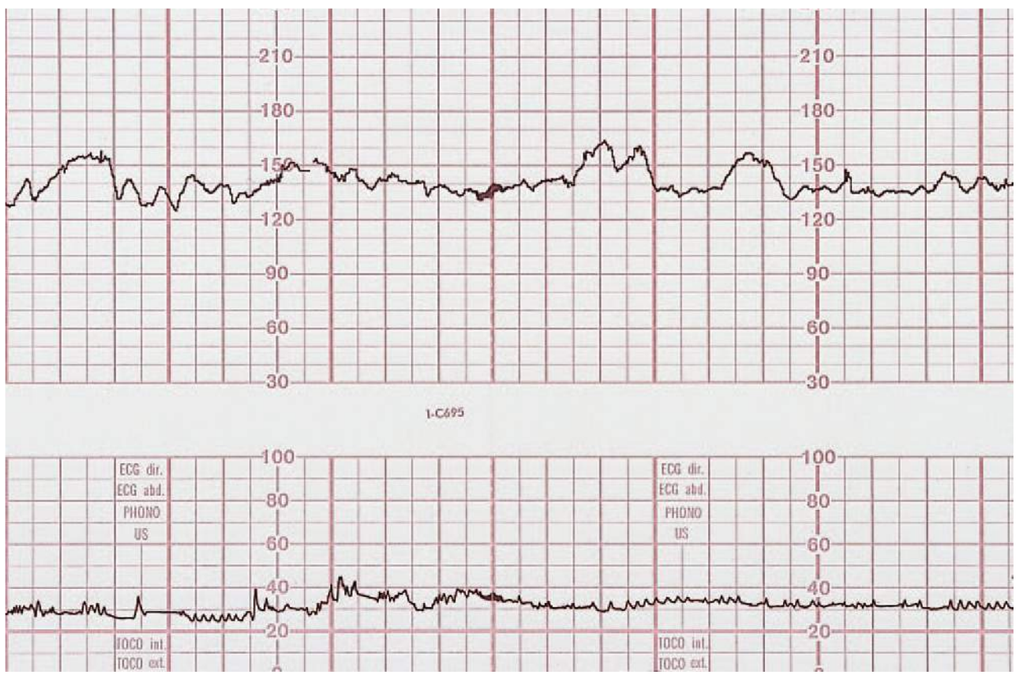


FIGURE 10.47 ■ **Normal Beat-to-Beat Variability (BBV).** A normal reactive fetal monitor strip showing a baseline heart rate between 120 and 160 bpm with fluctuations in the short- and long-term heart rate. (Photo contributor: Timothy Jahn, MD.)

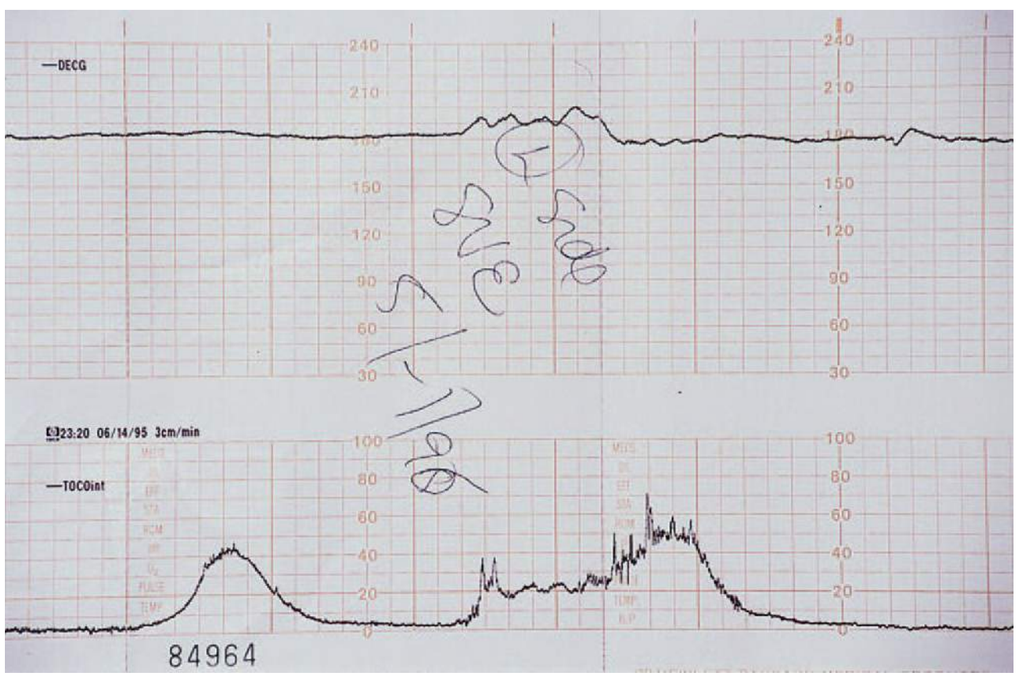


FIGURE 10.48 ■ **Loss of BBV.** Loss of beat-to-beat variability (BBV) in the fetal heart rate, which may forewarn of fetal distress. This same pattern may also be seen during a normal fetal sleep cycle or following maternal narcotic administration. (Photo contributor: Gerard Van Houdt, MD.)

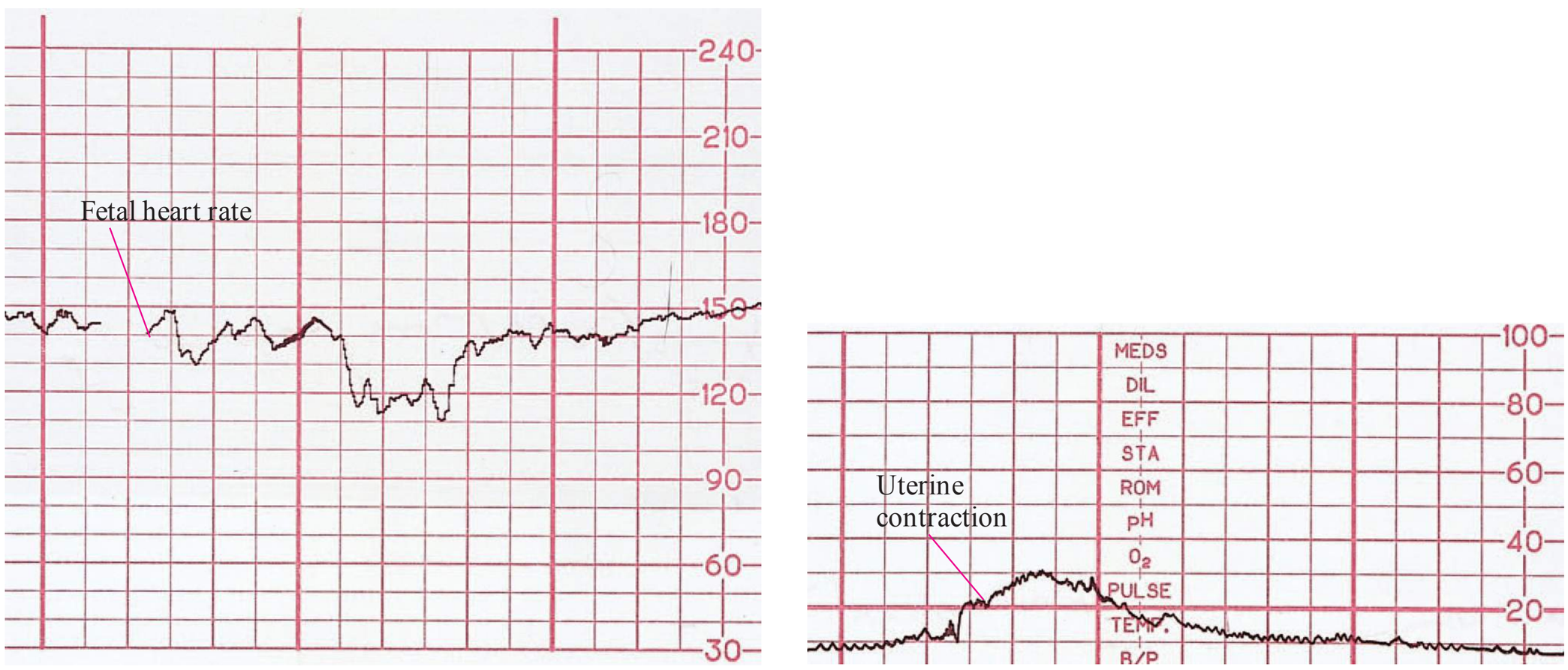


FIGURE 10.49 ■ **Late Deceleration.** The nadir of a late deceleration always follows the peak of the uterine contraction with the heart rate approaching the baseline after the completion of the uterine contraction; this is suggestive of hypoxia. (Photo contributor: James Palombaro, MD.)

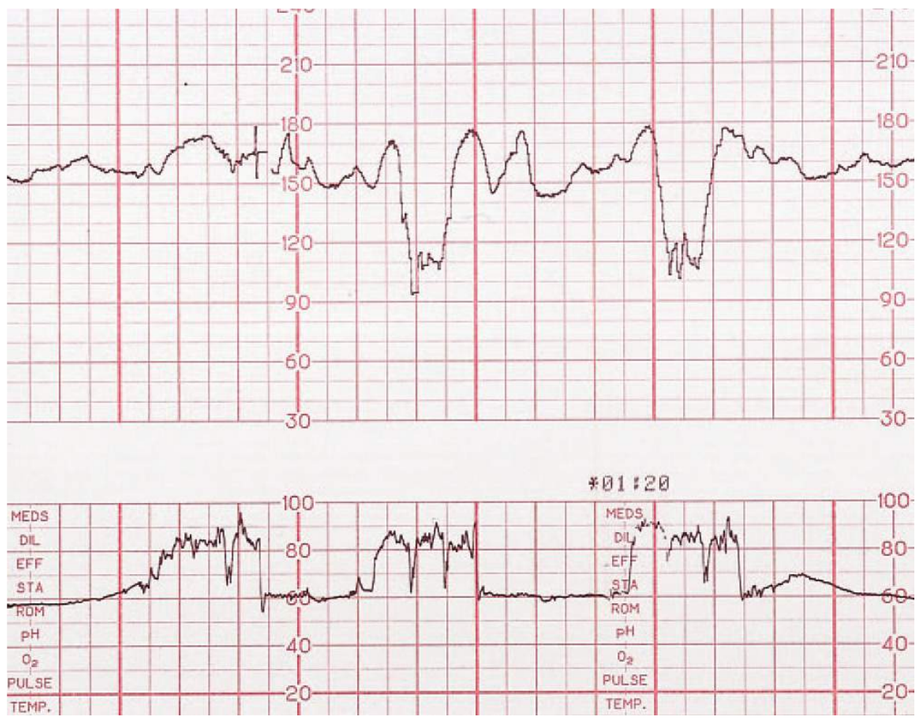


FIGURE 10.50 ■ **Variable Deceleration.** Variable decelerations are due to cord compression. They are characterized by a rapid onset and recovery and may occur slightly before, during, or after the onset of the contraction. They often include "peaks" on either side of the central deceleration. (Photo contributor: John O'Boyle, MD.)



FIGURE 10.51 ■ **Gravid Abdomen.** A third-trimester gravid abdomen with ecchymotic markings imparted by a significant blunt force. Fetal assessment should occur simultaneously with maternal resuscitation. (Photo contributor: John Fildes, MD.)

TABLE 10.2 ■ CHARACTERISTICS OF FETAL HEART RATE DECELERATIONS

Deceleration Type	Timing of Deceleration	Characteristics	Signif cance
Early	Starts after and ends before contraction	Coincides and "nadirs" with contraction peaks	Normal finding
Variable	Varies	Deep (even to <100 bpm), broad, often have "shoulders"	May indicate cord compression or may be normal finding
Late	Starts and ends after contraction	Prolonged, nadir comes after contraction peaks	Fetal hypoxia

Clinical Summary

The second stage of labor begins when the cervix is fully dilated, allowing for the gradual descent of the fetal head toward the vaginal outlet. As the head approaches the perineum, the labia begin to separate with each contraction and recede once the contraction subsides. Crowning is the term applied when the head separates the labial margins without receding at the end of the contraction. The appearance of crowning heralds imminent vaginal delivery.

Management and Disposition

Bring equipment for delivery and neonatal resuscitation to the bedside immediately. Begin continuous fetal monitoring if feasible and notify available obstetric and pediatric teams while making preparations for ED delivery. Alert the obstetric and pediatric teams to the presence of any meconium (greenish brown leakage) detected before delivery.

Support the maternal perineum with your hand and a clean, dry cloth to slow rapid fetal descent and reduce the risk of significant birth trauma to the mother. As soon as the head is delivered, check to ensure the umbilical cord is not wrapped around the fetal neck. Nuchal cord wrapping can lead to disruption of uterine blood flow during contractions, which may lead to variable decelerations in fetal heart rate and may impede delivery.

Pearls

1. Primagravida patients may still require many contractions and pushing to expel the head through the vaginal outlet; however, once crowning is present, be prepared for imminent delivery regardless of gravida status.
2. A nuchal cord should be pulled over the child's head where possible. If impeding delivery, it can be clamped and ligated on the perineum, followed by the immediate delivery of the shoulders and body.
3. If meconium secretions are detected well before delivery, continuous electronic toco fetal monitoring should be begun and the obstetric and pediatric consultants notified.
4. Avoid strong traction on the cord during or after delivery as this can tear the placenta and result in fetal or maternal hemorrhage.



FIGURE 10.52 ■ **Crowning.** Descent of the fetal head with separation of the labia is known as crowning and heralds imminent vertex delivery. (Photo contributor: William Leininger, MD.)

Clinical Summary

A gravid woman may present in any stage of labor. Those with regular forceful contractions and the urge to push are in active labor and may be near delivery. Crowning may be present and heralds imminent vaginal delivery. Important historical questions include the number of previous pregnancies, a diagnosis of twin/multiple gestations, and whether there is a history of prenatal care or complications. The presence of meconium (greenish brown fetal stool) is associated with fetal hypoxia and is a clinical indicator of fetal distress. Associated signs of fetal distress such as fetal bradycardia or late decelerations may also be present.

Management and Disposition

Immediately obtain maternal intravenous access, start oxygen, and prepare equipment for delivery and neonatal resuscitation (suction, oxygen, warming light, etc) for the impending delivery. Notify both obstetrics and pediatrics consultants of imminent ED delivery.

Delivery of the Head

As the point of the head passes through the vaginal outlet, extension of the head occurs, followed by the appearance of the face. Extension and delivery of the fetal head can be facilitated by applying gentle pressure upward on the chin through the perineum (modified Ritgen maneuver). Simultaneously elevate the scalp to help extend the head with the fingers of the other hand. Once the head has been delivered, the occiput promptly rotates toward a left or right lateral position. At this stage, sweep the infant's neck to detect the presence of the umbilical cord. If a nuchal cord is identified, slip it over the infant's head. If the cord is wrapped too tightly, it can be clamped and ligated on the perineum, followed by the immediate delivery of the shoulders and body.

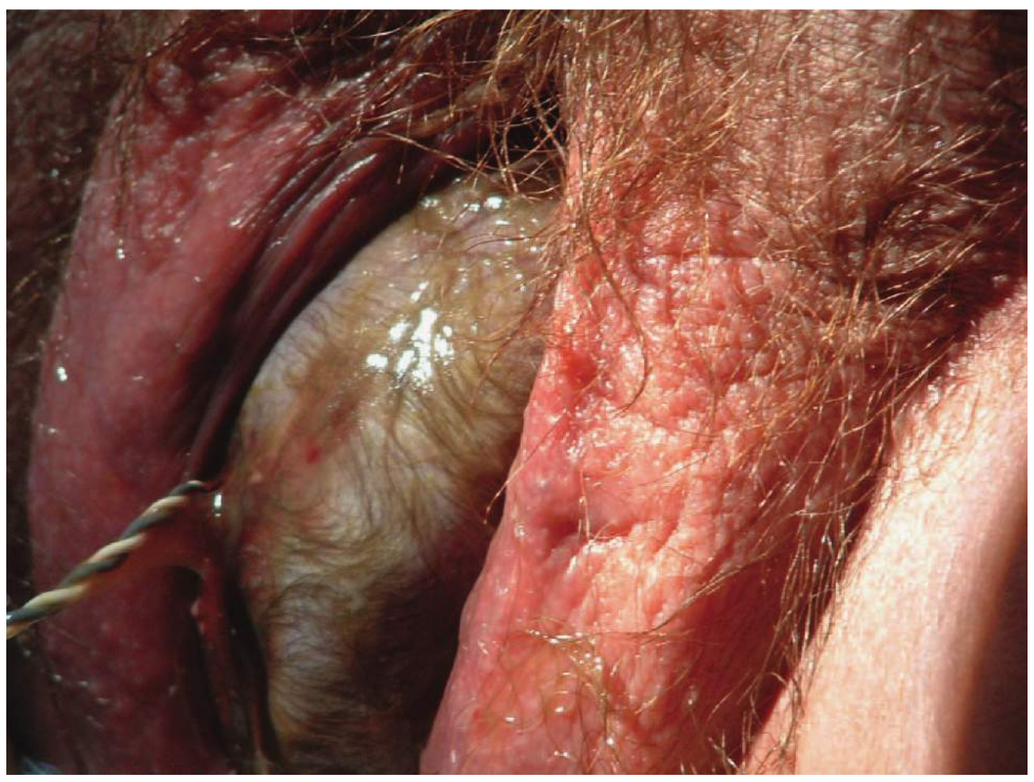


FIGURE 10.53 ■ **Meconium.** Meconium (greenish brown fetal stool), seen covering the scalp and perineum, is associated with fetal hypoxia and is a clinical indicator of fetal distress. Fetal bradycardia or late decelerations may be present and are also evidence of fetal distress. (Photo contributor: William Leininger, MD.)



FIGURE 10.54 ■ **Modified Ritgen Maneuver.** Modified Ritgen maneuver: upward pressure is applied on the fetal chin through the perineum. (Photo contributor: William Leininger, MD.)

Delivery of the Shoulders

Before delivery of the shoulders, suction the nasopharynx with a bulb syringe to clear away blood or debris. If thick



FIGURE 10.55 ■ **Anterior Shoulder Delivery.** Delivery of the anterior shoulder is facilitated with gentle downward traction. (Photo contributor: William Leininger, MD.)

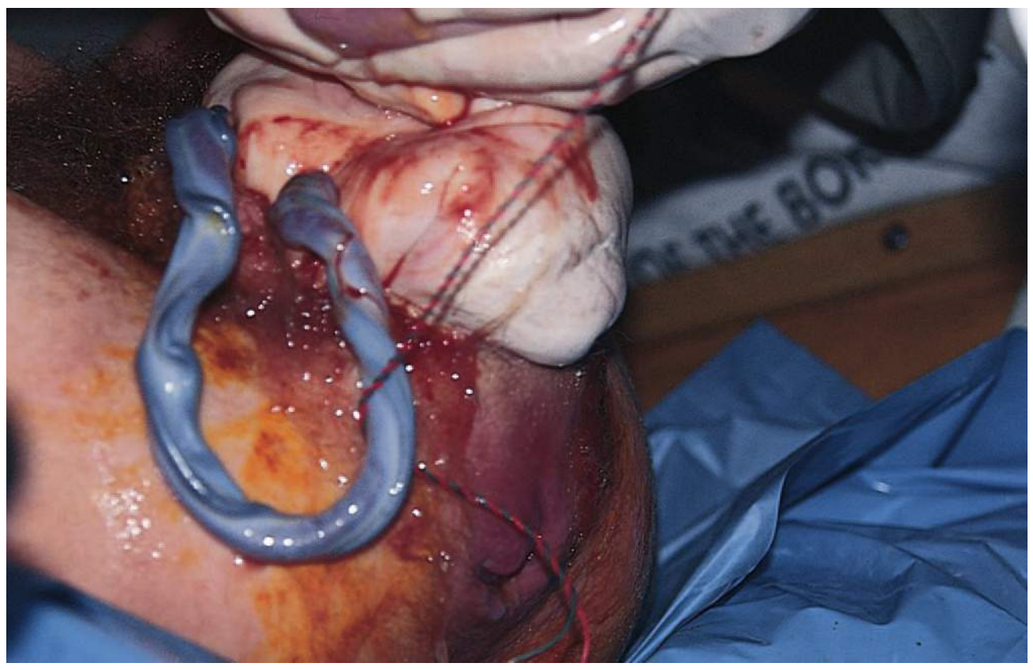


FIGURE 10.56 ■ **Posterior Shoulder Delivery.** Delivery of the posterior shoulder with gentle upward traction. (Photo contributor: William Leininger, MD.)



FIGURE 10.57 ■ **Clamping the Cord.** The cord is clamped immediately after delivery. (Photo contributor: William Leininger, MD.)

meconium is present, use a mechanical suction trap to obtain deeper and more thorough suctioning of the posterior pharynx and glottic region. Aspiration of thick meconium can lead to pneumonitis and hypoxia in the neonate. Delivery of the shoulders generally occurs spontaneously with little manipulation. Occasionally, gentle downward traction applied by grasping the sides of the head with two hands eases the delivery of the anterior shoulder. The head can then be directed upward to permit the delivery of the posterior shoulder. Following delivery of both shoulders, the body and legs are easily delivered. Attention is then directed toward the immediate care of the newborn. The cord is doubly clamped and ligated and inspected for three vessels: two umbilical arteries and one umbilical vein. The pediatrician should be notified of a two-vessel umbilical cord. The newborn is immediately placed under a warming lamp for drying and gentle stimulation while being observed for signs of distress (heart rate <100, limp muscle tone, poor color, or weak cry).

Delivery of the Placenta

Following delivery, gentle traction can be placed on the cord while your opposite hand is used to firmly massage the uterine fundus. The placenta is generally delivered within 20 minutes and should be grossly inspected for evidence of a missing segment or satellite placenta. Notify the obstetrician of these findings as retained placental fragments may warrant manual exploration of the uterus, especially with persistent postpartum bleeding. Unusual placental vasculature, such as a velamentous placenta, should also be communicated to obstetrician and pediatric consultants.



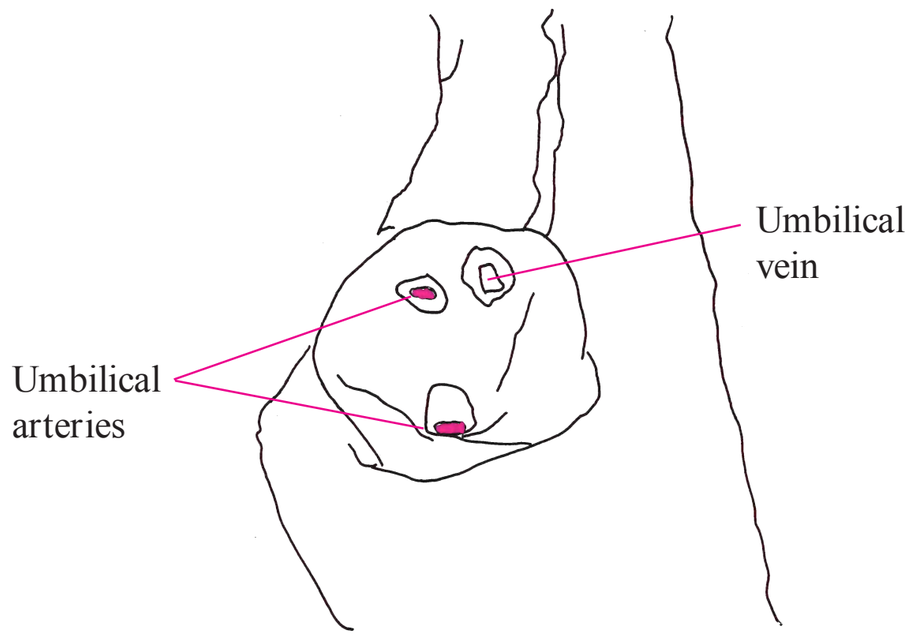


FIGURE 10.58 ■ **Normal Umbilical Cord.** Cross-sectional view of the two arteries and single vein of a normal three-vessel umbilical cord. (Photo contributor: Jennifer Jagoe, MD.)

Pearls

1. Maternal oxygen administration may help to alleviate fetal distress as seen on heart rate monitoring (see Third-Trimester Blunt Abdominal Trauma).
2. Nuchal cord occurs in about 20% of all deliveries. Occasionally two coils are identified.
3. A two-vessel cord (rare) is associated with an increased incidence of congenital defects.
4. Retained placental fragments may cause postpartum hemorrhage or endometritis.

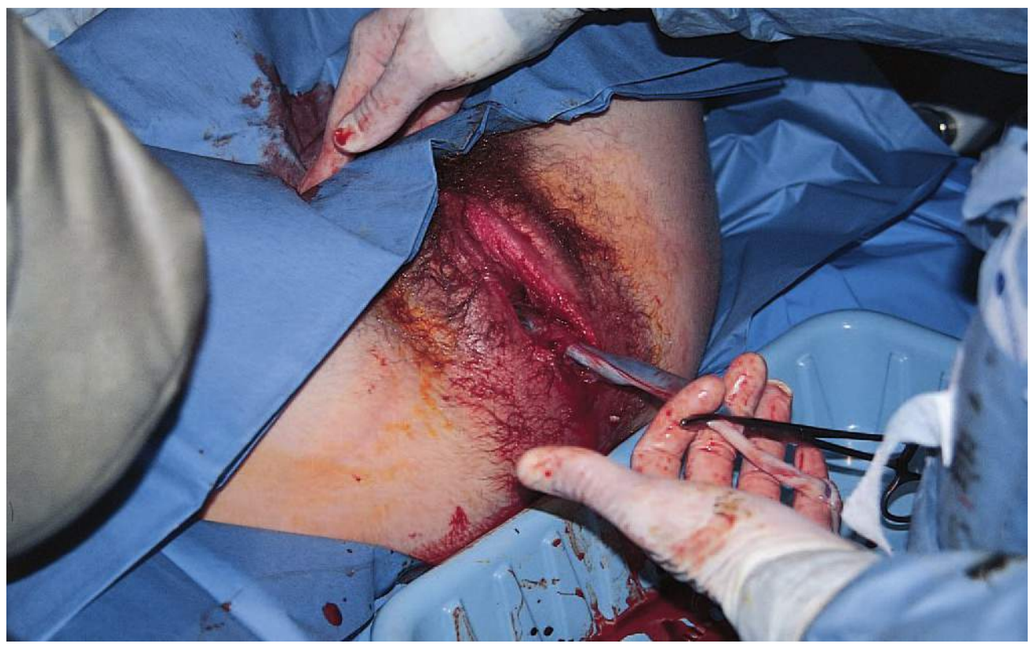


FIGURE 10.59 ■ **Placenta Delivery.** Gentle traction is applied to the cord while the opposite hand massages the uterus. (Photo contributor: William Leininger, MD.)



FIGURE 10.60 ■ **Placenta Delivery.** Delivery of the placenta. (Photo contributor: William Leininger, MD.)

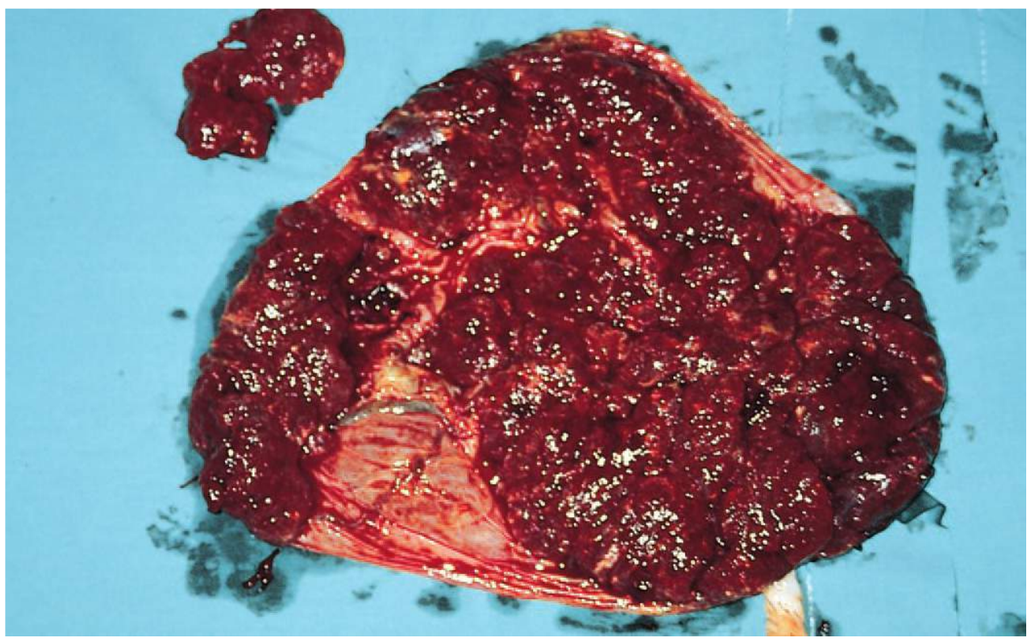


FIGURE 10.61 ■ **Placenta.** Placenta with a missing segment or cotyledon. The missing placental tissue can be seen in the upper left-hand corner of the photograph. (Photo contributor: John O'Boyle, MD.)

Clinical Summary

The incidence of singleton breech presentation is low (~3%) but increases (>20%) in preterm infants weighing less than 2 kg. In a frank breech, both hips are flexed and both knees extended. In a complete breech, both hips and knees are flexed. In both of these, the infant's buttocks are the usual presenting body part at the perineum. A footling breech has one or both legs extended below the buttocks and a foot is the presenting part. Frank breech is most common in full-term deliveries, whereas footling presentation can be found in up to half of all preterm deliveries. Breech deliveries carry a much higher neonatal mortality rate than normal deliveries. Complications of breech delivery include umbilical cord prolapse, nuchal arm obstruction, and difficulty in delivery of the head.

Management and Disposition

The specific maneuvers for breech extraction are beyond the scope of this text. If breech delivery appears imminent, prepare for ED delivery as in the previous section. Obtain immediate obstetric and pediatric consultation in all breech deliveries. During delivery, support the infant's parts and apply gentle traction as they spontaneously pass through the vaginal outlet. Keep in mind that the head diameter is greater than either the hip or shoulder diameter and may become entrapped by the cervix if it is not adequately dilated.

Pearl

1. Whenever possible and delivery is not imminent, immediately transfer stable breech laboring patients to an appropriate labor and delivery setting.



FIGURE 10.62 ■ **Breech Delivery.** Footling breech vaginal delivery of the following head. (Photo contributor: John O'Boyle, MD.)

UMBILICAL CORD PROLAPSE IN EMERGENCY DELIVERY

Clinical Summary

In an overt cord prolapse, a loop of umbilical cord is visualized either at the introitus or on sterile speculum examination following membrane rupture. Alternatively, a small loop of cord may be palpated at the cervical os. In a "funic" cord prolapse, a loop of umbilical cord is palpated directly through intact fetal membranes. Occult prolapse occurs when the umbilical cord descends between the presenting part and the lower uterine segment, but is not visible or palpable on examination. Intermittent compression of the umbilical cord with each uterine contraction may be detected by the presence of variable decelerations of the fetal monitor. Fetal hypoxia may ensue if cord compression is sustained beyond the duration of the contraction, which often happens with overt prolapse.

Management and Disposition

Prolapse of the umbilical cord presents an immediate threat to the fetal circulation and constitutes a true obstetrical emergency. If an overt prolapse is detected in the ED, do not attempt vaginal delivery. Immediately place the patient in a knee-chest position and apply continuous upward pressure with the examining hand to relieve the pressure of the presenting part on the lower uterine segment and cord. Immediately consult an obstetrician and transport the patient directly to the operating room for cesarean delivery. Maintain continuous upward pressure to the presenting part of the fetus at all times during transport. Resuscitative equipment should be available in anticipation of a hypoxic infant. If a funic prolapse is appreciated in the ED, notify an obstetrician and prepare the patient for cesarean delivery. Rupture of intact membranes may cause or worsen cord prolapse, and should never be performed in cases of suspected cord prolapse.

Pearl

1. Pelvic examination to exclude umbilical cord prolapse should be performed immediately following rupture of membranes, the appearance of new variable decelerations, or the detection of fetal bradycardia.



FIGURE 10.63 ■ **Umbilical Cord Prolapse.** Prolapsed umbilical cord visible at the vaginal introitus in a patient with twin gestations. (Photo contributor: Kevin J. Knoop, MD, MS.)

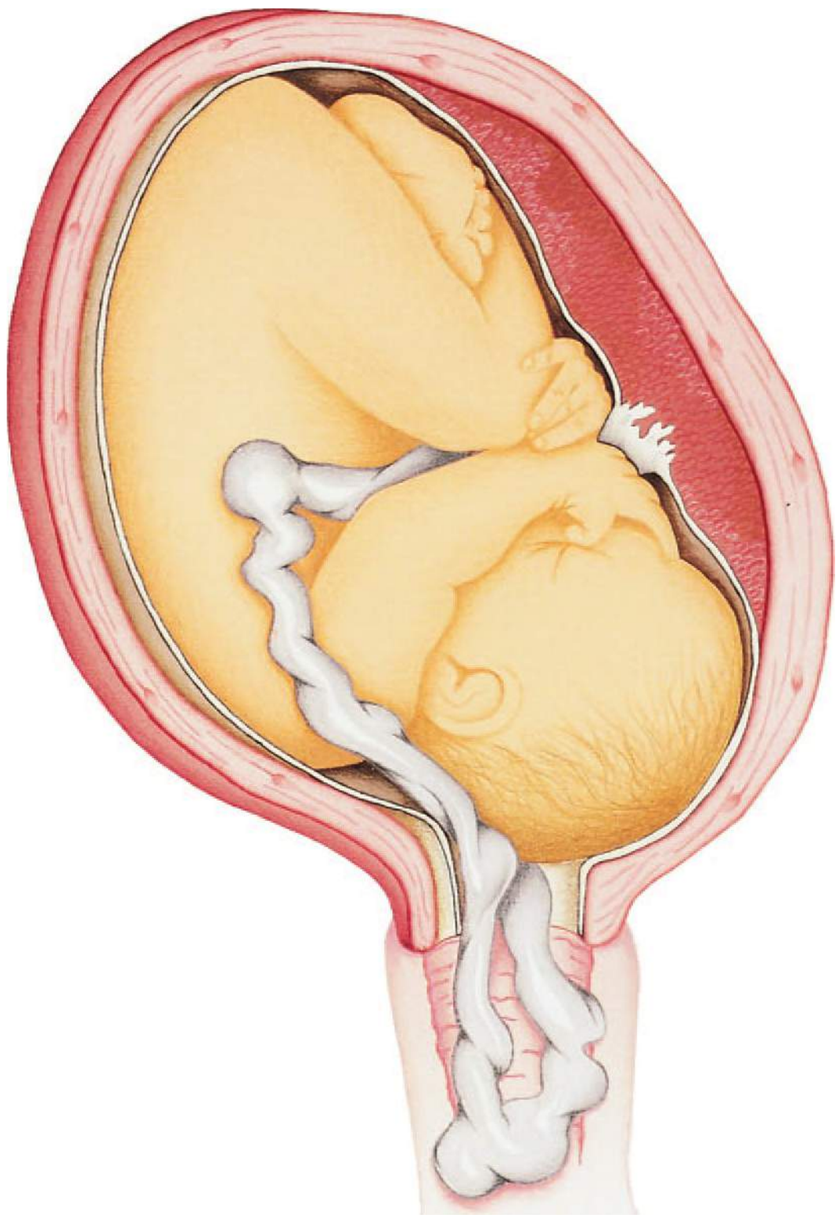


FIGURE 10.64 ■ **Umbilical Cord Prolapse.** Schematic drawing of an overt prolapse of the umbilical cord through a partially dilated cervical os. (Photo contributor: Judy Christensen.)

Clinical Summary

The circumferential wrapping of the umbilical cord around the child's neck occurs in about 20% of all deliveries. Tight approximation of the cord around the infant's neck can lead to transient disruption of uterine blood flow during contractions, leading to variable decelerations noted on the fetal heart rate monitor (Fig. 10.50); it may also impede delivery once the head passes through the introitus.

Emergency Department Treatment and Disposition

Once a cord is identified around the neck, it should be slipped over the head using the index and middle fingers. Occasionally two coils are identified.

Pearl

1. A loosely applied cord should be pulled over the child's head. If the cord is wrapped too tightly, it can be clamped and ligated on the perineum, followed by the immediate delivery of the shoulders and body.



FIGURE 10.65 ■ **Nuchal Cord.** A loose nuchal cord is seen around the neck. (Photo contributor: William Leininger, MD.)

SHOULDER DYSTOCIA IN EMERGENCY DELIVERY

Clinical Summary

Shoulder dystocia is defined as failure to deliver the fetal shoulders following delivery of the head. Dystocia is caused by impaction of the fetal shoulders against the pelvic outlet. Often, it is diagnosed by retraction of the fetal head against the perineum after delivery ("turtle sign"). Risk factors include gestational diabetes, prior shoulder dystocia or delivery of large infants, and postterm delivery.

Management and Disposition

Shoulder dystocia is an acute obstetric emergency with the immediate threat being fetal asphyxia from compression of cord and chest. Request emergent obstetric and pediatric consultation. Prepare equipment for delivery, possible episiotomy, and neonatal resuscitation. Upon identification of a shoulder dystocia, attempt McRobert maneuver (the least invasive procedure). This maneuver involves flexing the mother's knees toward and past her chest in an extreme dorsal lithotomy position while simultaneously applying firm suprapubic pressure in an attempt to disengage the infant's shoulder from the pelvic rim. Do not apply fundic pressure as this may worsen the fetal lodging against the pubic bone. If McRobert maneuver is unsuccessful, a Woods screw maneuver can be attempted by hooking two fingers behind the infant's posterior scapula and rotating the entire body. As the anterior shoulder rotates downward, it can generally be delivered past the symphysis pubis. If the Woods maneuver fails to deliver the anterior shoulder, delivery of the posterior arm may be attempted by inserting two fingers into the sacral fossa and delivering the entire posterior arm by flexing it at the elbow. The remaining shoulder should then deliver spontaneously or following rotation into the oblique position to facilitate its delivery. In extreme cases, emergency caesarian may be necessary.

Pearls

1. Shoulder dystocia is an acute obstetric emergency that requires quick action.
2. After delivery, look for fractures of the infant's clavicle or humerus and evidence of a brachial plexus injury. Damage to the fifth and sixth cervical nerve roots with resultant Erb palsy is the most common injury.



FIGURE 10.66 ■ **Shoulder Dystocia.** Firm approximation of the fetal head against the vaginal outlet consistent with shoulder dystocia. (Photo contributor: William Leininger, MD.)

Clinical Summary

Lacerations to the perineum commonly occur following a rapid, uncontrolled delivery of the fetal head. Postpartum perineal lacerations range from minor to severe and are categorized into four groups. First-degree lacerations are limited to the mucosa, skin, superficial subcutaneous, and submucosal tissues. Second-degree lacerations penetrate deeper into the superficial fascia and transverse perineal musculature. Third-degree lacerations extend through deeper tissues and disrupt the anal sphincter, while fourth-degree lacerations extend through rectal mucosa. These injuries can be associated with significant maternal blood loss and multiple long-term complications.

Management and Disposition

In ED deliveries, the repair of the episiotomy and/or perineal lacerations can be deferred to an obstetrician, the details of repair being beyond the scope of this book. If maternal blood loss is significant, apply pressure to control bleeding while awaiting definitive repair.

Pearls

1. Warm perineal compresses and manual perineal support reduce the risk of significant vaginal trauma from birth.
2. Perineal laceration repair fundamentally involves the sequential anatomic reapproximation of the rectal mucosa, anal sphincter, transverse perineal musculature, vaginal mucosa, and skin using absorbable suture material.

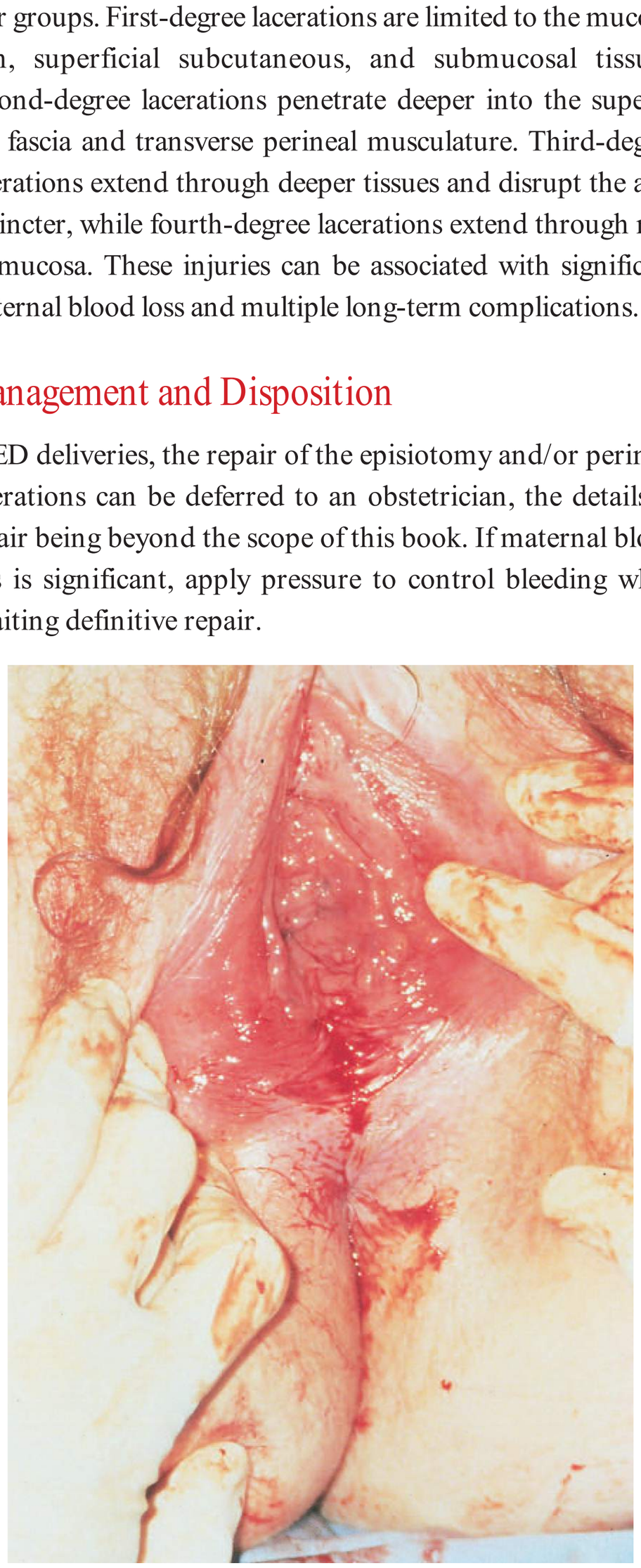
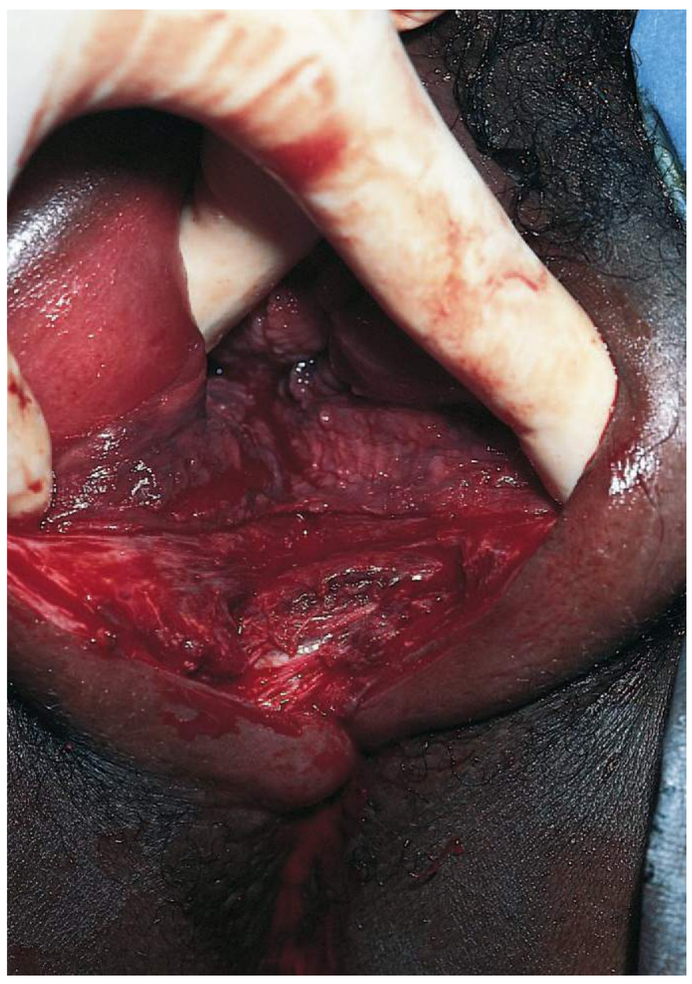


FIGURE 10.67 ■ **First-Degree Laceration.** First-degree laceration limited to the mucosa, skin, and superficial subcutaneous and submucosal tissues. There is no involvement of the underlying fascia and muscle. (Photo contributor: Jerry Van Houdt, MD.)



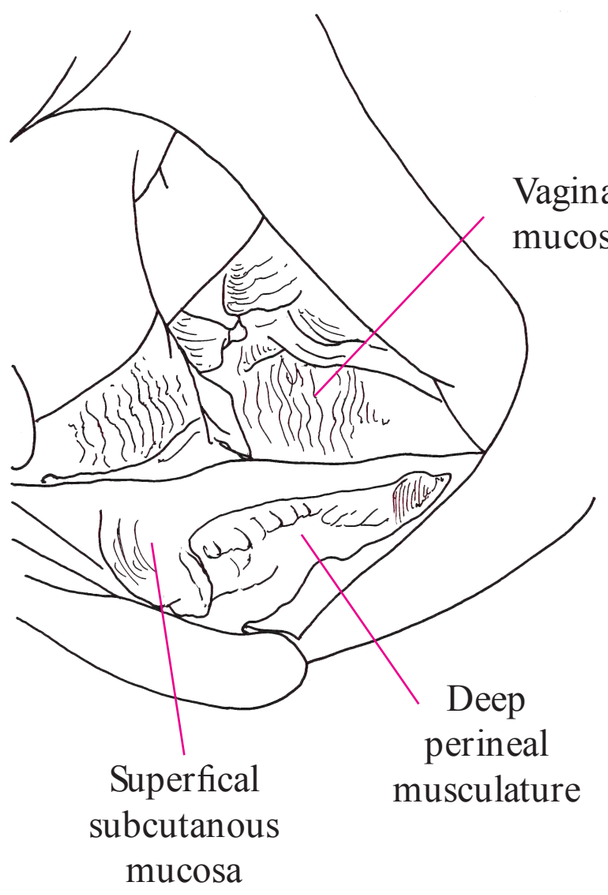


FIGURE 10.68 ■ **Second-Degree Laceration.** There is disruption of the hymenal ring and the deep perineal musculature, extending into the vaginal mucosa and transversalis fascia, but no involvement of the anal sphincter or mucosa. (Photo contributor: Pamela Ambroz, MD.)



FIGURE 10.69 ■ **Fourth-Degree Laceration.** Fourth-degree perineal laceration revealing wide separation of the perineal fascia and anal sphincter. The examiner's small finger is in the rectal lumen, showing extension of the tear proximally. (Photo contributor: Timothy Jahn, MD.)

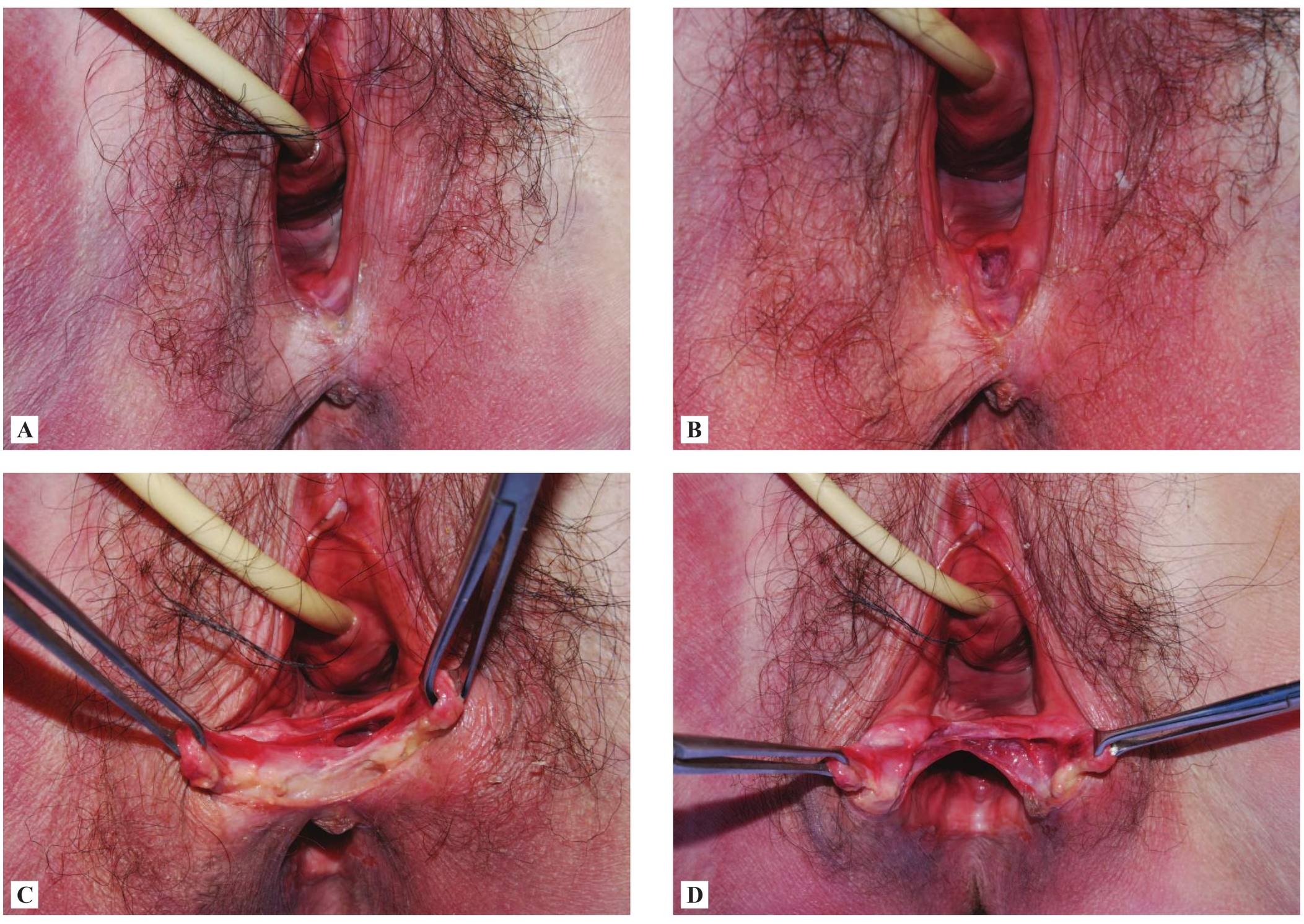


FIGURE 10.70 ■ **Postpartum Perineal Lacerations.** These cadaveric photographs illustrate the defining anatomy for first- through fourth-degree perineal lacerations. (**A**) First, (**B**) second, (**C**) third, and (**D**) fourth. (Photo contributor: Barbara Hoffman, MD.)

Clinical Summary

Intrauterine fetal demise (IUFD) refers to fetal death prior to delivery and may occur at any time prior to delivery. The timing of the IUFD is important for the ED physician to appreciate both for consoling and advising the parents and for anticipating the likelihood of successful neonatal resuscitation after delivery. The appearance of the fetus at delivery can be used to estimate whether the fetal demise occurred antepartum or peripartum.

The process of tissue degeneration (maceration) is due to the effects of autolytic enzymes on the fetus in a sterile environment. Lysis occurs at the epidermal-dermal junction with subtle changes in the gross appearance of the fetus (skin desquamation, positive Nikolsky sign) seen as early as 6 hours. Further changes involve desquamation and bullae formation of the face, back, or abdomen by 12 hours, at least 5% of the body surface at 18 hours, and generalized skin desquamation at 24 hours. Sloughing of skin from a large area indicates a prolonged interval between death and delivery. Mummification occurs after approximately 2 weeks.

Management and Disposition

Anticipate and prepare for a normal delivery unless information regarding the condition of the fetus is known. Obtain obstetric consultation immediately as for any emergency delivery. The presence of fetal heart tracings during delivery or a normal appearing infant without significant skin changes indicates peripartum demise and neonatal resuscitation should be attempted. If signs of maceration are present, no resuscitative efforts are indicated.

Pearl

1. Desquamation and/or sloughing of skin from large or numerous areas in an unresponsive fetus indicate antepartum IUFD.



FIGURE 10.71 ■ **Intrauterine Fetal Demise.** Desquamation of the entire skin surface, with sloughing of the abdominal and scrotal skin, is seen in this IUFD. (Photo contributor: Lawrence B. Stack, MD).

Chapter 11

EXTREMITY TRAUMA

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George J. Raukar
Daniel L. Savitt

Scan QR code or go to www.ematlas.com for video related to chapter



Degloving injury. (Photo contributor: Selim Suner, MD, MS.)

ACROMIOCLAVICULAR JOINT SEPARATION

Clinical Summary

Injury to the acromioclavicular (AC) joint usually results from an impact on the superior aspect of the acromion. The classification system for AC joint includes six types. A type I injury is equivalent to a stretching of the AC ligament. A type II injury consists of tearing of the AC ligaments and stretching of the coracoclavicular ligaments. Complete disruption of the AC and coracoclavicular ligaments is seen in types III to VI.

Patients complain of pain at the AC joint and will actively splint the injured shoulder. Ecchymosis may be present; however, an obvious deformity is not always seen. There is significant tenderness upon palpation of the AC joint.

Standard radiographs should include anteroposterior (AP) and axillary lateral views of the shoulder. Type I injuries will appear normal. Type II injuries may show 0% to 50% displacement at the AC joint but no increase in the coracoclavicular interval. Types III to VI will demonstrate displacement at the AC joint and the clavicle will appear to be displaced superiorly (the acromion actually is rotated inferiorly) 50% to greater than 100% its width when compared with the normal side.



FIGURE 11.1 ■ **AC Joint Separation.** Subtle prominence of the left distal clavicle. The upward clavicle displacement is due to suspending ligament stretching or disruption. (Photo contributor: Frank Birinyi, MD.)

Management and Disposition

Type I and type II injuries are treated with rest, ice, analgesics, and a simple sling until acute pain with movement is relieved. Treatment of type III injuries may be nonoperative or operative. However, these patients can be discharged from the emergency department in a sling without an emergent orthopedic consult. Types IV, V, and VI are treated operatively. Referral to a musculoskeletal specialist is essential for all AC joint injuries since many patients who initially appear to have minor injuries will have more obvious deformity after the swelling and pain have subsided.

Pearls

1. The early AC joint stress radiograph can be negative due to splinting of the shoulder girdle muscles and does not add anything to the acute patient management.
2. Differentiating between types I and II versus types IV to VI is the goal of the ED physician, since the latter will require an emergency orthopedic consult.

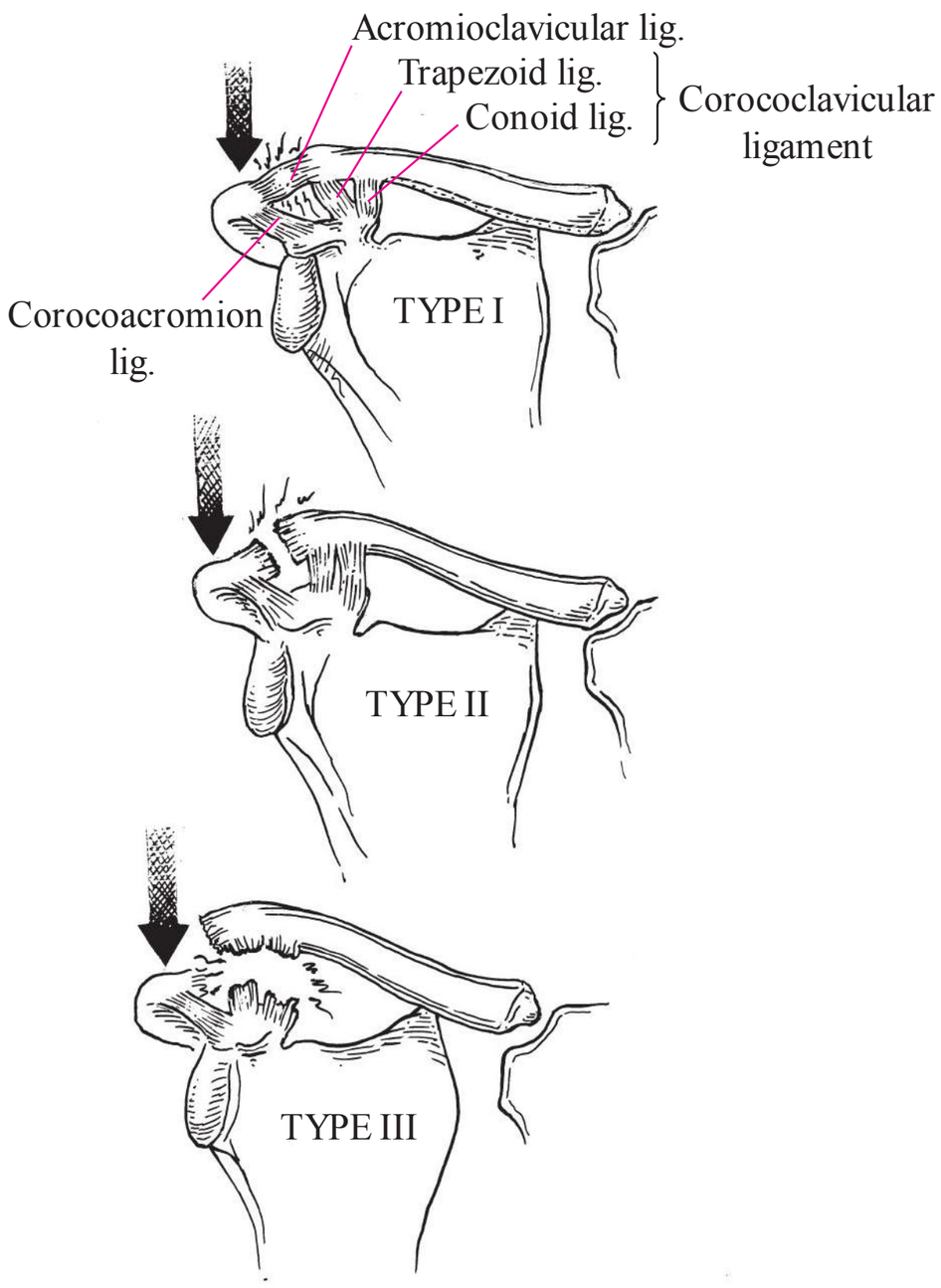


FIGURE 11.2 ■ **Types of AC Joint Injuries.** Classification schemes may subdivide type III injuries into III through VI depending on the position of the clavicle.

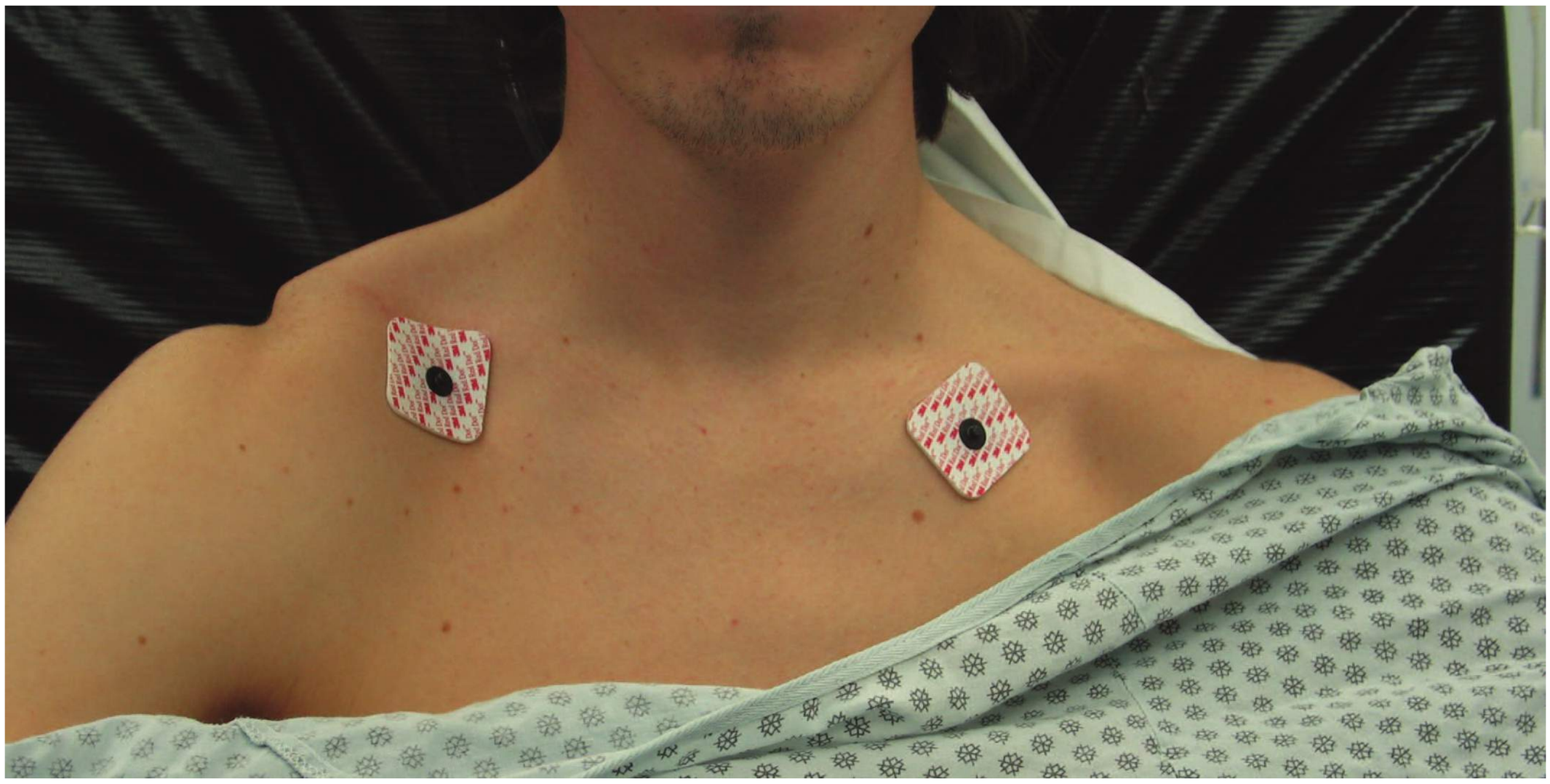


FIGURE 11.3 ■ **AC Joint Separation.** Large deformity at the right distal clavicle suggesting complete ligament disruption. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 11.4 ■ **AC Joint Separation, Third Degree.** A wide AC joint and the clavicle displaced from the acromion. (Reproduced with permission from Simon RR, Sherman SC, Koenigsknecht SJ. Emergency Orthopedics: The Extremities. New York: McGraw-Hill; 2007: p 297, Fig. 11-54.)

SHOULDER DISLOCATION

Clinical Summary

Anterior shoulder dislocations are the most common and frequently caused by falling with the arm externally rotated and abducted. Patients present with the affected extremity held in adduction and internal rotation. Often, they complain of shoulder pain, refuse to move the affected arm, and may support the dislocated shoulder with the other arm. The acromion becomes prominent with loss of the rounded contour of the deltoid. A neurovascular examination of the upper extremity should be performed to rule out associated injury, most commonly of the axillary nerve (sensation over the deltoid) and of the musculocutaneous nerve (sensation on the anterolateral forearm). Vascular injuries have rarely been reported to occur. Standard radiographic examination to evaluate for associated fracture should include AP and either axillary lateral or scapular Y views.

Posterior shoulder dislocations are commonly missed because of subtle radiographic findings. The arm is held internally rotated and slightly abducted. Patients are unable to externally rotate their shoulder. On examination, a posterior prominence exists. Posterior dislocations can occur with a posterior-directed force as seen during grand mal seizures or electric shock.

The Hill-Sachs lesion (an impaction of the humeral head) can occur in up to 50% of patients.



FIGURE 11.5 ■ **Anterior Shoulder Dislocation.** This right anterior shoulder dislocation occurred when the patient fell while playing basketball. There is an obvious contour deformity as well as prominence of the acromion. (Photo contributor: Kevin J. Knoop, MD, MS.)

Management and Disposition

Closed reduction is the treatment for all types of shoulder dislocations and should be completed as soon as possible to avoid avascular necrosis to the humeral head. Due to shoulder girdle spasm, conscious sedation is often required. There are many methods to reduce anterior shoulder dislocations, including Stimson, Rockwood traction and countertraction, and Milch. The basic premise is to apply axial traction, externally rotate, and abduct. Scapular manipulation and the Stimson method are successful techniques, especially in the thin patient. Neurovascular and radiographic examination should occur before and after reduction. The patient should be placed in a sling after reduction and follow-up with a musculoskeletal specialist should be recommended.

Pearls

1. Occult, nondisplaced greater tuberosity fracture can be identified on postreduction radiographs. These usually occur during the traumatic event but not seen on initial radiographs. They are not the result of the reduction and usually only identified when there is no bony overlap on the x-rays.

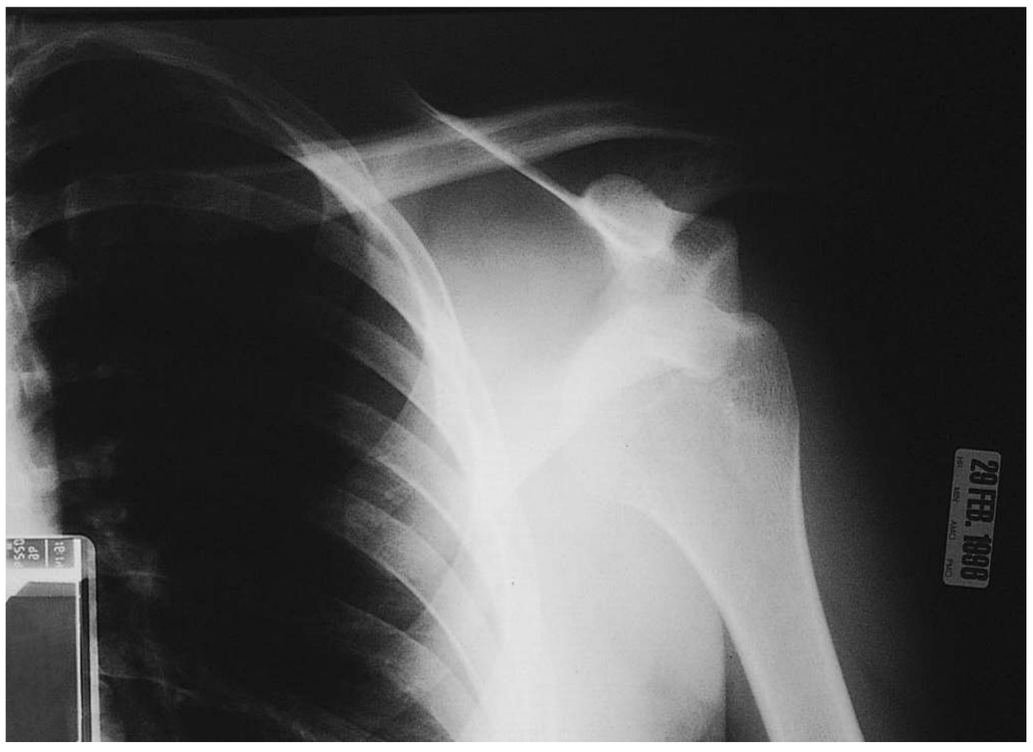


FIGURE 11.6 ■ **Anterior Shoulder Dislocation.** Radiographic evaluation demonstrates that the humeral head is not in the glenoid fossa but is located anterior and inferior to it. (Photo contributor: Kevin J. Knoop, MD, MS.)

2. Luxatio erecta is inferior glenohumeral dislocation that is rarely encountered. The humeral head is forced below the inferior aspect of the glenoid fossa due to a hyperabduction of the arm. These patients present with the arm locked fully abducted and externally rotated. Axillary nerve injury is reported to occur in 60%. Vascular injury occurs most frequently with this type of dislocation. Reduction is accomplished with overhead traction.

3. Posterior shoulder dislocations can be bilateral and are often missed due to preserved symmetry on standard chest x-rays.

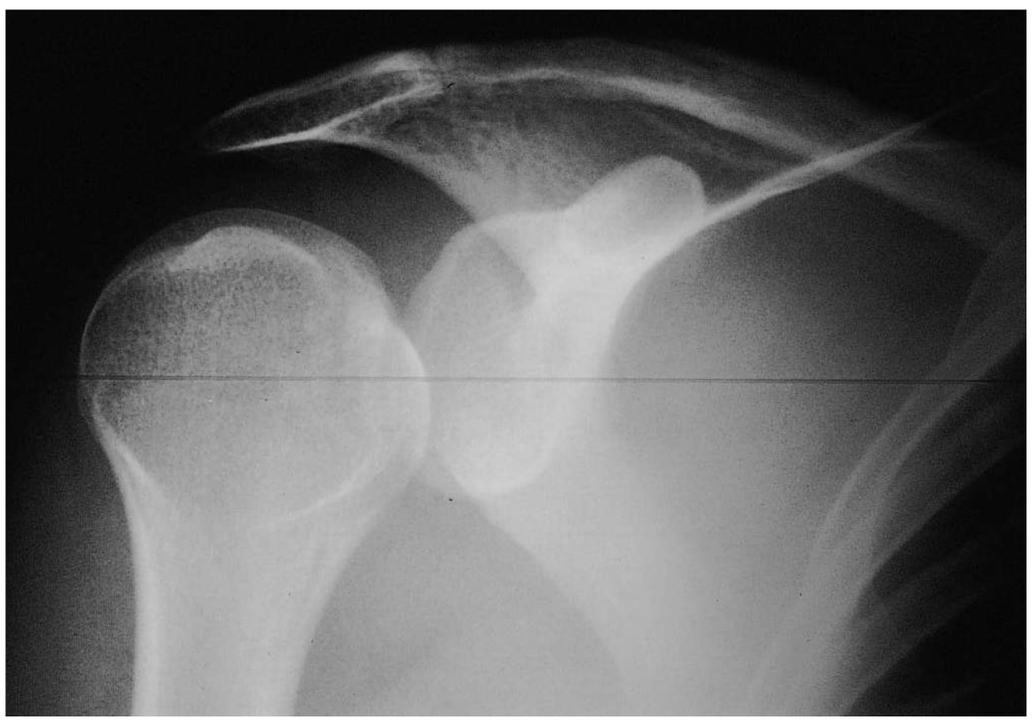


FIGURE 11.7 ■ **Posterior Shoulder Dislocation.** AP radiograph of this rare type of shoulder dislocation. Because of internal rotation of the greater tuberosity, the humeral head appears like a dip of ice cream on a cone, thus called the "ice cream cone sign." (Photo contributor: Alan B. Storrow, MD.)

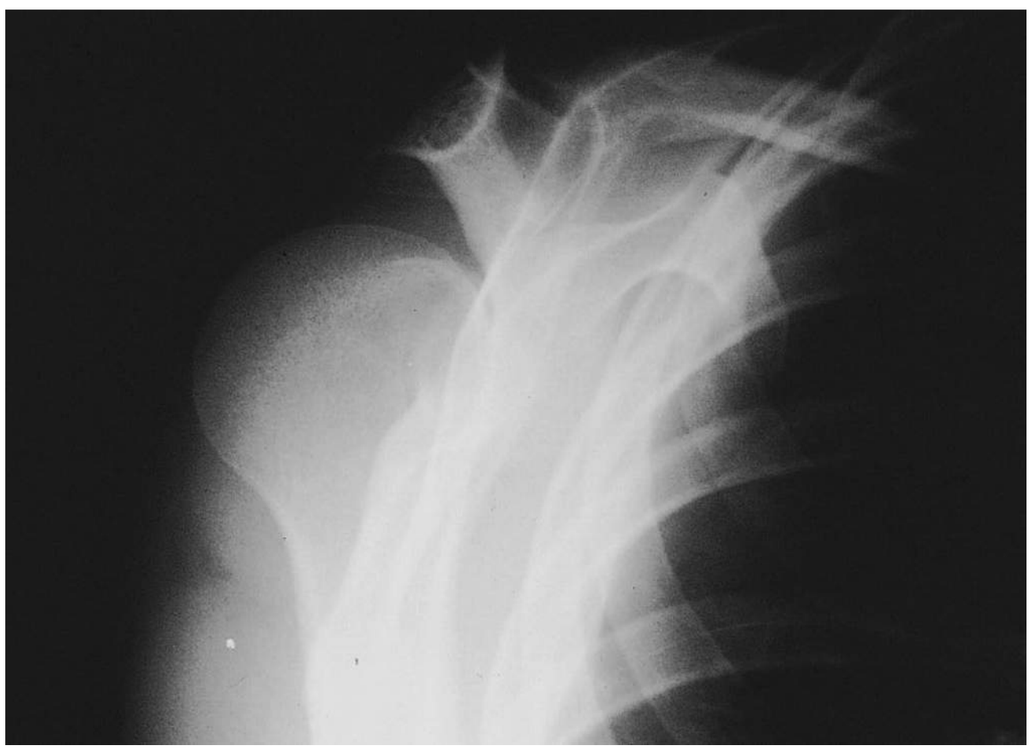


FIGURE 11.8 ■ **Posterior Shoulder Dislocation.** A scapular Y view of the same patient in Fig. 11.7 confirms the diagnosis. (Photo contributor: Alan B. Storrow, MD.)

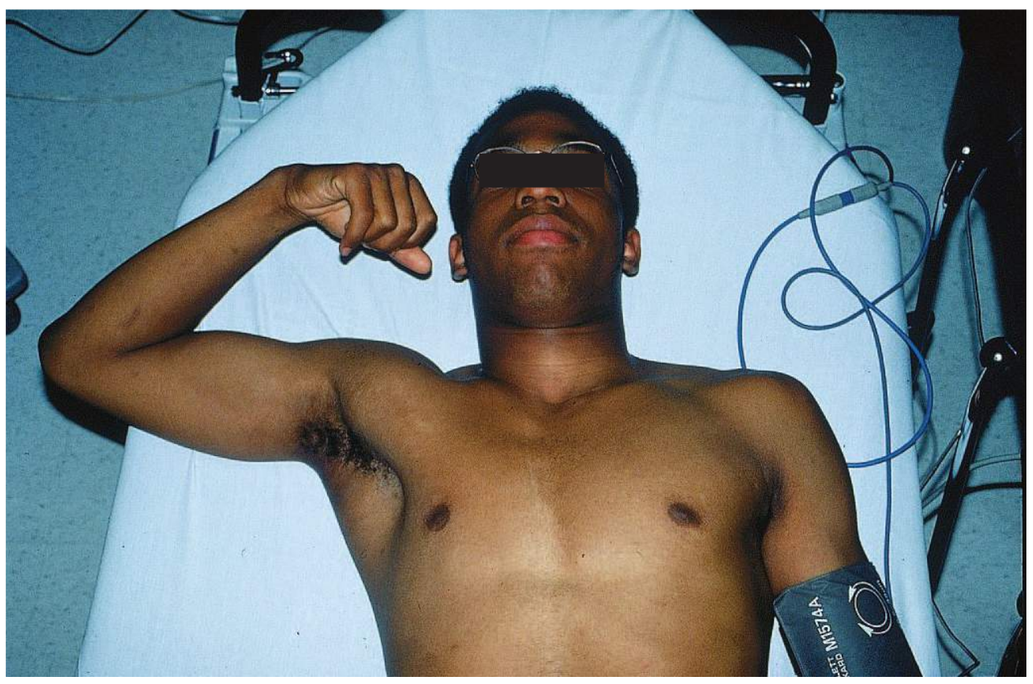


FIGURE 11.9 ■ **Luxatio Erecta.** Hyperabduction may cause the relatively rare inferior dislocation known as luxatio erecta. The arm is held in elevation and the humeral head may be palpated along the lateral chest wall. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 11.10 ■ **Luxatio Erecta.** An inferior dislocation of the humeral head with the arm above consistent with luxatio erecta. (Photo contributor: Benjamin Milligan, MD.)

ELBOW FRACTURES

Clinical Summary

Direct trauma or fall on an outstretched hand may result in elbow fractures. The patient may be unable to extend the elbow and have pain on supination/pronation. AP, lateral, and oblique radiographic views visualize most. The radial head should be aligned with the capitellum on all views. The anterior fat pad may be seen on normal radiographs but displacement anteriorly and superiorly (sail sign) suggests effusion or hemarthrosis. The posterior fat pad is not normally visualized and, if seen, is indicative of effusion or hemarthrosis; it is often associated with a radial head fracture in adults.

Supracondylar fractures often occur in pediatric patients. Neurovascular insult occurs in 7%, with the anterior interosseous nerve most commonly injured. This can be checked by having the patient make an "ok" sign. This neuropraxia usually resolves in 6 months. Arterial injury to the brachial artery occurs in approximately 5% to 20%, with less than 1% of these complicated by compartment syndrome. Arteriography is indicated if the radial pulse is decreased after reduction; however, it should not delay operative evaluation.

Capitellum fractures represent less than 1% of adult elbow fractures, occur with high-energy trauma, and are difficult to diagnose radiographically; a CT scan may be required. This fracture is commonly associated with fractures of the radial head.

Management and Disposition

Treatment of supracondylar fractures is influenced by stability of the fracture pattern as well as associated neurovascular injuries. If neurovascular compromise exists, the emergency physician may need to apply forearm traction to reestablish distal pulses. If the pulse is not restored with traction, emergent operative intervention for brachial artery exploration or fasciotomy is indicated. In children, nondisplaced fractures can be splinted in 90 degrees of flexion. Angulated or displaced fractures often require operative intervention.

Capitellum fractures are treated with immobilization in a posterior long-arm splint with the elbow in 90 degrees flexion and the forearm in supination. Complications of displaced capitellum fractures include arthritis, avascular necrosis, and decreased range of motion.

Radial head fractures can be treated with a sling for comfort and patients should be told to discontinue the sling as early as possible. Fractures that are greater than 2 mm displaced, those that have greater than 20% of articular depression, or

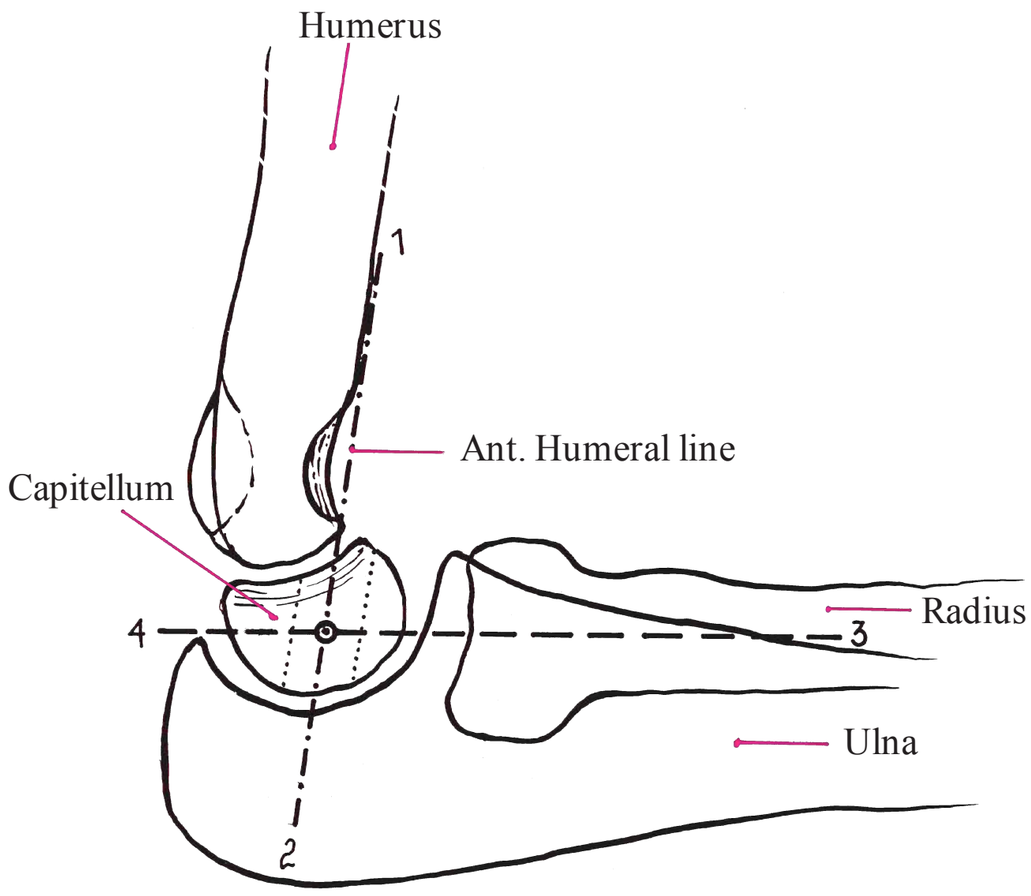


FIGURE 11.11 ■ **Radiographic Elbow Relationships.** In lateral views, the anterior humeral line (1-2) should bisect the middle third of the capitellum. The radiocapitellar line (drawn through the center of the radius, 3-4) should also pass through the center of the capitellum. Disruption of these relationships may indicate fracture.



FIGURE 11.12 ■ **Supracondylar Fracture.** This radiograph shows both a pronounced anterior fat pad (sail sign) and posterior fat pad indicative of a supracondylar fracture. (Photo contributor: Alan B. Storrow, MD.)

open fractures may need operative treatment and should follow up within a week with an orthopedic surgeon.

Pearls

1. An intimal arterial injury may not be initially apparent and frequent radial artery checks are warranted in the pediatric population. Some practitioners opt to admit supracondylar fractures so these neurovascular checks can be done.
2. Capitellum and radial head fractures often occur together.
3. The presence of a joint effusion or a posterior fat pad with a history of trauma is presumptive evidence of a fracture.

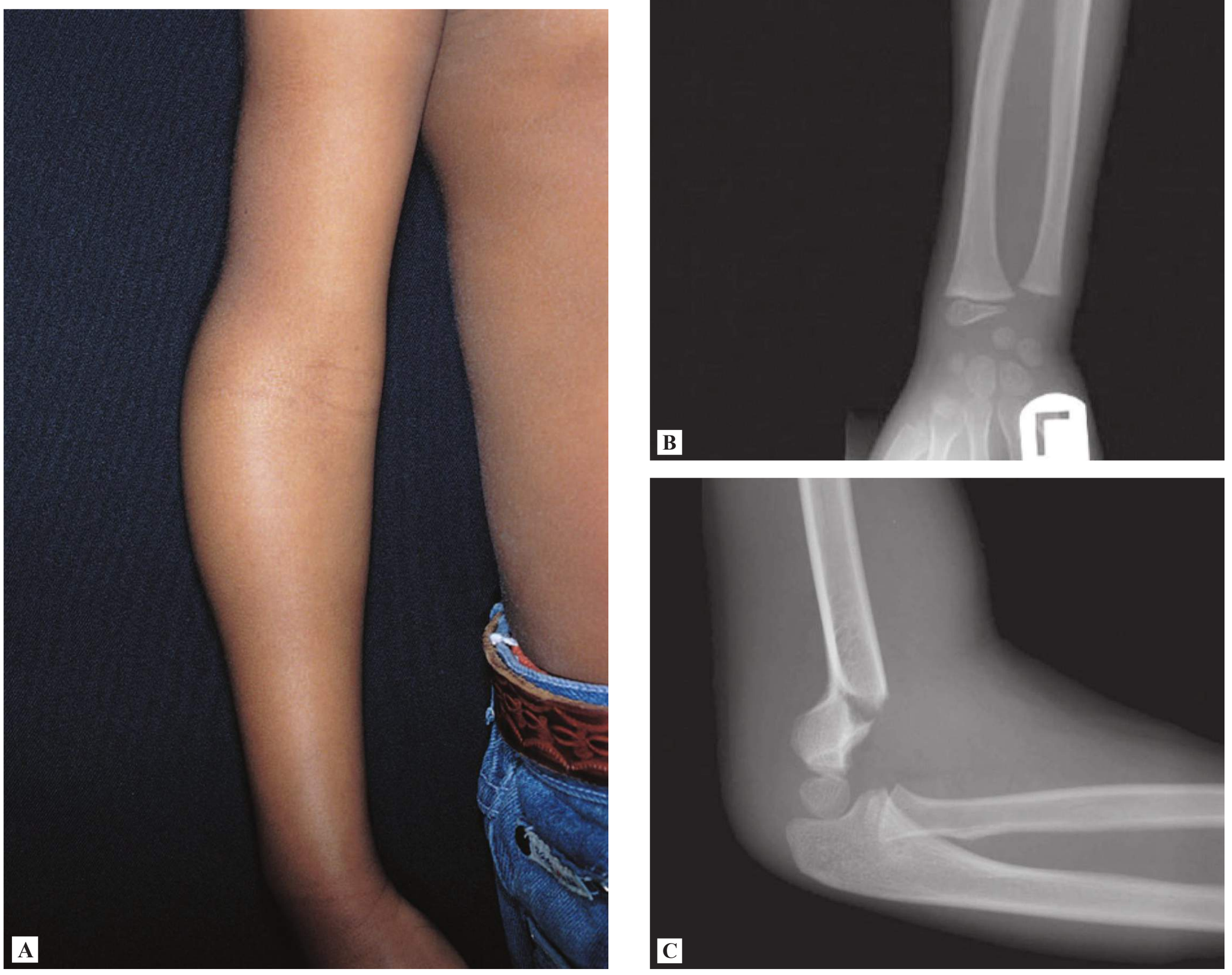


FIGURE 11.13 ■ **Supracondylar Fracture.** (**A**) Clinical picture with marked soft tissue swelling in a child with a history of falling on an outstretched arm. (**B** and **C**) AP and lateral views of the elbow, demonstrating a distal humerus fracture with posterior displacement of the medial and lateral condyles. (Reproduced with permission from Shah BR, Lucchesi M. Atlas of Pediatric Emergency Medicine. New York: McGraw-Hill; 2006.)

ELBOW DISLOCATION

Clinical Summary

Elbow dislocations are the second most common major joint dislocation and usually occur posteriorly, although they can be anterior, medial, or lateral. All require immediate reduction to relieve pain and prevent neurovascular compromise. Brachial artery function and ulnar, median, and radial nerve integrity must be evaluated when examining any patient. Elbow dislocations are often associated with a radial head fracture. Patients with posterior dislocation present with their elbow held in flexion and examination reveals a swollen, tender, and deformed elbow with a prominent olecranon. Anterior dislocations, though rare, present with the elbow extended with the forearm supinated and elongated.

Radiographic evaluation should include an AP and a lateral view. The presence of fractures should be noted, as this may complicate reduction.

Management and Disposition

Most patients require conscious sedation prior to reduction. Posterior dislocations are accomplished by applying posterior pressure to the humerus while an assistant applies longitudinal forearm traction. Alternatively, the patient is placed in the prone position so the humerus hangs perpendicular to the stretcher. A 5- to 10-lb weight is applied to the wrist or axial traction is applied to the wrist while the elbow at the humerus is stabilized. After a few minutes, the olecranon slips back into place. It is possible to entrap the nerve while attempting reduction, so neurovascular integrity must be checked before and after reduction. If this occurs, an orthopaedic surgeon should be consulted immediately. After successful reduction, the elbow should be immobilized in 90 degrees of flexion in a posterior splint and sling. Elbow dislocations with associated fractures may make closed reduction difficult and also leave the joint unstable. In these cases, consultation with an orthopedic surgeon is recommended prior to reduction attempts.

Pearls

1. The ulnar nerve is the most common nerve injured.
2. The olecranon should form a straight line with the two epicondyles when the elbow is extended. At 90 degrees of flexion, the olecranon and the two epicondyles should form a triangle. This relationship is disrupted in the dislocated elbow.



FIGURE 11.14 ■ **Posterior Elbow Dislocation.** This patient dislocated his elbow while playing basketball. Note the flexed position and the prominence of the olecranon. (Photo contributor: Frank Birinyi, MD.)



FIGURE 11.15 ■ **Posterior Elbow Dislocation.** Lateral radiograph demonstrating posterior elbow dislocation. (Photo contributor: Selim Suner, MD, MS.)

Clinical Summary

The biceps has two origins, crosses the shoulder and elbow joints, and inserts on the proximal radius. The long head of the biceps is much more susceptible to injury and rupture may occur anywhere along its route. Clinically, patients with proximal rupture present with pain along the anteromedial aspect of the shoulder. On inspection, ecchymosis is often acutely noted. Muscle retraction within the arm may create a "Popeye" deformity. Due to the two proximal attachments, the short head of the biceps can allow for maintenance of forearm supination strength. Rupture may also occur at the tendon insertion into the radial tuberosity at the elbow. This diagnosis is made on the basis of a history of a painful, tearing sensation in the antecubital region. A snap or pop may also occur. The ability to palpate the tendon in the antecubital fossa may indicate partial tearing.

Management and Disposition

Proximal and distal biceps tendon ruptures can be discharged with a sling, pain control, physical therapy, and referral for operative repair consideration. Distal biceps tendon ruptures often require surgical management due to the significant loss of forearm supination strength. Partial proximal biceps tendon ruptures should also be referred to a specialist.

Pearls

1. Functional deficits from a long head rupture are usually temporary and are influenced by coexistent tears.
2. Biceps rupture occurs most commonly in the dominant extremity of men between 40 and 60 years of age when an unexpected extension force is applied to the flexed arm.



FIGURE 11.16 ■ **Biceps Tendon Rupture.** The tendon is noted to contract within the arm after rupture. (Photo contributor: Daniel L. Savitt, MD.)

Clinical Summary

Fractures of the wrist and elbow usually involve a fall onto the outstretched arm, while isolated fractures of the ulnar shaft are more commonly from a direct blow. AP and lateral views of the wrist, forearm, and elbow are required when a forearm fracture is suspected. Functional deficits in the hand are important clues to identification of occult injury to forearm neurovascular structures, and could require immediate surgical intervention. The Monteggia fracture-dislocation is a fracture of the ulna (usually proximal third) with an associated proximal radial head dislocation; it is occasionally associated with radial nerve injury.

Galeazzi fracture-dislocation is a fracture of the distal one-third of the radius with dislocation of the distal radioulnar joint. It occurs three times more often than a Monteggia fracture and can be associated with ulnar nerve injury.

Isolated fractures of the ulna's middle third may result from direct trauma, while the forearm is used to block the blow (nightstick fractures). Fractures of the forearm may result in compartment syndrome.

Management and Disposition

Both Monteggia and Galeazzi fracture-dislocations require emergent orthopedic consultation and are treated with immobilization in a long-arm splint (with elbow flexed at 90 degrees). The forearm is placed in a neutral position for a Monteggia fracture and supinated for a Galeazzi fracture. Treatment is usually surgical for both injuries, although children may be treated by reduction and casting.

Pearls

1. Any ulnar fracture with greater than 10 degrees of angulation or with a bony fragment displaced more than 50% usually requires surgical correction.
2. Do not be satisfied with the diagnosis of an isolated proximal third ulnar shaft fracture. A line drawn through the radial shaft and head must align with the capitellum in all views to exclude dislocation.
3. An ulnar styloid base fracture, if found, can be a clue to a Galeazzi fracture.
4. Since the radius and the ulna create a ring, injury to one of the bones of the forearm is often associated with fracture or dislocation of the other. Consequently, upon recognition, one must continue to seek out the other half of the injury by examining both the elbow and wrist joints.



FIGURE 11.17 ■ **Nightstick Fracture.** Isolated fracture of the middle third of the ulna. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 11.18 ■ **Monteggia Fracture.** Patients present with swelling and pain in the forearm, and often a palpable radial head in the antecubital fossa. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 11.19 ■ **Monteggia Fracture Radiograph.** A Monteggia fracture is defined by a fracture of the proximal one-third of the ulna combined with dislocation of the radial head. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 11.20 ■ **Galeazzi Fracture.** Note the fracture of the distal one-third of the radius and dislocation of the distal radioulnar joint. (Photo contributor: Lawrence B. Stack, MD.)

FRACTURES OF THE DISTAL RADIUS

Clinical Summary

Falls on an outstretched arm are common and, often, the radius is first to fracture. If there is a supinating component to the fall, the distal ulna may also fracture. In the elderly, distal radius fractures are usually extra-articular metaphyseal fractures, whereas in younger patients they are usually intra-articular with displacement of the joint surface. There are four types of radial fractures, associated with commonly known eponyms: Colles, Smith, Barton, and Hutchinson (chauffeur).

Colles fracture is the most common. There is dorsal displacement of the distal fragment and apex palmer angulation of the distal fracture fragments; on examination, a "dinner fork" deformity is often described.

The Smith fracture is of the distal metaphysis with volar displacement and apex dorsal angulation. This usually results from a blow to the dorsum of the wrist or a hyperflexion injury. Examination reveals a reverse dinner fork deformity. Barton fracture is a fracture of the dorsal or palmer rim of the distal radius. It may be associated with dislocation of the radiocarpal joint. A Hutchinson fracture is due to a direct impact to, or an avulsion of, the radial styloid.

Management and Disposition

Evaluation requires AP, lateral, and oblique views. Orthopedic consultation is required for comminuted, displaced, unstable, or open fractures, as well as those with >20 degrees of angulation or more than 1 cm of shortening. The patient can be immobilized in a sugar-tong splint. Detailed discharge instructions should be given regarding symptoms of median nerve impingement, including paresthesias and hand weakness, which should prompt return evaluation.

Pearls

1. All fractures of the distal radius must be evaluated for median nerve function before and after reduction.
2. Colles fractures warrant a high index of suspicion for intra-articular injury, especially when a radial styloid fracture is noted.
3. With a Hutchinson fracture, associated ligamentous injuries should be sought, especially scapholunate dissociation, and perilunate or lunate dislocation.



FIGURE 11.21 ■ **Colles Fracture.** The classic dinner-fork deformity is demonstrated. The distal forearm is displaced dorsally. (Photo contributor: Kristin L. Stevens, MD.)



FIGURE 11.22 ■ **Colles Fracture.** Dorsal displacement of the distal fragment. (Photo contributor: Kristin L. Stevens, MD.)



FIGURE 11.23 ■ **Smith Fracture.** A Smith fracture is sometimes described as a reverse Colles. (Photo contributor: Frank Birinyi, MD.)



FIGURE 11.24 ■ **Smith Fracture.** The radiograph reveals volar displacement of the distal radial fragment together with the bones of the wrist and hand. (Photo contributor: Frank Birinyi, MD.)

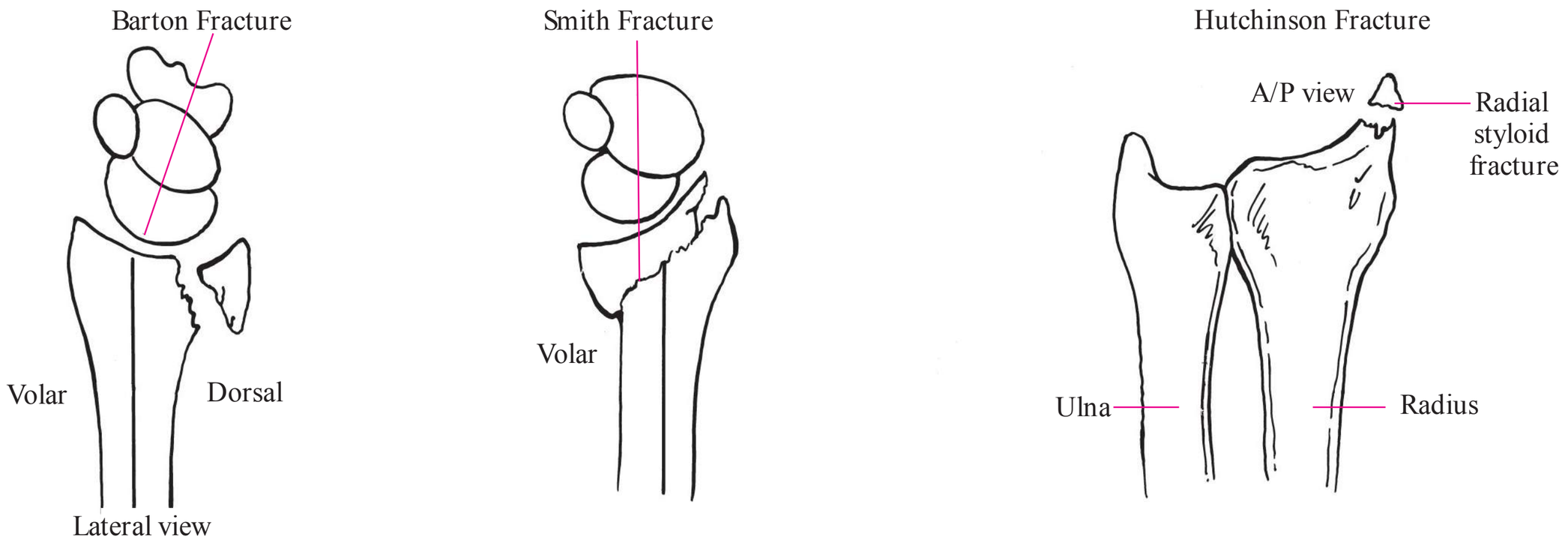


FIGURE 11.25 ■ **Distal Forearm Fractures.** Three different types of distal forearm fractures: Smith, Barton, and Hutchinson. (Adapted with permission from Simon R. Emergency Orthopedics: The Extremities. Norwalk, CT: Appleton & Lange; 1987: pp 118-119.)

CARPAL AND CARPOMETACARPAL DISLOCATIONS

Clinical Summary

Carpal and carpometacarpal dislocations are serious wrist injuries usually occurring from hyperextension. Patients complain of decreased range of motion, pain, swelling, and ecchymosis.

Lunate dislocation can occur in a palmer or dorsal position with the lunate displaced relative to the other carpals ("spilled tea cup sign"). The normal lunoradial relationship is disrupted. The median nerve is commonly involved.



FIGURE 11.26 ■ **Lunate Dislocation.** This photograph demonstrates swelling associated with a volar lunate dislocation. (Photo contributor: Cathleen M. Vossler, MD.)

If the lunoradial articulation is intact and the other carpal bones are dislocated relative to the lunate, it is termed a perilunate dislocation.

Another potentially serious injury is scapholunate dissociation, often mistakenly diagnosed as a sprained wrist. Although examination may be unremarkable except for wrist pain, an AP radiograph reveals a widening of the scapholunate joint space. A space of ≥4 mm should prompt suspicion of disruption of the scapholunate ligament. The lateral radiograph may reveal an increase of the scapholunate angle to greater than 60 to 65 degrees (normal 45-50 degrees). All these injuries may present with concomitant fractures of the carpal bones or distal forearm.

Carpometacarpal dislocations of the index and long metacarpals are fortunately rare since functional loss is often marked. Thumb, ring, and small finger carpometacarpal dislocations are more common and are frequently missed injuries.

Management and Disposition

Initial management includes adequate radiographic evaluation followed by ice, elevation, and splinting. Referral to a hand specialist is essential for adequate reduction and long-term care.

Pearls

1. A true lateral wrist radiograph best demonstrates a lunate dislocation by exhibiting the usual cup-shaped lunate bone as lying on its side and displaced either dorsally or palmerly.
2. On lateral wrist radiographs, the metacarpal, capitate, lunate, and radius should all align within a line drawn through their long axes. If this is not found, some element of dislocation, subluxation, or ligamentous instability likely exists.
3. Carpometacarpal dislocations are frequently difficult to reduce and require open reduction and fixation in approximately 50%.

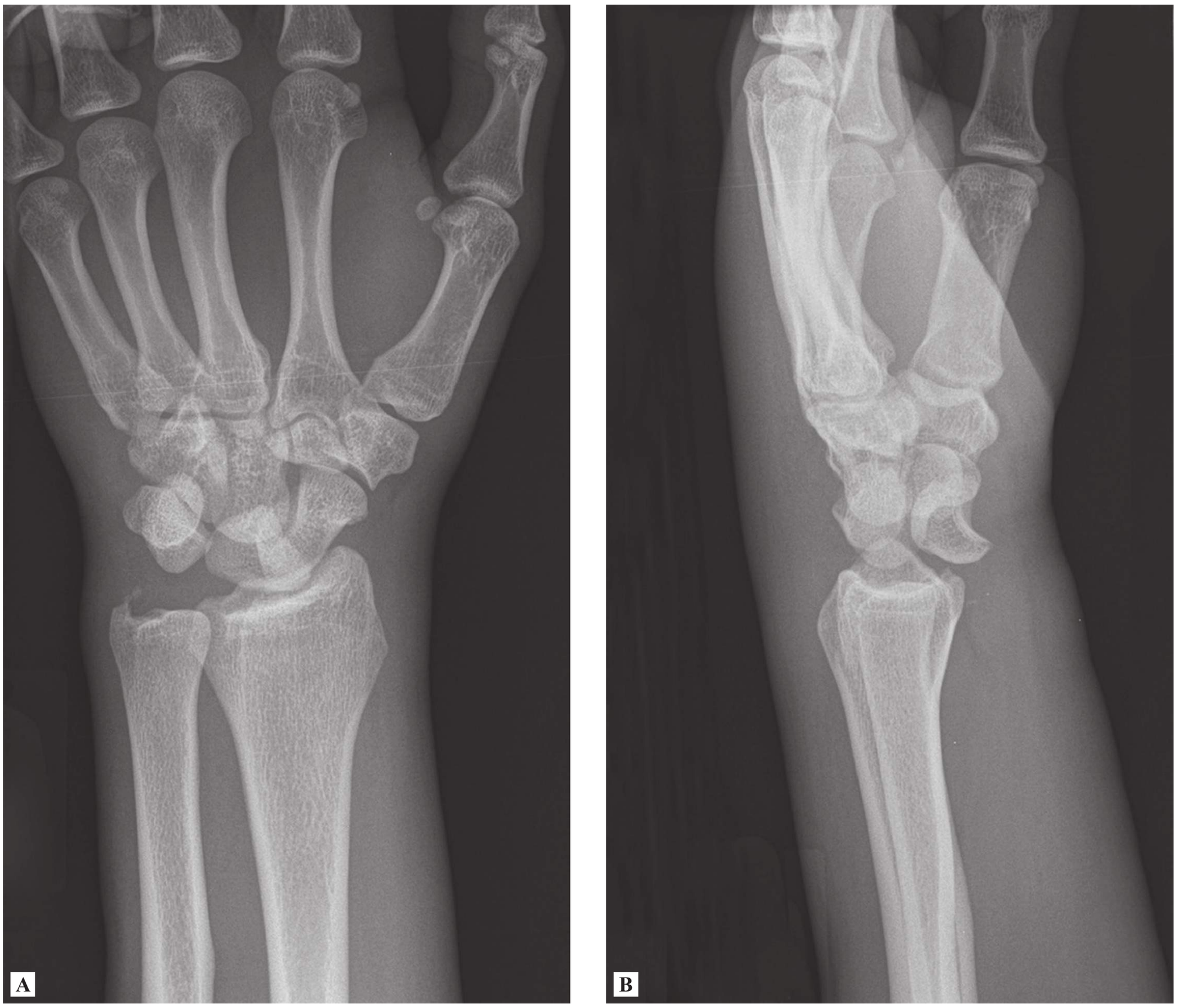


FIGURE 11.27 ■ **Lunate Dislocation.** (**A**) PA wrist view shows disruption of the carpal arcs. (**B**) Lateral reveals volar displacement of the lunate. The radius, capitate, and third metacarpal align. (Reproduced with permission of Block J, Jordanov MI, Stack LB, Thurman RJ. The Atlas of Emergency Radiology. McGraw-Hill; 2013.)

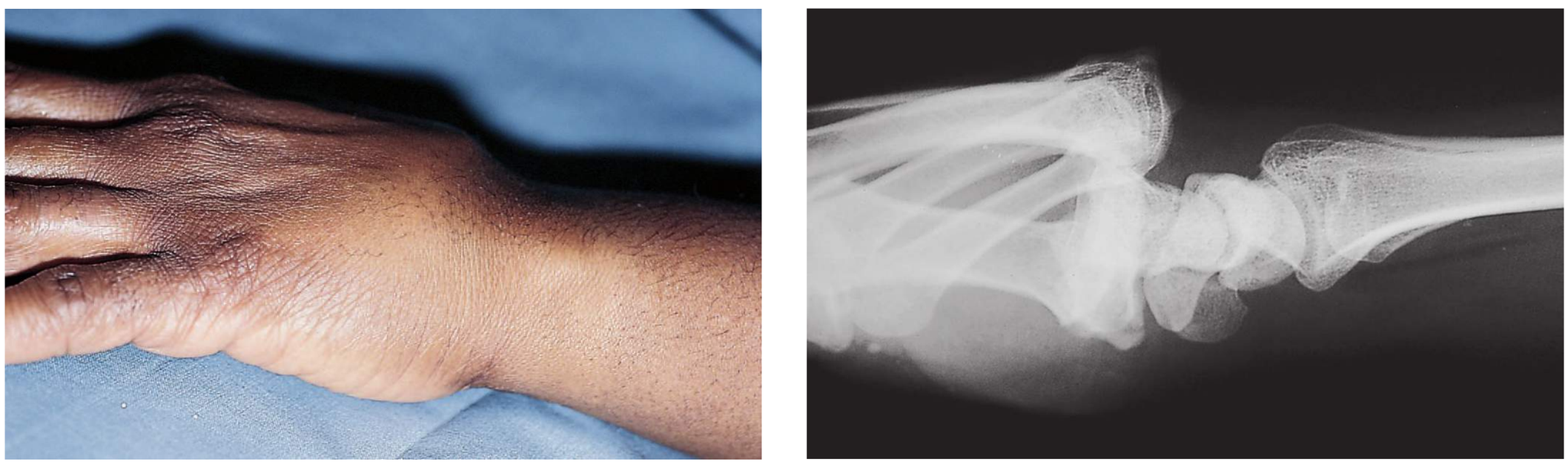


FIGURE 11.28 ■ **Carpometacarpal Dislocation.** Note the prominent deformity of the proximal metacarpals, II to IV, on the dorsal hand and the normal prominence of the ulnar styloid. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 11.29 ■ **Perilunate Dislocation.** The force from a fall on an outstretched hand disrupted the lunate-capitate articulation; the capitate and other carpal bones were driven posteriorly with respect to the lunate. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 11.30 ■ **Scapholunate Dissociation.** Note the widened scapholunate joint space. This injury is often misdiagnosed as simple wrist sprain. (Photo contributor: Alan B. Storrow, MD.)

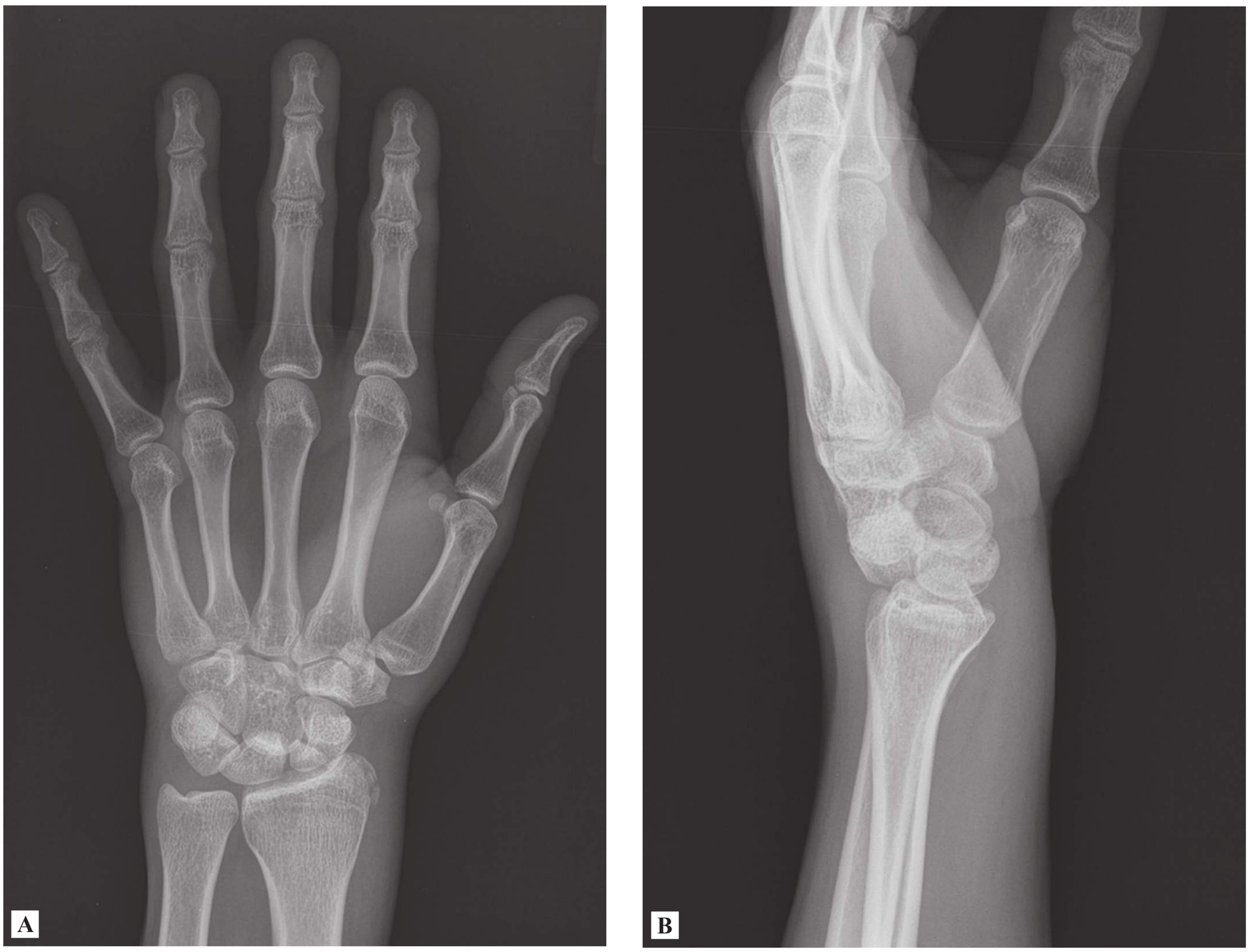


FIGURE 11.31 ■ **Perilunate Dislocation.** (**A**) PA wrist view shows disruption of the carpal arcs. (**B**) Lateral reveals the lunate aligns with the distal radius, while the capitate and metacarpals are dislocated dorsal to the lunate. (Reproduced with permission of Block J, Jordanov MI, Stack LB, Thurman RJ. The Atlas of Emergency Radiology. McGraw-Hill; 2013.)

Clinical Summary

The scaphoid is the most common carpal bone fractured. Injuries result from either a fall on an outstretched dorsiflexed hand or an axial load along the thumb's metacarpal. Misdiagnosis of a scaphoid fracture can result in delayed healing or avascular necrosis due to lack of a direct blood supply to the proximal portion of the bone. Tenderness on palpation of the anatomic snuffbox is a common finding. Examination of the wrist in ulnar deviation exposes more of the scaphoid to palpation within the anatomic snuffbox. Eliciting pain in this area when the patient resists supination or pronation of the hand, or pain with axial pressure directed along the thumb's metacarpal, is also suggestive of injury.

Negative radiographs do not rule out an occult scaphoid fracture.

Management and Disposition

Initial management includes adequate radiographic evaluation followed by ice, elevation, and a thumb spica splint. Referral to a hand specialist is essential.

Pearls

1. Patients in whom there is a clinical suspicion of an occult scaphoid fracture should receive a thumb spica splint and a repeat examination in 7 to 10 days.
2. Avascular necrosis can lead to disabling arthritis.
3. MRI is the gold standard for diagnosis.



FIGURE 11.32 ■ **Scaphoid Fracture.** Fracture of the waist, or middle third, of the scaphoid. These injuries can be associated with delayed healing and avascular necrosis. (Photo contributor: Alan B. Storrow, MD.)

CLENCHED FIST INJURY (FIGHT BITE)

Clinical Summary

The clenched fist injury classically occurs when the metacarpophalangeal (MCP) joint contacts human teeth, resulting in a laceration. Many patients will not divulge the true circumstances surrounding the injury; therefore, all wounds at the MCP joint must be considered a clenched fist injury until proven otherwise. These wounds occur most often in the dominant hand. Serious complications can result, including infection, loss of function, and amputation. Most wounds are polymicrobial. Patients who present early may have little evidence of intra-articular injury on examination, whereas those who present more than 18 hours after injury are more likely to have evidence of infection, including pain, swelling, erythema, and purulent drainage.

Management and Disposition

All wounds should be irrigated, debrided, explored, and immobilized. Patients should receive antibiotics directed at both oral and skin flora. Tetanus prophylaxis is given if needed. Radiographs should be obtained to evaluate for fractures and foreign bodies. These wounds should never be closed and should be allowed to heal by secondary intention. Reliable patients who present early, without evidence of infection or significant comorbidity, and no involvement of bone, joint, or tendon, may be treated on an outpatient basis with 24-hour follow-up. Any patient who does not meet these requirements must be hospitalized for intravenous antibiotics and wound care.

Pearls

1. Complications include cellulitis, lymphangitis, septic arthritis, abscess formation, osteomyelitis, and flexor tenosynovitis.
2. All wounds need to be examined in full flexion and extension to evaluate for tendon injuries.



FIGURE 11.33 ■ **Clenched Fist Injury.** The lacerations in this photograph were sustained from teeth during a fight. Note the subtle black ink stamp across the proximal metacarpals possibly revealing a clue about the wound's etiology. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

A boxer's fracture is a metacarpal neck fracture of the fifth digit, which commonly occurs after a direct blow to the MCP joints of the clenched fist. The apex of the fractured metacarpal bone is dorsal. On physical examination, the "knuckle" is flattened and can be palpated on the volar surface.

Examination should address neurovascular integrity and rotational deformity. Rotational deformity is evaluated by having the patient flex the digits at the proximal interphalangeal (PIP) and distal interphalangeal (DIP) joints; the four digits should point towards the scaphoid. If the involved digit overlaps another digit or does not point toward the scaphoid, a rotational deformity is present and needs to be corrected.

Management and Disposition

Radiographic evaluation is needed to determine the degree of angulation. Angular deformity of up to 40 degrees is acceptable for the fourth and fifth metacarpals, while 10 to 20 degrees is acceptable for the second and third metacarpals. Closed reduction for angulation that exceeds the limits should be attempted in the ED under appropriate nerve block.

Treatment includes ice, elevation, and immobilization in a short-arm gutter splint in the intrinsic plus position. For reduction, the MCP joint is held in 90 degrees of flexion and pressure exerted on the metacarpal head, directed dorsally. Simultaneously, the apex of the fracture is directed palmerly. Postreduction radiographs are needed to ensure adequate reduction. Early follow-up (within 7 days) with a hand specialist is essential, as the reduction can be lost with simple splinting. Higher degrees of angulation and fractures with any rotational deformity require follow-up for possible open reduction and fixation.

Pearls

1. Subtle malrotation can be recognized by looking at the alignment of the nail beds with the digits flexed.
2. Complications include collateral ligamentous damage, extensor injury damage, and malposition or clawing of the fingers secondary to incomplete reduction.



FIGURE 11.35 ■ **Boxer's Fracture.** Radiograph reveals a fracture through the neck of the metacarpal and volar displacement of the fractured segment. (Photo contributor: Cathleen M. Vossler, MD.)



FIGURE 11.34 ■ **Boxer's Fracture.** This fracture occurred when the patient punched a wall. There is loss of the "knuckle" when the dorsum of the hand is examined, especially noticeable when the patient makes a fist. (Photo contributor: Cathleen M. Vossler, MD.)



FIGURE 11.36 ■ **Boxer's Fracture: Rotational Deformity.** Malpositioning of the right fifth digit. Normally, all digits point toward a single spot on the scaphoid. (Photo contributor: Alexander T. Trott, MD.)

Clinical Summary

Chronic ulnar nerve injury results in the classic claw-hand (intrinsic minus) deformity due to atrophy and contracture of the lumbrical and interosseus hand muscles. The deformity is formed by hyperextension of the MCP joint and flexion at the PIP and DIP joints of the fourth and fifth digits. There is wasting of the interosseous and hypothenar muscles, as well as the hypothenar eminence. The acutely injured patient is unable to abduct or adduct the digits.

Chronic median nerve damage also results in the claw-hand deformity, but to the second and third digits. Thenar eminence atrophy also occurs. Acute and chronic damage to the proximal portion of the median nerve results in weakness of wrist flexion, forearm pronation, thumb apposition, and flexion of the first three digits.

Wrist drop is the most common symptom seen with radial nerve damage, occurring in situations of acute compression. It is frequently referred to as "Saturday night palsy" (as when a person falls asleep on an arm, or with the arm over a chair, resulting in temporary radial nerve neuropraxia).

Sensory and two-point discrimination sense is lost in the distribution of any acutely or chronically injured nerve.

Management and Disposition

Treatment is aimed at recognizing the underlying cause, including laceration of the nerve, compression from swelling, or hematoma formation. Treatment is splinting and appropriate referral.

Pearl

1. Long-term nerve injury results in muscle wasting. Prior to any nerve damage, the thenar and hypothenar eminences have a full appearance. Initially, there is flattening of each eminence, followed by a concave or hollow appearance.



FIGURE 11.37 ■ **Claw Hand.** Claw-hand appearance resulting from median and ulnar nerve injury. Note metacarpophalangeal joint hyperextension. (Photo contributor: Daniel L. Savitt, MD.)



FIGURE 11.38 ■ **Claw Hand.** Atrophy of the thenar and hypothenar eminences also occurs as a result of damage to the median and ulnar nerves, respectively. Note the concavity to the hypothenar eminence. (Photo contributor: Cathleen M. Vossler, MD.)

Clinical Summary

These patients complain of pain, swelling, and decreased range of motion at the thumb carpometacarpal joint. Bennett fracture is an intra-articular fracture at the ulnar aspect of the base of the first metacarpal with radial displacement of the thumb metacarpal and subluxation or dislocation of the carpometacarpal joint. A Rolando fracture is an intra-articular comminuted fracture at the base of the first metacarpal, with radial and ulnar fragments resulting in a Y- or T-shaped intra-articular fragment.



FIGURE 11.39 ■ **Bennett Fracture.** Bennett fracture involves the base of the first metacarpal. The digit is swollen and ecchymotic over the affected area. (Photo contributor: Daniel L. Savitt, MD.)

Management and Disposition

Treatment consists of ice, elevation, immobilization in a thumb spica splint, and early referral to a hand specialist. These fractures generally require operative reduction and fixation.

Pearls

1. Osteoarthritis is a common long-term complication, even after optimal management.
2. Swelling can mask significant angulation.
3. Neurovascular and tendon injuries are not commonly associated with Bennett and Rolando fractures.



FIGURE 11.40 ■ **Bennett Fracture.** Radiographic examination of a Bennett fracture illustrates an intra-articular fracture at the base of the first metacarpal with the metacarpal displaced radially and proximally. (Photo contributor: Cathleen M. Vossler, MD.)

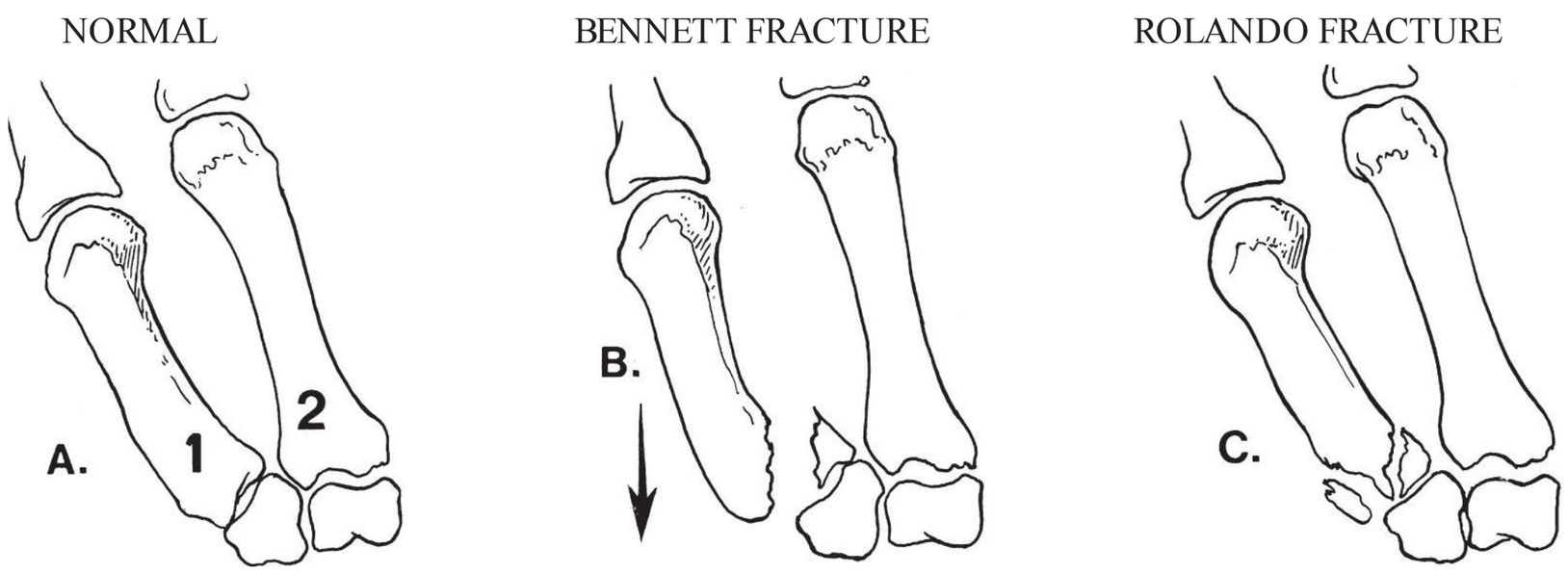


FIGURE 11.41 ■ **Intraarticular Fractures of the First Metacarpal Base.** A radial, intraarticular fracture at the base of the first metacarpal is a Bennett fracture (**B**). A comminuted intra-articular fracture at the base of the first metacarpal is a Rolando fracture (**C**). Normal anatomy (**A**).



FIGURE 11.42 ■ **Rolando Fracture.** Note the comminuted intra-articular fracture at the base of the first metacarpal. (Photo contributor: Cathleen M. Vossler, MD.)

Clinical Summary

The boutonnière deformity results from injury to the central slip insertion of the extensor hood on the dorsal surface of the middle phalanx. After a tear of the central slip, the flexor tendon is unopposed at the PIP and the lateral bands of the extensor tendon contract. With time, these displace volarly resulting in additional PIP joint flexion and DIP joint extension. The central slip rupture may result from forceful flexion of the PIP joint during full extension, a dorsal PIP joint laceration, or a palmar PIP joint dislocation. The deformity may not be immediately apparent as it takes time for the lateral bands to slide down to create extension of the DIP joint. Pain and swelling over the dorsal PIP joint, tenderness over the PIP central slip, inability to extend the PIP, and possible DIP joint hyperextension are common. Radiographically, a small bone fragment may be seen at the proximal portion of the dorsal middle phalanx.

Swan-neck deformity occurs as a result of the contracture of intrinsic hand muscles secondary to systemic diseases such as rheumatoid arthritis and SLE. The digit is contorted with hyperextension of the PIP and flexion of the DIP and MCP joints.

Management and Disposition

In dealing with a closed injury resulting in a boutonnière deformity, immobilization of the PIP joint in extension for 4 weeks is adequate, followed by active range of motion. Open injuries must be carefully explored and repaired. If the deformity is associated with a bony fragment, surgical repair may be necessary. Swan-neck deformities should be splinted and referred as an outpatient. Both deformities require outpatient referral to a hand specialist.

Pearls

1. Boutonnière deformity generally develops weeks after the initial injury as the lateral bands contract.
2. Surgical repair may be required for patients when conservative therapy yields inadequate results.



FIGURE 11.44 ■ **Boutonnière Deformity.** Fourth digit flexion of the PIP joint and extension of the DIP joint. (Photo contributor: E. Lee Edstrom, MD.)

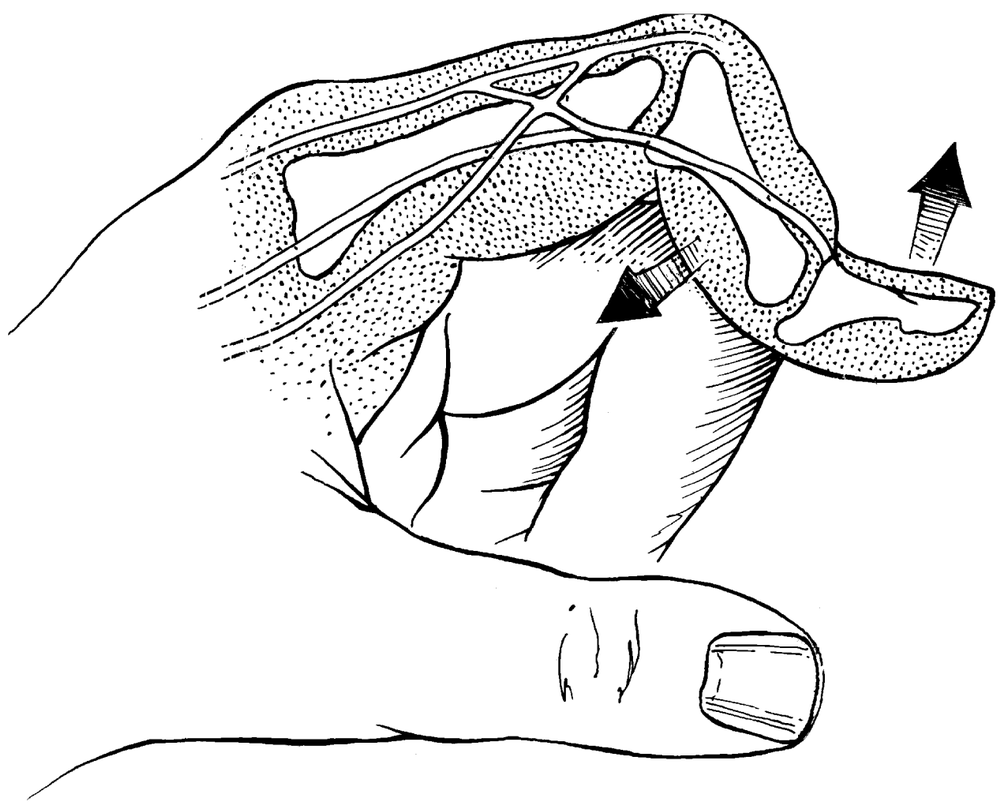


FIGURE 11.43 ■ **Boutonnière Deformity.** Illustration of the central slip rupture and the resultant subluxation of the lateral bands. The subluxation exerts a pull on the middle phalanx resulting in the deformity.

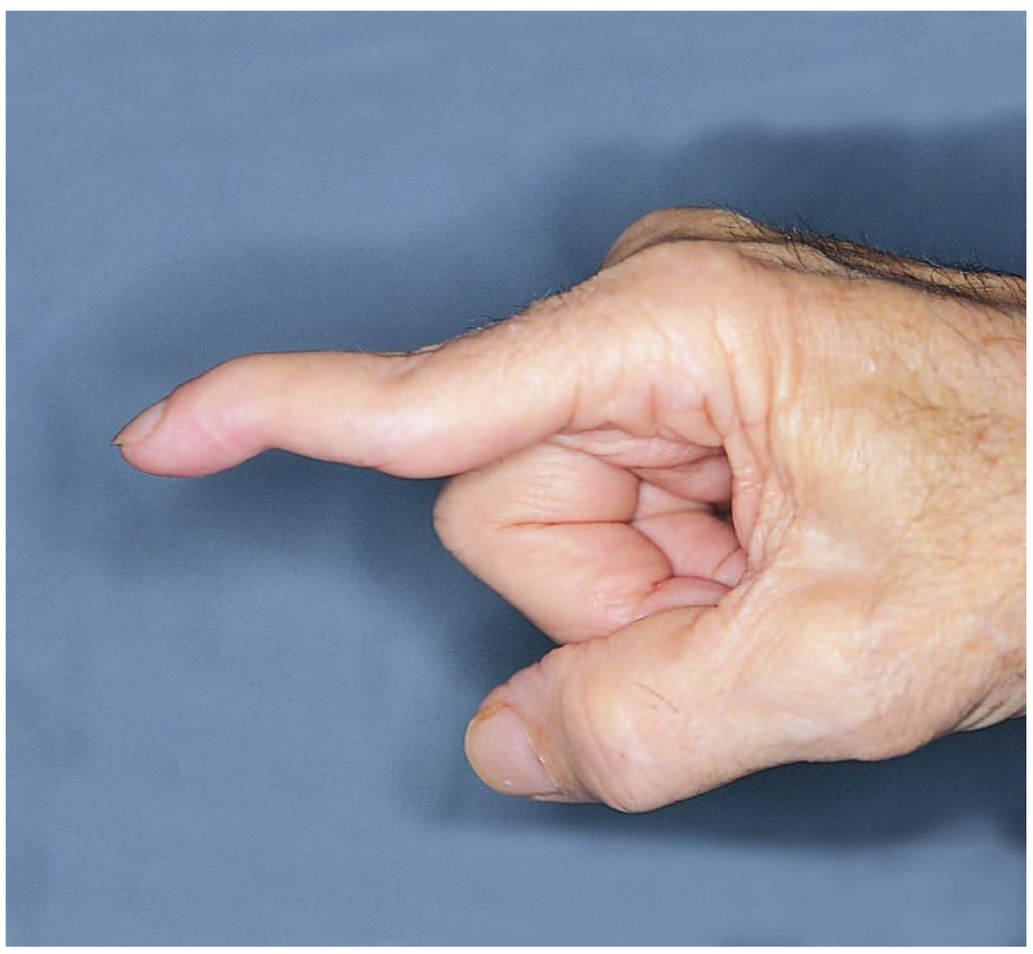


FIGURE 11.45 ■ **Swan-Neck Deformity.** Hyperextension of the PIP joint and flexion of the DIP joint. (Photo contributor: Cathleen M. Vossler, MD.)

HIGH-PRESSURE INJECTION INJURY

Clinical Summary

A large number of commercial devices are able to deliver liquids and gases at pressures exceeding 5000 psi. Unfortunately, these devices may accidentally introduce these substances into the body, especially the upper extremities, leading to possible ischemia. This can result as a consequence of direct chemical irritation, venous outflow obstruction, arterial compression secondary to the volume of material, spasm, or edema. Soon after the injection, the injury can appear very innocuous. The injected material spreads along fascial planes, so the extent of injury can be quite misleading and is often subtle on initial presentation. A small puncture wound, or no apparent skin break, with minimal swelling may be found. Over time, swelling and pain increase.

Management and Disposition

Immediate operative debridement is the treatment of choice; early consultation with a hand specialist is necessary. Radiographic examination to evaluate for fractures and to delineate the spread of the injected material should be considered. Tetanus, analgesia, and broad-spectrum antibiotics should be administered. The affected extremity should be elevated and splinted.

Pearls

1. Do not be misled by the "benign" appearance of the initial injury.
2. Delays in treatment can lead to compartment syndrome.
3. Digital blocks are contraindicated because of the potential for increased tissue pressure and compromise of tissue perfusion.



FIGURE 11.46 ■ **High-Pressure Injection Injury.** Injury incurred by a grease gun that accidentally discharged into a hand. Note the swelling and erythema. (Photo contributor: Richard Zienowicz, MD.)



FIGURE 11.47 ■ **High-Pressure Injection Injury.** Industrial worker with initially benign appearing high-pressure injection injury at the base of his third finger (**A**). Note the spread along fascial planes demonstrated radiographically (**B**). (Photo contributor: Kevin J. Knoop, MD, MS.)

GAMEKEEPER'S THUMB (SKIER'S THUMB)

Clinical Summary

Forced radial deviation (abduction) of the thumb can cause a rupture of the ulnar collateral ligament. Known as "gamekeeper's" or "skier's" thumb, the tear usually occurs at the proximal phalanx insertion and can be associated with volar plate and dorsal capsule injury. Pain and swelling are present over the ulnar aspect of the proximal phalanx and thumb metacarpal.

Radiographs may reveal a small avulsion fracture of the proximal phalanx. Abduction stress testing (stabilizing the metacarpal with one hand while applying radial stress on the proximal phalanx) may provide additional clinical information, especially in patients with normal radiographs. Classically, more than 30 to 40 degrees radial angulation indicates complete rupture. Stress testing should be done on both sides in extension and 30 degrees of flexion while feeling for a firm endpoint.

Management and Disposition

Apply a thumb spica splint and provide analgesia. Patients with pincer function weakness, point tenderness at the volar-ulnar aspect of the thumb MCP joint, a bony fragment of >15% of the articular surface, avulsed fragment displacement of >5 mm, or significant angulation on stress testing should prompt hand surgery referral. Complete tears need repair within 1 week. A sprain without instability is commonly treated with thumb spica casting or splinting for 4 to 6 weeks followed by range of motion exercises.

Pearls

1. Radial collateral ligament rupture can also occur with forced adduction, but is uncommon.
2. Skier's thumb refers to an acute injury, while gamekeeper's has classically been associated with repetitive trauma.
3. Thumb MCP joint laxity is highly dysfunctional, painful, and may lead to late arthritis.



FIGURE 11.48 ■ **Gamekeeper's Thumb.** Laxity of 30 to 40 degrees more than the uninjured thumb, measured in neutral and 30 degrees of flexion, is strongly suggestive of a complete ulnar collateral ligament tear. There is no "endpoint" to the radial deviation of the phalanx. (Used with permission from Brunicardi FC, Anderson DK, Billar TR, et al. Schwartz's Principles of Surgery. 8th ed. New York: McGraw-Hill; 2005. © 2005 by The McGraw-Hill Companies, Inc.)



FIGURE 11.49 ■ **Gamekeeper's Thumb.** Stress x-ray of a thumb with a complete ulnar collateral ligament tear demonstrates marked instability of the ulnar side of the MCP joint and radial deviation of the proximal phalanx. (Used with permission from Brunicardi FC, Anderson DK, Billar TR, et al. Schwartz's Principles of Surgery. 8th ed. New York: McGraw-Hill; 2005. © 2005 by The McGraw-Hill Companies, Inc.)

PHALANGEAL DISLOCATIONS

Clinical Summary

Phalangeal dislocations are common and can occur at all three finger joints. These are generally dislocated dorsally, caused by hyperextension and axial compression, and may have associated volar plate damage. PIP volar dislocations can be irreducible secondary to rupture of the extensor tendon and herniation of the proximal phalanx through the extensor hood, requiring operative repair. MCP joint dorsal dislocations are often due to hyperextension. DIP dislocations are the rarest but can occur when an axial force is applied to the distal phalanx. Gross deformity is noted on examination, with the distal phalanx generally displaced dorsally.

Management and Disposition

Digital nerve block for reduction and splinting is appropriate anesthesia for the PIP and DIP joints. Ulnar, median, or radial nerve blocks are necessary for the MCP joints. Reduction of dorsal dislocations is accomplished via joint hyperextension with concurrent application of horizontal traction followed by joint flexion. Flexion at the MCP joint will facilitate reduction of interphalangeal joints. Postreduction radiographs are necessary to ensure adequate reduction. After closed reduction of a dorsal dislocation, the DIP joint should be splinted in slight flexion and the PIP joint in at least 20 degrees of flexion for 3 to 5 weeks, depending on the degree of ligamentous damage. Hand specialist follow-up is recommended.

Pearls

1. All joints should be tested for instability after reduction, using a digital nerve block to facilitate testing.
2. PIP joint volar dislocation can be unstable, requiring open reduction and internal fixation.
3. Joint dislocations that have volar plate entrapment may be impossible to reduce and require surgical repair for successful reduction.



FIGURE 11.50 ■ **Phalangeal Dislocation.** This patient dislocated the long finger PIP joint during an altercation. The PIP joint is displaced dorsally with an obvious deformity. (Photo contributor: Cathleen M. Vossler, MD.)



FIGURE 11.51 ■ **Phalangeal Dislocation.** Medial angulation of the ring finger suggests PIP dislocation. (Photo contributor: Daniel L. Savitt, MD.)

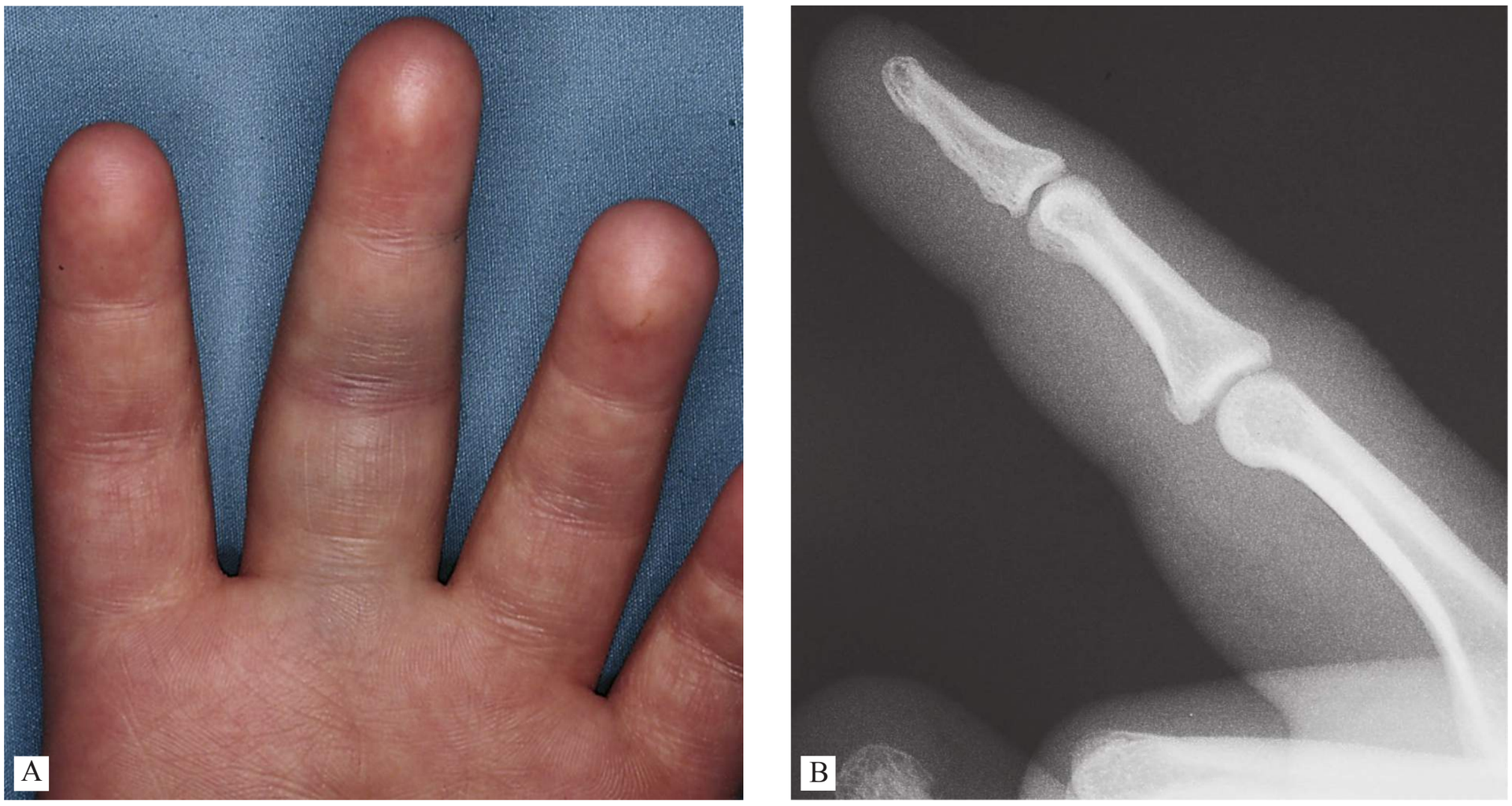


FIGURE 11.52 ■ **Volar Plate Injury.** Subtle PIP swelling and ecchymosis of the long digit seen with a volar plate injury (**A**). Radiographs of volar plate injuries often reveal a small fragment on the volar surface of the PIP joint (**B**). (Photo contributor: Cathleen M. Vossler, MD.)

MALLET FINGER AND JERSEY FINGER

Clinical Summary

Mallet finger commonly occurs after the DIP joint is forcibly flexed against an actively extended finger, tearing the extensor mechanism as it inserts on the distal phalanx. This can occur after a sudden axial blow to an extended fingertip. The patient presents with an inability to actively extend the distal phalanx while maintaining a normal passive range of motion and the DIP joint remains passively flexed. On radiography, there may be a small bony avulsion fragment on the dorsum at the distal phalanx.

A Jersey finger involves an avulsion of the distal phalanx flexor mechanism. The flexor digitorum profundus (FDP) tears as a result of forced extension of a fully flexed DIP, as would occur when someone grabs the jersey of an opponent while attempting to tackle them. Most commonly, the ring finger is involved. Clinically, the patient presents with an inability to actively flex the DIP joint while maintaining a full passive range of motion. Radiographically, a bony avulsion fragment may be present.

Management and Disposition

A closed mallet finger without involvement of the joint can be treated by splinting the DIP joint in extension; the PIP joint should not be splinted. This splint should be worn continuously for at least 6 weeks. Operative treatment is not usually required.

Jersey finger often requires surgical repair; early referral is recommended. Prognosis worsens if treatment is delayed or severe tendon retraction is present.

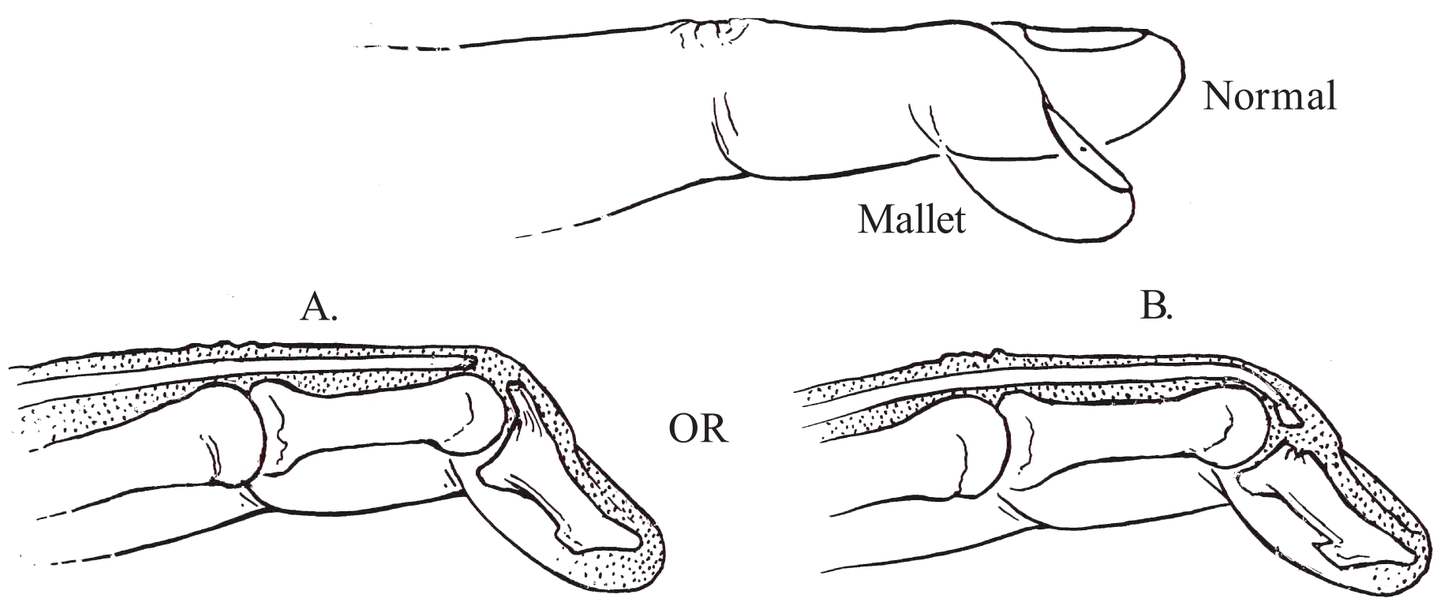


FIGURE 11.53 ■ **Mallet Finger.** This illustration demonstrates that the unopposed flexion of the DIP joint is secondary to the complete tear of the tendon (**A**), or an avulsion of a small chip fragment (**B**).



FIGURE 11.54 ■ **Mallet Finger.** The distal phalanx is held in flexion and the patient is unable to extend it. (Photo contributor: Kevin J. Knoop, MD, MS.)

Pearls

1. Avulsion of a significant portion of the articular surface of a mallet finger (more than one-third) may require open reduction with internal fixation.
2. The Jersey finger involves the FDP tendon at the DIP joint. It may be tested by isolating the affected DIP (ie, holding the MCP and PIP joints in extension while the other fingers are in flexion) and asking the patient to flex the DIP joint.

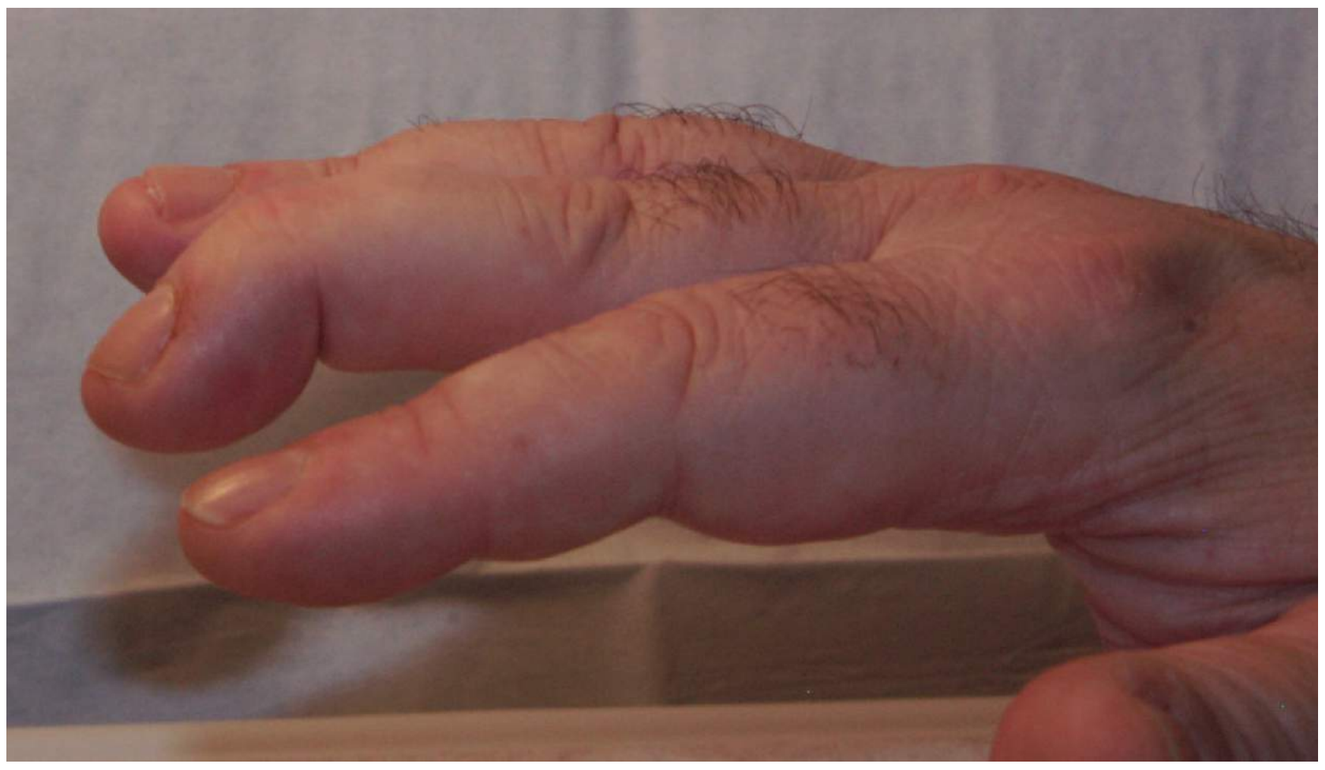


FIGURE 11.55 ■ **Mallet Finger.** Classic mallet finger—the long finger remains flexed at the DIP joint while the patient is attempting to actively extend his fingers. (Photo contributor: Matthew Kopp, MD.)



FIGURE 11.56 ■ **Jersey Finger.** The normal cascade of flexion is disrupted in the injured hand, consistent with a Jersey finger. (Used with permission from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz's Principles of Surgery. 8th ed. New York: McGraw-Hill; 2005. © 2005 by The McGraw-Hill Companies, Inc.)

COMPARTMENT SYNDROME

Clinical Summary

Compartment syndrome develops when the pressure in the inelastic fascial space increases to a point where it causes compression and dysfunction of venous outflow. Major vascular and neural compromise lead to the classic five "Ps" that characterize late compartment syndrome: pallor, paresthesias, poikilothermia, paralysis, and pulselessness. Compartment syndrome may result from exertion, circumferential burns, frostbite, constrictive dressings, arterial bleeding, severe soft tissue injury, and fracture. It can occur anywhere in the body, most commonly in the anterior compartment of the leg.

The earliest symptom is severe pain out of proportion to the physical findings. The involved compartment is extremely firm. The pain is worsened with passive range of motion due to ischemic muscle fiber stretch. Consequently, the patient often holds the injured part in a position which relaxes the involved muscle groups.

Paresthesia is a late sign of nerve compromise, commonly with vibratory sensation lost first. Motor weakness, pallor, poikilothermia, and pulselessness are very late signs and only occur after irreversible muscle, nerve, and vascular damage have occurred. The goal of the emergency physician is to identify compartment syndrome before these late signs occur.

The diagnosis is confirmed by measuring the compartment pressures; >30 mm Hg are suggestive of compartment syndrome and should prompt surgical consultation for fasciotomy consideration.

A serious complication is Volkmann ischemic contracture, classically following a supracondylar fracture. Postischemic swelling, producing increased pressure within the enclosed osteofascial forearm compartment, reduces capillary blood perfusion below the level necessary for tissue viability. If not addressed, muscle and nerve necrosis, and eventual replacement by fibrotic tissue, produces a contracture. Refusal to open the hand, pain with passive extension of the fingers, and forearm tenderness are signs of impending Volkmann ischemia.

Management and Disposition

The initial treatment is removal of constrictive dressings and jewelry as well as frequent reevaluation. If there is no improvement, decompression via a fasciotomy should be considered. Aside from muscle and nerve damage, complications of compartment syndrome include myonecrosis which can cause myoglobinuria and renal failure.

Pearls

1. The diagnosis of compartment syndrome should be made early and be based on clinical evaluation and the mechanism of injury. Crush or compression injuries should heighten suspicion.
2. The most common areas of the extremities affected by compartment syndrome are the anterior compartment of the leg due to proximal tibial fractures, and the volar compartment of the forearm secondary to ulna, radius, or supracondylar fractures.



FIGURE 11.57 ■ **Compartment Syndrome.** A swollen and tense right forearm as a result of compartment syndrome. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 11.58 ■ **Compartment Syndrome.** Late anterior compartment syndrome of the left lower extremity is manifested by anterior tibial pain and tense "woody" swelling. (Photo contributor: Timothy Coakley, MD.)



FIGURE 11.59 ■ **Compartment Syndrome.** Volkmann contracture is a serious late complication of unrelieved compartment syndrome. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 11.60 ■ **Compartment Pressures.** Intracompartmental pressure monitoring can be accomplished with commercially available devices. Normal tissue pressures should be less than 10 mm Hg; orthopedic consultation is recommended when pressures exceed 30 mm Hg. (Photo contributor: Selim Suner, MD, MS.)

SUBUNGUAL HEMATOMA

Clinical Summary

A subungual hematoma is a collection of blood underneath the nail, usually occurring secondary to distal digit trauma. Such patients usually present with throbbing pain secondary to pressure beneath the nail. Associated injuries include nail bed trauma and distal tuft fractures. When fractures are present, these are considered open.

Management and Disposition

A radiograph should be done to evaluate for fracture. Acutely, if the subungual hematoma involves less than 50% of the nail matrix, trephining the nail with a sterile needle or electrocautery is adequate to relieve pain by allowing drainage. A digital block may be required prior to trephination. The involved digit can be soaked in sterile water with peroxide after trephination to encourage drainage.

Management of larger hematomas is controversial. Some authors advocate nail removal for bed injury repair if the hematoma covers more than 50% of the nail, or if there is an associated fracture. Some believe nail removal is best reserved for injuries damaging the nail plate and surrounding tissues, regardless of the size of the hematoma or presence of fracture. After removal, the patient's clean nail, a piece of foil, or petroleum gauze should be placed between the eponychial nail fold and the germinal matrix.

The patient with a fracture should be discharged with a splint supporting the distal joint in extension and all dressings should be kept dry. Antibiotics are not required unless the area is contaminated.

Pearls

1. Subungual hematomas are a sign of nail bed injury.
2. Subungual hematomas with surrounding nail bed and nail fold injuries require nail removal and evaluation of the nail bed for injury and careful repair if needed.
3. A hand-held, high-temperature, portable cautery device is a good tool for trephination.



FIGURE 11.61 ■ **Subungual Hematoma.** Two subungual hematomas secondary to crush injury. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 11.62 ■ **Nail Bed Laceration.** Bleeding from a nail bed laceration causes a subungual hematoma. This image depicts a nail bed laceration seen after nail removal. (Photo contributor: Alan B. Storrow, MD.)

Clinical Summary

Hip dislocations are often associated with fractures of the acetabulum, ipsilateral femur, or knee, and can be anterior, posterior, or inferior. Ninety percent are posterior, resulting from significant forces exerted on a flexed hip and knee (eg, a passenger in a motor vehicle accident whose knees hit the dashboard). Anterior hip dislocations occur when there is forced external rotation of the extended hip, which forces the head out of the acetabulum, either by tearing the anterior capsule or by fracturing the anterior wall of the acetabulum.

Patients complain of severe hip pain and decreased range of motion. Posterior dislocations present with the extremity shortened, internally rotated, adducted, and flexed. With anterior dislocations, the leg is often abducted, externally rotated, and slightly flexed; however, this presentation can vary.

Management and Disposition

Treatment for dislocations is early closed reduction, often with conscious sedation, although ultrasound-guided femoral nerve blocks are a popular option. A neurovascular and radiographic evaluation should occur before and after any reduction attempts. Since the muscles around the hip are so strong, a general anesthetic with complete paralysis may be required. Posterior dislocations are reduced using in-line traction with the hip and knee flexed to 90 degrees, followed by gentle internal to external rotation. Anterior dislocations are reduced using strong in-line traction with the hip in neutral flexion extension or slight extension, slight adduction and internal rotation, followed by abduction.

Orthopedic consultation should be obtained as early as possible. These patients require admission, with frequent neurovascular evaluation.

Hip replacements have a tendency to dislocate more easily than native hips and usually require less energy for reduction. Radiographs should be done pre- and postreduction to evaluate for periprosthetic fractures.

Pearls

1. Complications of posterior hip dislocations include sciatic nerve injury and avascular necrosis.
2. In the young patient, immediate reduction of a native hip is imperative and should be accomplished within 6 hours.

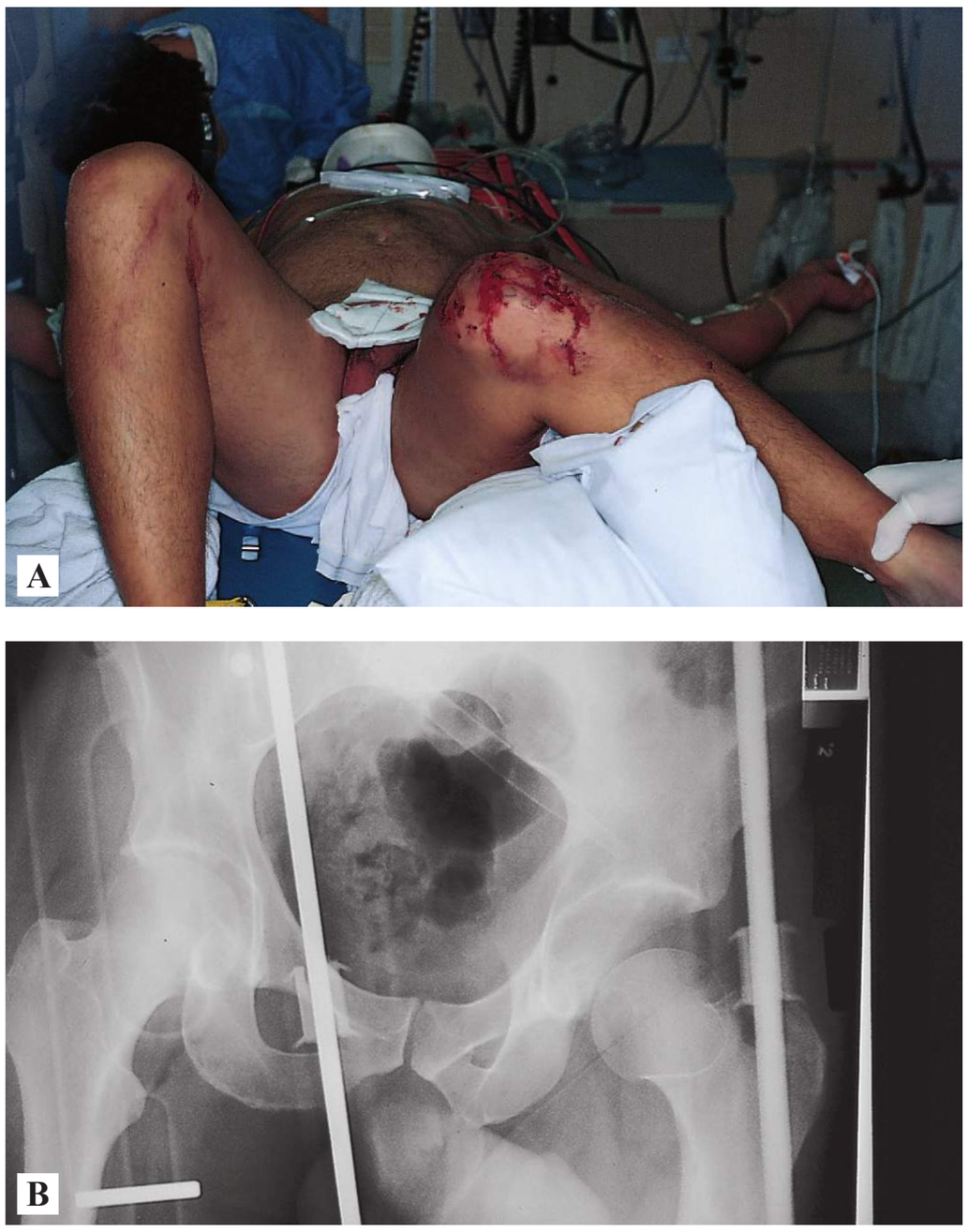


FIGURE 11.63 ■ **Hip Dislocation.** Typical patient appearance of a left posterior hip dislocation. Note internal rotation of the affected extremity (**A**). Radiograph (**B**). (Photo contributor: Cathleen M. Vossler, MD.)

Clinical Summary

Fractures above the level of the lesser trochanter are termed hip fractures. The femoral head has a tenuous blood supply and fractures can compromise blood flow, resulting in avascular necrosis. For classification, hip fractures are generally divided into intracapsular (femoral head and neck fractures) and extracapsular (trochanteric, intertrochanteric, and subtrochanteric). Accurate classification is important because intracapsular fractures are more likely to have vascular disruption.

Complaints include groin or buttock pain, tenderness, and an inability to bear weight on the affected side. There can be shortening of the affected leg, as well as abduction and external rotation.

Both the Shenton line (the curved line formed by the top of the obturator foramen and the inner side of the neck of the femur) and the neck shaft angle should be evaluated, but can be normal in nondisplaced fractures.

Management and Disposition

Fractures of the hip require early orthopedic consultation for admission and, in most cases, surgical reduction and fixation. Femoral head fracture-dislocations are an orthopedic emergency and require immediate reduction. A neurovascular examination should be carefully performed before and after any reduction attempts.

Pearls

1. Fracture-dislocation of the femoral head requires great forces; intra-abdominal and retroperitoneal injuries should be considered.
2. Hip fractures may be diagnosed by auscultation of differences in bone conduction between the two extremities. This is performed by placing the stethoscope's diaphragm on the anterosuperior iliac spine and giving the patella soft taps.
3. Any elderly patient with an inability to bear weight has a hip fracture until proven otherwise. Hip fractures can be secondary to osteoporosis, so there does not need to be a history of trauma. Since plain films may not be sensitive enough to identify some hip fractures, other imaging such as CT or MRI should be considered.

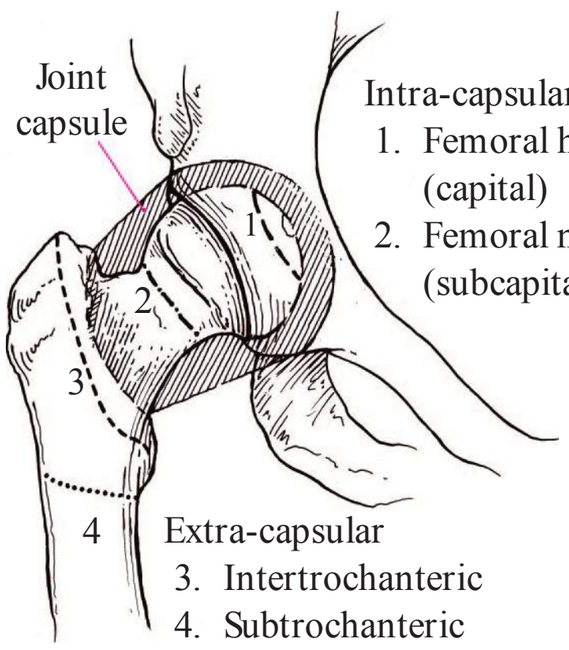


FIGURE 11.64 ■ **Hip Fractures.** This illustration depicts the different types of proximal femoral fractures.



FIGURE 11.65 ■ **Hip Fracture.** Patients with hip fractures often present with the affected extremity shortened, externally rotated, and abducted. Note the rotation and shortening in this patient with a right intertrochanteric fracture. (Photo contributor: Cathleen M. Vossler, MD.)



FIGURE 11.66 ■ **Hip Fracture.** Radiographic examination reveals an intertrochanteric fracture. (Photo contributor: Cathleen M. Vossler, MD.)

Clinical Summary

Knee extensor injuries usually occur from three different mechanisms: quadriceps tendon tear, patellar tendon tear, and patellar fracture. Extension may be limited by any disruption of these three elements. Patients with a history of trauma, other systemic conditions, steroid injections, or fluoroquinolone use are predisposed to tendon disruptions.

Quadriceps tendon ruptures are the most common extensor failure and are more often seen in the elderly. Forced flexion during quadriceps contraction may cause rupture; patients may experience sudden buckling and pain. The patella is inferiorly displaced with proximal patellar tenderness and swelling. A soft tissue defect at the distal aspect of the quadriceps is often apparent.

Patellar tendon rupture occurs in the younger, more active population and results in proximal displacement of the patella with inferior pole tenderness and swelling.

Patellar fractures may be transverse (most common), comminuted, or vertical. They may be caused by direct trauma or as a result of high eccentric tension forces. Tenderness, swelling, and sometimes a palpable defect are present.

Examination will reveal weakness of knee extension against gravity. Patients with complete tears will not be able to extend their knee.



FIGURE 11.67 ■ **Quadriceps Tendon Rupture.** Inferior displacement of the patella and a distal quadriceps defect suggest quadriceps tendon rupture. (Photo contributor: Robert Trieff, MD.)



FIGURE 11.68 ■ **Patellar Tendon Rupture.** Proximal displacement of the patella and inferior pole tenderness in a patient with right patellar tendon rupture. (Photo contributor: Kevin J. Knoop, MD, MS.)

Management and Disposition

Lateral radiographs may help distinguish between the two tendon injuries. Patients demonstrating either partial or complete tendon ruptures can be discharged with their knee in extension and follow up with orthopedics. Care must be taken to differentiate between a bipartite patella and a fracture. Nondisplaced and displaced patellar fractures should receive splinting in full extension and orthopedic referral.

Pearls

1. MRI may distinguish partial from complete tears.
2. Patellar fractures may be complicated by future degenerative arthritis.

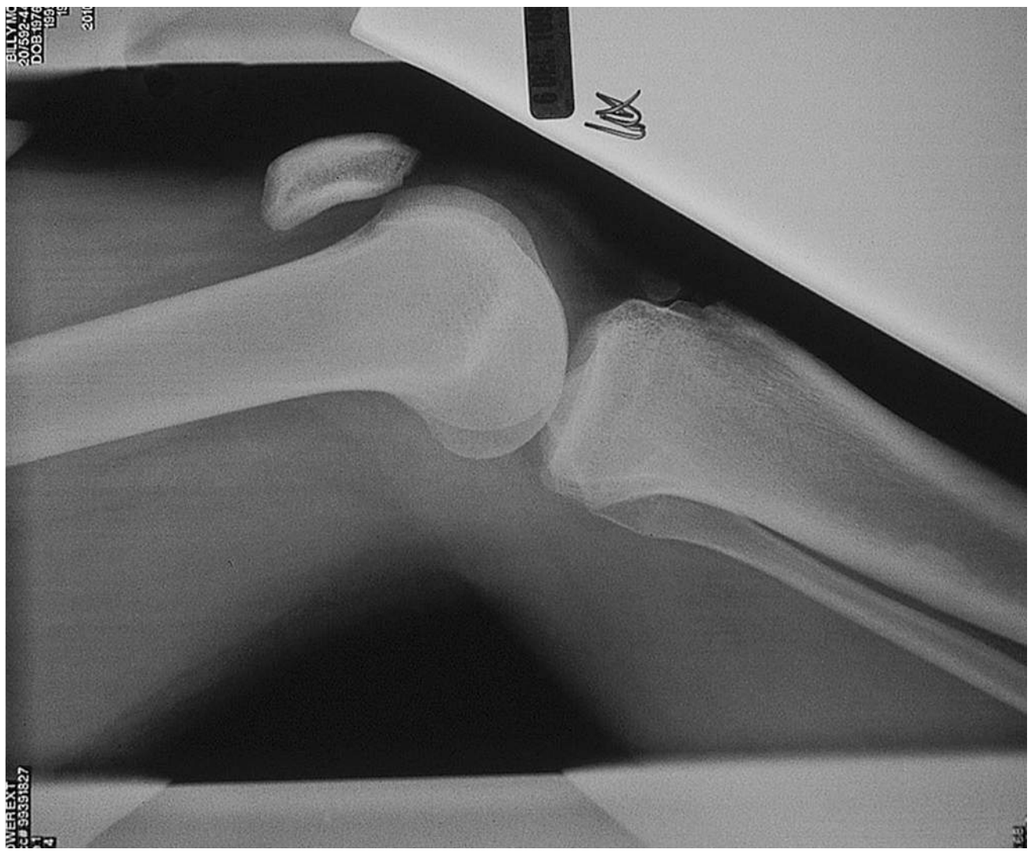


FIGURE 11.69 ■ **Patellar Tendon Rupture.** A lateral radiograph reveals the proximal patellar displacement seen with complete patellar tendon rupture. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 11.70 ■ **Patella Fracture.** The inferior pole of the patella is comminuted and displaced. Internal fixation is indicated if the patient cannot actively extend the knee. (Used with permission from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz's Principles of Surgery. 8th ed. New York: McGraw-Hill; 2005. © 2005 by The McGraw-Hill Companies, Inc.)

Clinical Summary

Patellar dislocations can result from either direct trauma or powerful quadriceps contraction with knee flexion. Most commonly, the patella dislocates laterally. Patients who present after a patellar dislocation may state their knee dislocated and spontaneously reduced. A hemarthrosis can be present. Common complaints include pain, swelling, and knee deformity. Examination reveals fullness or deformity in the lateral aspect of the knee. The patellar apprehension test (gently displacing the patella laterally or medially while in extension) is often positive in spontaneously reduced patellar dislocations. The patient will become apprehensive as you attempt to reproduce the dislocation. Radiography will confirm the dislocation and help rule out associated fracture. Fractures of the patella or lateral femoral condyle occur in 5%.

Management and Disposition

Lateral dislocations are reduced by flexing the hip, extending the knee, and gently directing pressure medially on the patella. Reduction is easily accomplished and results in immediate relief of pain. Postreduction films should be taken. After successful reduction, patients require a knee immobilizer in full extension for 4 to 6 weeks. Outpatient referral to a musculoskeletal specialist should be obtained for long-term follow-up

Pearls

1. A dislocated patella may reduce spontaneously prior to presentation; it is a possibility in any patient who presents with knee pain. This may be elucidated by inquiring about a knee deformity at the time of injury that is no longer present.
2. Complications of patellar dislocation include degenerative arthritis, recurrent dislocations, and chondral or osteochondral loose bodies.



FIGURE 11.71 ■ **Patellar Dislocation.** An obvious lateral deformity of the right patella in a patient with dislocation. (Photo contributor: Cathleen M. Vossler, MD.)



FIGURE 11.72 ■ **Patellar Dislocation.** Lateral deformity of the right patella in a patient with dislocation. (Photo contributor: Lawrence B. Stack, MD.)

KNEE DISLOCATION

Clinical Summary

Knee dislocations are classified by the direction of tibial displacement relative to the femur (anterior, posterior, medial, lateral, or rotatory), invariably cause multiple ligamentous injuries, and are usually the result of motor vehicle crashes, falls, sports, and industrial injuries. Anterior dislocations are more common and usually occur after high-energy hyperextension injuries. Knee dislocations are associated with popliteal artery and common peroneal and tibial nerve injuries. Popliteal artery injury can result from both anterior and posterior dislocations, is more common than nerve injury, and is more devastating. Injury can be present despite normal pulses and, if not identified and repaired within 8 hours, amputation may be necessary. Common peroneal nerve injury can cause decreased sensation on the lateral foot, impaired dorsiflexion and eversion, and impaired sensation over the first dorsal web space. Knee dislocations can spontaneously relocate, so the physician must maintain a high index of suspicion. If a patient presents with a grossly unstable knee after trauma, this may be a reduced dislocation and should be evaluated as such.

Injuries are painful and visually striking. An effusion will often be absent since the capsule has been violated. On examination, the knee will be grossly unstable since dislocations tend to injure most of the surrounding ligaments.

Management and Disposition

Management includes early reduction, immobilization, assessment of distal neurovascular function, and emergent orthopedic referral. Reduction of anterior dislocation is accomplished by flexing the hip 20 degrees and having an assistant apply longitudinal traction on the leg while keeping one hand on the tibia and simultaneously lifting the femur back into position. A posterior splint with the knee in 20 degrees of flexion is used for immobilization and to avoid tension on the popliteal artery. The patient should be admitted for observation and, likely, arteriography.

Pearls

1. Knee dislocations are often associated with a fracture of the tibial plateau.
2. The presence of distal pulses in the foot does not rule out an arterial injury.
3. Vascular repair after 8 hours of injury carries an amputation rate of greater than 80%.

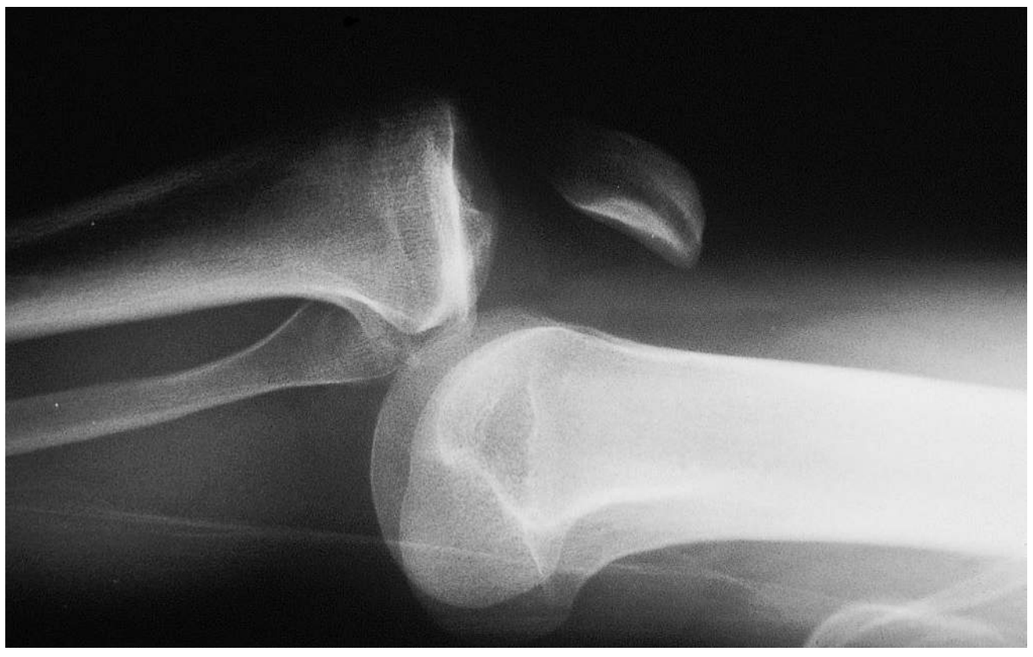


FIGURE 11.73 ■ **Anterior Knee Dislocation.** Anterior displacement of the tibia in relation to the femur. (Photo contributor: Selim Suner, MD, MS.)



FIGURE 11.74 ■ **Posterior Knee Dislocation.** Posterior displacement of the tibia in relation to the femur. (Photo contributor: Paul R. Sierzenski, MD.)



FIGURE 11.75 ■ **Posterior Knee Dislocation.** Posterior displacement of the tibia in relation to the femur. (Photo contributor: David Effron, MD.)

Clinical Summary

While femoral fractures often occur secondary to serious trauma, they can be seen in low-energy injuries associated with the elderly, osteoporosis, or bone cancer. Diagnosis is usually evident on visualization and confirmed radiographically. Significant hematoma formation and blood loss is common. Patients with comminuted femoral shaft fractures are at risk for fat emboli syndrome. For distal fractures, it is important to rule out intra-articular involvement.

Management and Disposition

Initial management includes stabilization and evaluation for any life-threatening injuries. A large amount of blood loss (average 1000 mL) can occur; patients should have two large-bore intravenous lines and be cross-matched. Radiography should include the hip and knee. In-line traction may be required for initial stabilization, and to reduce blood loss and pain. This can be held temporarily in the acute setting with a Hare traction splint.

An open fracture is an orthopedic emergency; these patients require tetanus prophylaxis, antibiotic coverage, and emergent irrigation and debridement in the operating room. Orthopedic consultation should be obtained with any femur fracture since the majority requires operative fixation and stabilization.

Pearls

1. Pain can be referred. Any injury between the lumbosacral spine and the knee can be referred to the thigh or knee.
2. Vascular compromise can occur and should be suspected with an expanding hematoma, absent or diminished pulses, or progressive neurologic signs.
3. Femur fractures can mask the clinical findings of a hip dislocation; thus, radiographs of the pelvis, hips, and knees should be obtained routinely.

FIGURE 11.76 ■ **Midshaft Femur Fracture.** A closed midshaft femoral fracture. Note the deformity in the middle of the thigh. (Photo contributor: Daniel L. Savitt, MD.)



FIGURE 11.77 ■ **Femur Fracture.** Radiographic examination reveals a comminuted displaced distal femoral fracture. (Photo contributor: Cathleen M. Vossler, MD.)



FIGURE 11.78 ■ **Pathologic Femur Fracture.** A varus-angulated and mildly displaced fracture through an aggressive, moth-eaten, lytic lesion in the proximal left femoral diaphysis. (Reproduced with permission of Block J, Jordanov MI, Stack LB, Thurman RJ. The Atlas of Emergency Radiology. McGraw-Hill; 2013.)

TIBIAL-FIBULAR FRACTURES

Clinical Summary

Fibular fractures may be isolated or associated with tibia injuries. Isolated fibular fractures are caused by direct lateral leg trauma and are anatomically splinted by an intact tibia. Distal fibular fractures may include a disrupted ankle joint, as evidenced by a widened mortise on the AP radiograph. Tibial fractures are classified by their location, amount of displacement, and presence of comminution.

Compartment syndrome can be seen following a tibia fracture; distal neurovascular status should be documented. Suspect tibial fractures with trauma to the lower extremity, pain, and inability to bear weight.

The Maisonneuve fracture is a combination of an oblique proximal fibular fracture, disruption of the interosseous membrane and tibiofibular ligament distally, and a medial malleolar fracture or tear of the deltoid ligament. It occurs when an external rotational force is applied to the foot, producing a proximal third fibula fracture. Findings include tenderness at the medial and anterolateral ankle joint in combination with proximal fibular tenderness.



FIGURE 11.79 ■ **Tibial-Fibular Fracture.** Deformity associated with a midshaft tibial and fibular fracture. (Photo contributor: Kevin J. Knoop, MD, MS.)

Management and Disposition

Treatment of fibular fractures is dictated by the degree of pain and the involvement of the ankle joint. Nondisplaced fractures can be treated with an air cast; those with displacement should receive a sugar-tong splint and referred for short-term orthopedic evaluation. Treatment of a Maisonneuve fracture is most commonly operative.

Treatment of tibial fractures depends on whether they are open or closed, and on the degree of displacement. All open fractures require immediate orthopedic evaluation. Closed fractures that cannot be reduced may also need open reduction. Patients with isolated nondisplaced tibial fractures may be splinted, started on ice therapy, and referred for outpatient treatment. Displaced tibial fractures should also be evaluated by an orthopedics due to risk for compartment syndrome.

Pearls

1. Early follow-up is required for all tibial fractures owing to the risk of compartment syndrome.
2. The peroneal nerve crosses over the head of the fibula and is subject to injury with a Maisonneuve fracture.
3. Some patients with Maisonneuve fracture may complain only of ankle pain; always examine the proximal fibula in patients complaining of ankle pain.



FIGURE 11.80 ■ **Maisonneuve Fracture.** (**A**) Abnormal widening of the medial aspect of the ankle mortise and distal tibiofibular syndesmosis, as well as slight lateral subluxation of the talar dome. Because no "exit" fracture is present on the distal fibula, a fracture of the proximal fibula (**B**) was suspected and confirmed. (Reproduced with permission of Block J, Jordanov MI, Stack LB, Thurman RJ. The Atlas of Emergency Radiology. McGraw-Hill; 2013.)

FRACTURE BLISTERS

Clinical Summary

Fracture blisters are vesicles or bullae secondary to swelling from soft tissue injury. The most commonly affected areas include the tibia, ankle, and elbow. Patients note formation within 1 to 2 days after trauma and complain of pain, swelling, ecchymosis, and decreased range of motion. Complications include infection, deep venous thrombosis, and compartment syndrome.

Management and Disposition

Blisters are generally left intact, and the underlying fracture is treated.



FIGURE 11.81 ■ **Fracture Blisters.** This patient fell down four steps on the evening prior to presentation. Upon awakening the next morning, he noted ecchymosis, swelling, and blister formation. Radiographs revealed fracture of the fibula. (Photo contributor: Daniel L. Savitt, MD.)

Pearls

1. Blisters can be seen with other conditions, including barbiturate overdose and burns. In the setting of trauma, however, they frequently indicate an underlying fracture.
2. Blisters are managed in a similar fashion to second-degree burns.



FIGURE 11.82 ■ **Fracture Blisters.** Blisters associated with an underlying ankle fracture. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 11.83 ■ **Fracture Blisters.** Blisters associated with an underlying ankle fracture. (Photo contributor: Selim Suner, MD, MS.)

Clinical Summary

Rupture of the Achilles tendon occurs most frequently in middle-aged males involved in athletic activities, but patients with other systemic conditions, steroid injections, or fluoroquinolone use are predisposed. Rupture occurs 2 to 6 cm above the tendon's attachment to the calcaneus. Patients may hear or feel a pop. There is weakness when pushing off of the foot; pain, edema, and ecchymosis may develop. Note that loss of plantar flexion is not necessarily seen as there are other tendons that can compensate. Thompson test can be diagnostic of an Achilles rupture. The patient should be placed in a prone position, the knee and ankle flexed to 90 degrees, and the gastrocnemius muscle should be grasped and squeezed. If the Achilles tendon is even partially intact, then the foot will plantar flex; if completely ruptured, there will be no movement of the foot. Radiographic analysis should include a lateral view of the ankle as the Achilles tendon can sometimes be seen.



FIGURE 11.84 ■ **Achilles Tendon Rupture.** Note the loss of the normal resting plantar flexion due to right Achilles tendon rupture. Swelling is also apparent over the injury site. (Photo contributor: Kevin J. Knoop, MD, MS.)

Management and Disposition

Treatment is either operative or conservative. In either case, the extremity is immobilized in slight plantar flexion and the patient is made non-weight bearing upon ED discharge. Acute treatment also involves elevation, analgesia, and ice. These patients can be discharged home with follow-up with orthopedics.

Pearls

1. Approximately 25% of these injuries are initially misdiagnosed as ankle sprains.
2. Palpation of the tendon alone may not detect rupture, as the tendon sheath is often intact.



FIGURE 11.85 ■ **Thompson Test.** Compression of the gastrocnemius-soleus complex normally produces plantar flexion of the foot (**A**). If the tendon is completely ruptured, this will not occur (**B**).

ANKLE DISLOCATION

Clinical Summary

The ankle is a hinge joint with the talus sitting in the mortise formed by the distal tibia and distal fibula. Ankle dislocations require forces of great magnitude and usually the malleoli are also fractured. Posterior and lateral dislocations are the most common, but the ankle can also dislocate medially, superiorly, or anteriorly. A posteriorly dislocated ankle is locked in plantar flexion with the anterior tibia easily palpable. The foot has a shortened appearance, with the ankle very edematous. Anterior dislocations present with the foot dorsiflexed and elongated. Lateral dislocations present with the entire foot displaced laterally. Ankle dislocations are frequently associated with fractures and may be open.

Management and Disposition

A complete neurovascular examination should be performed and, if vascular compromise is present, the ankle should be emergently reduced, even if it is open. The skin may also be taut and can be at risk for necrosis. If time permits, radiography should include AP and lateral ankle views.

To reduce the ankle, gentle traction is applied to the foot with one hand cupping the heel and the other hand on the dorsal aspect of the foot while an assistant applies countertraction. Neurovascular status should be checked before and after any attempts at reduction or immobilization. Reduction usually requires conscious sedation or general anesthesia. Patients should be placed in a posterior splint with immediate referral to an orthopedic surgeon for hospitalization.



FIGURE 11.87 ■ **Posterior Ankle Dislocation.** The talus is dislocated posteriorly in relation to the tibia. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 11.86 ■ **Posterior Ankle Dislocation.** Deformity associate with posterior dislocation. (Photo contributor: Mark L. Madenwald, MD.)

Pearls

1. These injuries are commonly associated with malleolar fractures and often require open reduction and internal fixation.
2. Fifty percent of ankle dislocations are open and require surgical debridement.
3. The subtalar joint may also dislocate and appear clinically similar. The lateral x-ray will show overlap of the talus and calcaneus, while the AP mortise view will show an intact mortise.
4. The worse the ankle appears clinically and radiographically, the easier it is to reduce due to more severe ligamentous disruption.

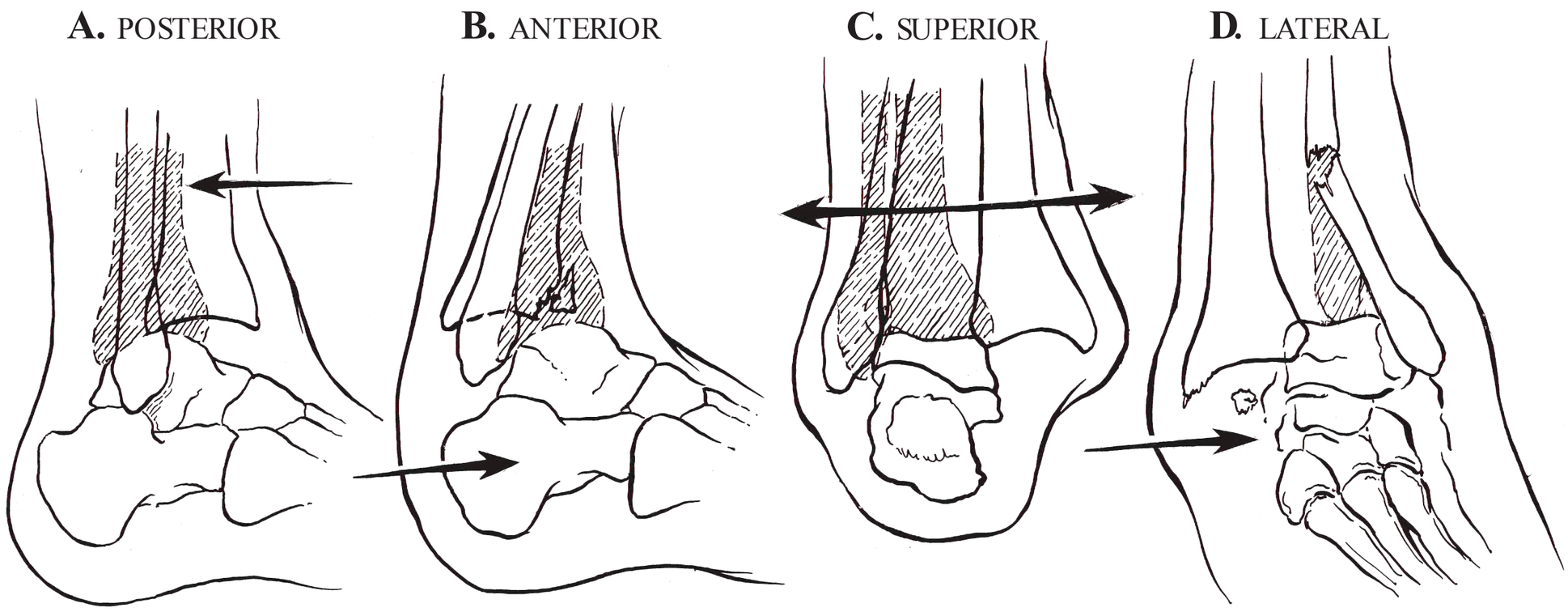


FIGURE 11.88 ■ **Ankle Dislocations.** This illustration depicts different types of ankle dislocations. Arrows denote direction of the injury force. (Adapted with permission from Simon R. Emergency Orthopedics: The Extremities. Norwalk, CT: Appleton & Lange; 1987: p 402.)



FIGURE 11.89 ■ **Lateral Ankle Dislocation.** The talus is laterally displaced in relation to the tibia. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 11.90 ■ **Subtalar Dislocation.** Complete dislocation of the hindfoot at the subtalar joints while playing basketball. Neurovascular status was intact, and the ankle, which was promptly reduced after x-ray, showed no associated fracture. (Photo contributor: Kevin J. Knoop, MD, MS.)

ANKLE FRACTURE

Clinical Summary

Most ankle fractures are isolated malleolar, but bimalleolar and trimalleolar occur in up to one-third. While there are numerous classification systems, the simplest is based on the radiographic appearance of these malleoli.

Management and Disposition

Essential treatment is directed at stability and exclusion of associated injuries. Neurovascular status (deep peroneal, superficial peroneal, medial and lateral plantar nerves, posterior tibial artery, and dorsalis pedis artery) should be assessed. The entire length of the fibula, including the proximal portion should be palpated to rule out additional fractures. All ankle fractures, with the exception of fibular avulsions, require immobilization by cast, or reduction followed by casting. With the exception of unimalleolar fractures, most require open reduction and fixation, thus orthopedic consultation is recommended. In the ED, fractures should be splinted with a posterior mold, kept non–weight bearing, elevated, and iced for 24 hours. Appropriate analgesia and consultation is addressed. The overall goal is to restore anatomic relationships, maintain them during healing, and institute early mobilization. Complications, although rare, include skin necrosis, osteomyelitis, osteoarthritis, and malunion.

Pearls

1. Postreduction arthritis can occur in up to 30%.
2. Avulsion fractures may be treated as stable ankle sprains if they are <3 mm in diameter, minimally displaced, and there is no evidence of significant ligamentous injury.



FIGURE 11.91 ■ **Trimalleolar Ankle Fracture.** (**A-C**) A tiny avulsion fracture of the medial malleolus tip, an oblique fracture of the lateral malleolus, and an oblique fracture of the distal tibia's posterior lip. (**D-E**) Reformatted CT images nicely demonstrate the same fractures. (Reproduced with permission of Block J, Jordanov MI, Stack LB, Thurman RJ. The Atlas of Emergency Radiology. McGraw-Hill; 2013.)









FIGURE 11.91 ■ **Trimalleolar Ankle Fracture.** (Continued)

ANKLE SPRAIN

Clinical Summary

Classification of these injuries is based on examination; radiographs are often not required to guide management. The most common mechanism is an inversion stress that injures, in order, the anterior talofibular, calcaneofibular, and posterior talofibular ligaments. The medial deltoid is the strongest ligament; therefore, isolated injuries are rare and medial ankle sprains are often associated with lateral malleolar or syndesmotic injuries.

Management and Disposition

Examination should include an anterior drawer test to assess the integrity of the anterior talofibular ligament. The Ottawa Ankle Rules recommend for the following: (a) tenderness over the lateral or medial malleolus, or (b) inability to bear weight for four steps both immediately postinjury and in the ED.

If able to bear weight, injuries are treated with ice packs, elevation, air cast or splint, and early mobilization. If unable to bear weight despite normal radiographs, use a posterior splint, crutches, and close follow-up with orthopedics.

Pearls

1. Ankle injuries are the most common musculoskeletal problem in emergency medicine.
2. Complications include instability, persistent pain, recurrent sprains, and peroneal tendon dislocation.
3. Both malleoli, the proximal fibula, and the fifth metatarsal should be examined for tenderness when evaluating a patient with an ankle sprain.



FIGURE 11.93 ■ **Medial Ankle Sprain.** An eversion injury in a professional athlete caused this rare isolated deltoid ligament sprain. Note localized ecchymosis. Tarsal tunnel syndrome developed over several weeks, taking months to resolve. (Photo contributor: Mimi Knoop.)



FIGURE 11.92 ■ **Ankle Sprain.** Note the dependent ecchymosis and swelling in this patient with a Grade 2 left lateral ankle sprain. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 11.94 ■ **Ankle Sprain.** Marked swelling and ecchymosis in a patient with a probable Grade 3 ankle sprain. (Photo contributor: Selim Suner, MD, MS.)

Clinical Summary

The calcaneus is the most frequently fractured tarsal bone. Patients present with severe heel pain in association with soft tissue swelling and ecchymosis extending to the arch. The normal contour of the heel can be distorted. Radiographs should include AP and lateral views of the foot and, if possible, a Harris axial view. Fractures can involve the tuberosities, the sustentaculum, or the body and are classified as intra- or extra-articular. Bohler angle should be calculated for all fractures involving the body to rule out a depression, as this will change management. It is normally between 20 and 40 degrees; if approaching or less than 20 degrees, a depressed fracture should be suspected.

Management and Disposition

CT scans should be obtained to further delineate fracture patterns and rule out involvement of the subtalar joint. Intra-articular fractures require orthopedic consultation; open reduction and internal fixation are usually needed.

Nondisplaced extra-articular fractures generally heal well with bulky compressive dressings, rest, ice, elevation, and non-weight bearing for 8 weeks. Orthopedic referral is necessary since some may require open reduction. Complications include fracture blisters, nonunion, and chronic pain.

Pearls

1. Calcaneal fracture warrants a diligent search for associated injuries. Twenty percent are associated with spinal fractures, 7% have contralateral calcaneal fractures, and 10% are associated with compartment syndromes. The subtalar joint is disrupted in 50% of cases. A high index of suspicion for thoracic aortic rupture and renal vascular pedicle disruption must be maintained.
2. Minimally displaced fractures of the anterior calcaneus are easily missed and should be suspected in a patient who does not recover appropriately from a lateral ankle sprain.
3. CT scanning is the optimal imaging technique for characterizing fracture dislocations, while MRI may be used to evaluate ligamentous injury.

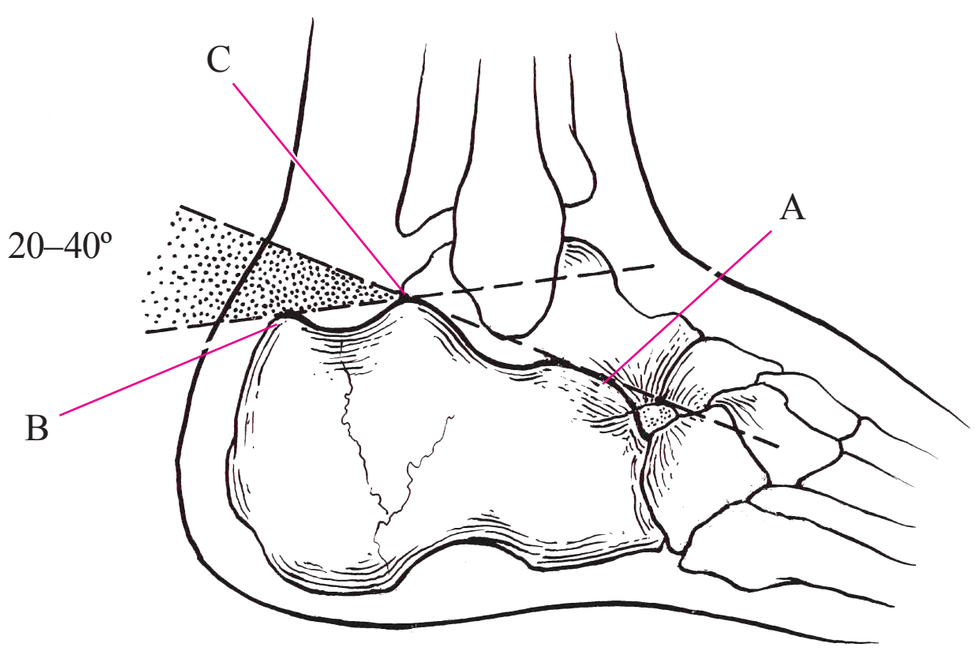


FIGURE 11.95 ■ **Calcaneus Fracture: Bohler Angle.** The Bohler angle is formed by the intersection of lines drawn tangentially to the anterior (**A**) and posterior (**B**) elements of the superior surface of the calcaneus (**C**). A normal angle is approximately 20 to 40 degrees.

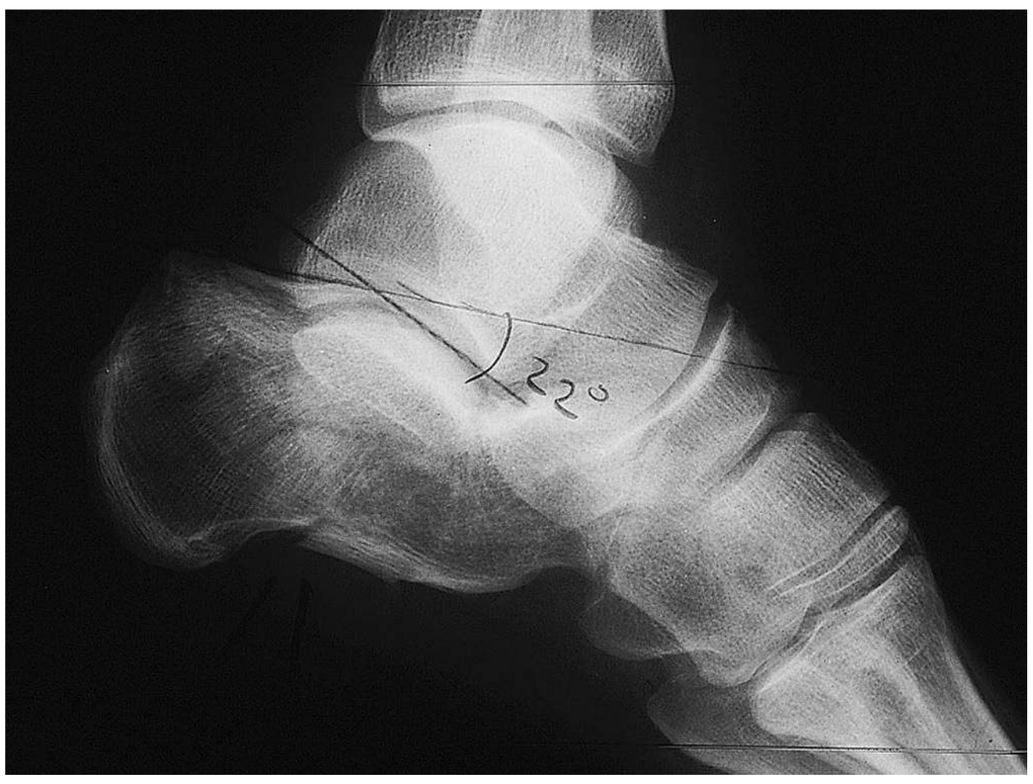


FIGURE 11.96 ■ **Calcaneus Fracture.** This patient fell from a ladder and struck his heel. A cortical step-off is seen on the inferior aspect of the calcaneus. The Bohler angle has been calculated at approximately 22 degrees. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 11.97 ■ **Bilateral Calcaneal Fracture.** A fall from a ladder caused bilateral calcaneal fractures. Note swelling and ecchymosis. (Photo contributor: Lawrence B. Stack, MD.)

FRACTURES OF THE FIFTH METATARSAL BASE

Clinical Summary

Patients complain of pain, swelling, decreased range of motion, and tenderness over the lateral aspect of the midfoot. Fractures of the fifth metatarsal base have been generically referred to as Jones fractures. However, acute fractures can be divided into two types, depending on their anatomic location. Treatment is determined by this delineation.

The classic Jones fracture is a transverse fracture of the fifth metatarsal at the metadiaphyseal-diaphyseal junction, just distal to the fourth and fifth intermetatarsal joint. It occurs when a force is applied to a plantar flexed and inverted foot.

This is not to be confused with an avulsion fracture of the fifth metatarsal base, resulting from sudden foot inversion. The avulsion injury is caused by traction on the lateral cord of the plantar fascia.

Management and Disposition

The patient with a Jones fracture should be discharged in a posterior splint and crutches and be non-weight bearing for 6 to 8 weeks. Surgical treatment is sometimes required since there is a risk of nonunion.

The avulsion fracture usually heals rapidly and seldom leads to permanent disability. This patient can be discharged in a hard-soled shoe or walking cast for 2 to 3 weeks and can bear weight as tolerated. A significantly displaced fracture may require operative intervention. Both types of patients should be referred to a musculoskeletal specialist as an outpatient.

Pearls

1. The original description of these fractures was by Sir Robert Jones, who personally sustained this injury while dancing. The avulsion fracture is sometimes referred to as a dancer's fracture.
2. The classic Jones fracture has a high incidence of delayed healing and nonunion.

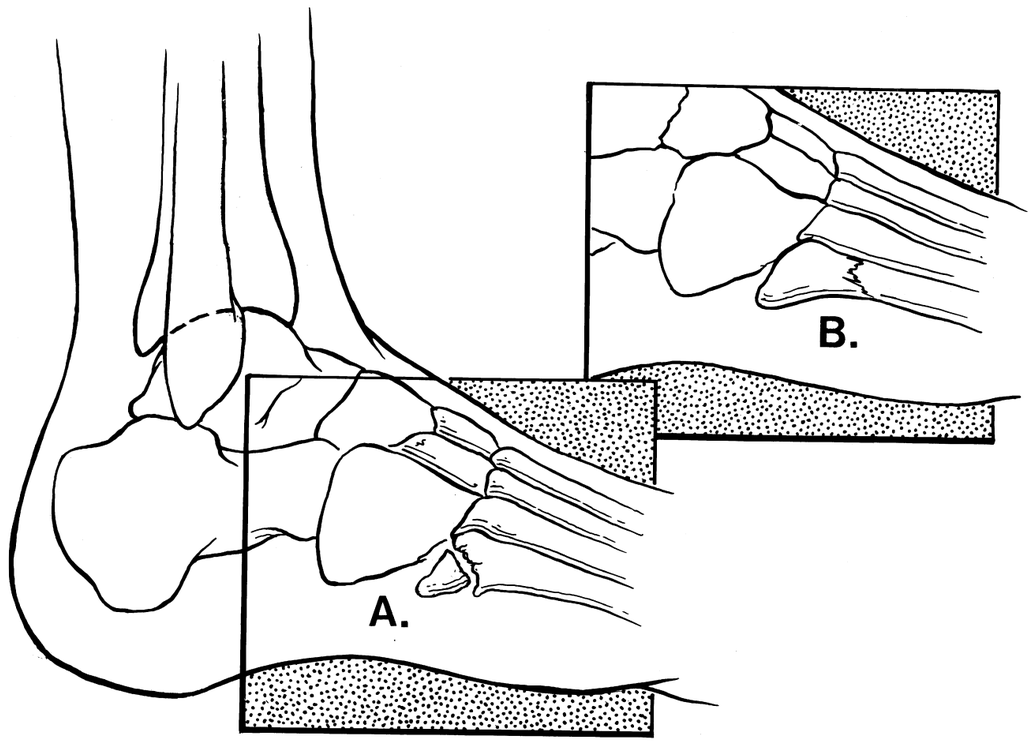


FIGURE 11.99 ■ **Fifth Metatarsal Base Fractures.** This illustration depicts an avulsion fracture (**A**) and a classic Jones fracture (**B**).



FIGURE 11.98 ■ **Jones Fracture.** This patient sustained an injury of the fifth metatarsal and presented with pain and swelling over this site. His radiograph revealed a fracture. (Photo contributor: Cathleen M. Vossler, MD.)



FIGURE 11.100 ■ **Jones Fracture.** Radiograph with typical appearance for a diaphyseal fracture of the fifth metatarsal base. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 11.101 ■ **Fifth Metatarsal Avulsion Fracture.** Radiograph illustrating an avulsion-type fracture of the fifth metatarsal base. (Photo contributor: Alan B. Storrow, MD.)

LISFRANC FRACTURE-DISLOCATION

Clinical Summary

The Lisfranc joint (tarsometatarsal joint) connects the midfoot and forefoot. It is defined by the articulation of the bases of the first three metatarsals with the cuneiforms and the fourth and fifth metatarsals with the cuboid. The Lisfranc ligament anchors the second metatarsal base to the medial cuneiform. Disruption of the Lisfranc joint is typically associated with high-energy mechanisms; however, they may occur with less force. Clinical presentation is variable, but severe midfoot pain and the inability to bear weight are usually present. Radiographs may reveal displacement of the metatarsals in one direction (homolateral) or a split, usually between the first and second metatarsals (divergent).



FIGURE 11.102 ■ **Lisfranc Fracture-Dislocation.** This patient presented with extreme midfoot pain and swelling. (Photo contributor: Lawrence B. Stack, MD.)

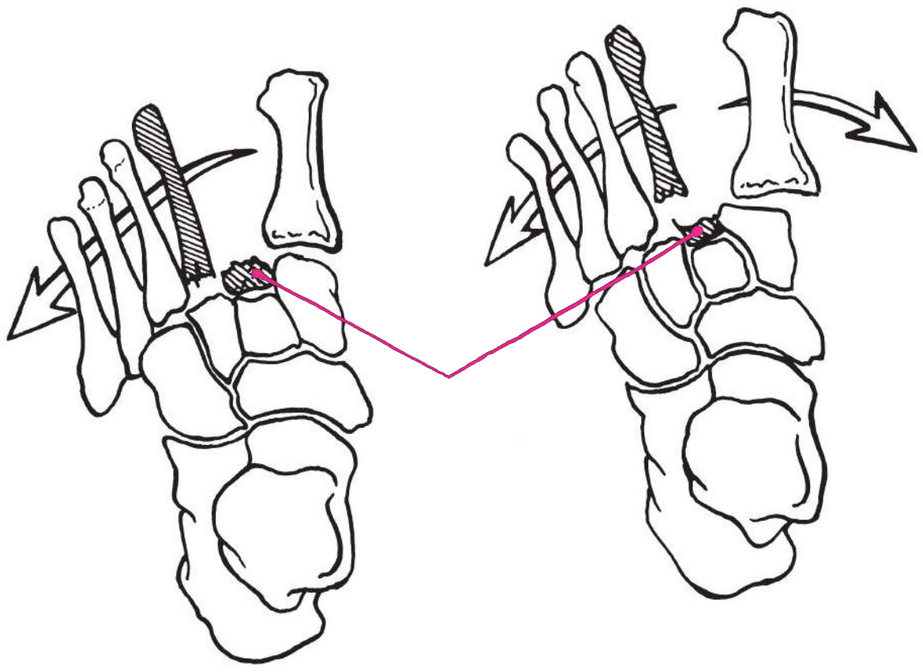


FIGURE 11.103 ■ **Lisfranc Fracture-Dislocations.** Two different types of Lisfranc fracture-dislocations. Homolateral (left) and divergent (right).

Management and Disposition

Meticulous evaluation of foot radiographs is the key to diagnosis. There are three radiographic findings that suggest a Lisfranc injury. Normally, the medial aspect of the second metatarsal should align with the medial borders of the middle cuneiform on the dorsoplantar (DP) foot x-ray. Second, on a DP and an oblique view, there should not be any widening between the first and second metatarsals. Third, on the oblique view of the foot, the medial aspect of the fourth





FIGURE 11.104 ■ **Homolateral Lisfranc Fracture-Dislocation.** (**A-C**) DP, oblique, and lateral views demonstrate the radiographic hallmark: lateral subluxation of the second metatarsal base in relationship to the middle cuneiform. The first and third metatarsal bases have also migrated laterally. (**C**) The lateral view demonstrates superior subluxation in relationship to the cuneiform bones. (Reproduced with permission of Block J, Jordanov MI, Stack LB, Thurman RJ. The Atlas of Emergency Radiology. McGraw-Hill; 2013.)

metatarsal should align with the medial aspect of the cuboid. A disruption of these relationships is suggestive of a Lisfranc injury and warrants orthopedic evaluation in the ED. Closed reduction can be attempted; however, due to significant ligamentous disruption, the injury is often unstable and necessitates operative stabilization. Tenderness over the Lisfranc complex with normal radiographs can reflect a strain of the complex. Weight-bearing radiographs may unmask joint instability, but is often not tolerated in the acute setting.

Pearls

1. Early recognition of Lisfranc fracture-dislocations is facilitated by assessing for normal bony alignments on x-ray.
2. Fractures of the second metatarsal base are considered pathognomonic of a Lisfranc injury since these fractures are often associated with Lisfranc ligament disruption.



FIGURE 11.105 ■ **Lisfranc Fracture-Dislocation.** A divergent Lisfranc fracture-dislocation. Note the disruption of the alignment of the second metatarsal and the medial cuneiform. Sometimes these injuries are not as apparent and comparison radiographs are necessary. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 11.106 ■ **Lisfranc Fracture-Dislocation, weight-bearing view.** Weight-bearing DP view of both feet reveals a subtle malalignment of the right midfoot with subtle lateral subluxation of the second metatarsal base. The alignment was normal on the non–weight bearing view. Notice the normal alignment of the medial cortices of the second cuneiform and second metatarsal on the left. (Reproduced with permission of Block J, Jordanov MI, Stack LB, Thurman RJ. The Atlas of Emergency Radiology. McGraw-Hill; 2013.)

Clinical Summary

Major trauma may result in partial or complete removal of a limb. The high kinetic injury, crush, or avulsion forces required often cause extensive damage and heavy contamination. Other significant injuries to the thorax and abdominal cavities should be considered, and may be life-threatening.

Management and Disposition

Advanced trauma life support protocols should be rapidly initiated. The injury should be managed as an open fracture with appropriate antibiotics and tetanus prophylaxis. The amputated part should be kept clean, irrigated with sterile saline, wrapped in a sterile dressing, placed in a plastic bag, and put on ice. With the possible exception of minor digit amputations, patients should be admitted under trauma and orthopedic consultation for close monitoring of neurologic and vascular status.

While reimplantation is often not possible due to tissue loss and contamination, all patients should receive consideration. Young, healthy individuals with sharp, guillotine injuries without significant avulsion or crushing damage are the best candidates. Radiographs may help delineate the exact spot of injury and reveal associated dislocations or fractures.

Pearls

1. Cooling the amputated part will increase viability from approximately 6 to 8 hours to 12 to 24 hours.
2. Postreimplantation limb shortening may create significant disability. Proper use of postinjury prosthetics may be the better option.



FIGURE 11.107 ■ **Lower Extremity Amputation.** A below-the-knee amputation from a motorcycle accident. These injuries are often associated with significant, and potentially life-threatening, abdominal or thoracic trauma. (Photo contributor: Selim Suner, MD, MS.)



FIGURE 11.108 ■ **Lower Extremity Amputation.** Amputation from a motor vehicle accident. (Photo contributor: Selim Suner, MD, MS.)



FIGURE 11.109 ■ **Finger Amputation.** Digit avulsion and tendon rupture. (Photo contributor: Selim Suner, MD, MS.)



FIGURE 11.110 ■ **Table Saw Amputation.** The high kinetic injury of a table saw can produce significant avulsion forces and contamination. (Photo contributor: Selim Suner, MD, MS.)

Chapter 12

EXTREMITY CONDITIONS

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Paronychia. (Photo contributor: Lawrence B. Stack, MD.)

CELLULITIS

Clinical Summary

Cellulitis is a common infection of the skin or subcutaneous tissues with characteristic findings: erythema with poorly defined borders, edema, warmth, pain, and limitation of movement. Fever and constitutional symptoms may be present and are commonly associated with bacteremia. Predisposing factors include trauma, lymphatic or venous stasis, immunodeficiency (including diabetes mellitus), and foreign bodies. Common etiologic organisms include group A β-hemolytic Streptococcus and Staphylococcus aureus in nonintertriginous skin, and gram-negative organisms or mixed flora in intertriginous skin and ulcerations. In immunocompromised hosts, Escherichia coli, Klebsiella species, Enterobacter species, and Pseudomonas aeruginosa are common. In recent years, there has been a dramatic increase in the incidence of community-acquired methicillin-resistant S aureus (CA-MRSA), particularly in cellulitis associated with a cutaneous abscess.



FIGURE 12.1 ■ **Cellulitis.** Cellulitis of the left leg characterized by erythema and mild swelling. (Photo contributor: Frank Birinyi, MD.)

The differential diagnosis includes deep venous thrombosis (DVT), venous stasis, erythema nodosum, septic or inflammatory arthritis/bursitis, and allergic reactions.

Management and Disposition

Treatment of minor cases commonly consists of immobilization, elevation, analgesia, and oral β-lactam antibiotics with reevaluation in 48 hours. The increase in the incidence of CA-MRSA has prompted some, especially in highly endemic areas, to advocate coverage with trimethoprim/sulfamethoxazole or other agents (refer to current recommendations) in addition to conventional β-lactam antibiotics. Admission and parenteral administration of antibiotics may be necessary for immunocompromised or toxic-appearing patients, or those who do not respond to outpatient therapy.

Pearls

1. Rapidly progressive cellulitis or one that progresses despite treatment with β-lactam antibiotics should raise suspicion for CA-MRSA or deeper infections such as fasciitis.
2. Known risk factors for CA-MRSA include military personnel, prison inmates, and competitive sports players.
3. Routine blood or leading-edge cultures in nontoxic patients are generally low yield.

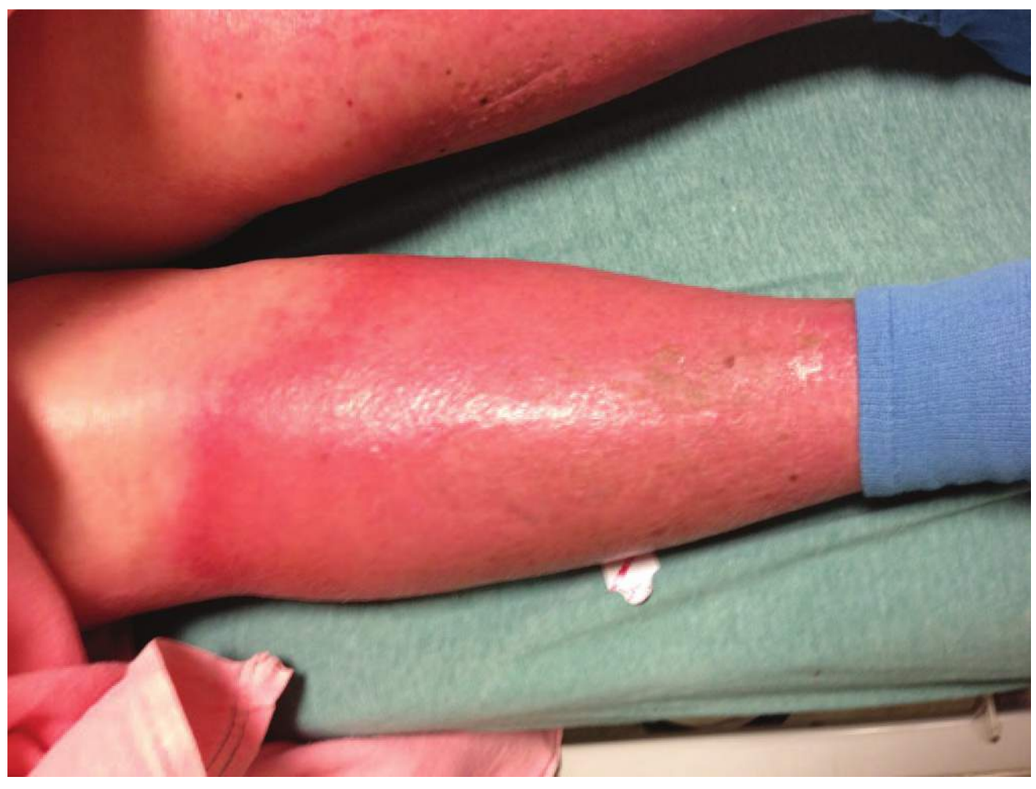


FIGURE 12.2 ■ **Cellulitis.** Cellulitis of the right lower extremity characterized by sharply demarcated erythema and edema. (Photo contributor: Robert Tubbs, MD.)

Clinical Summary

A felon is a pyogenic infection of the distal digital pulp space, with pus collecting in the spaces formed by the vertical septa anchoring the pad to the distal phalanx. This condition is characterized by severe pain, exquisite tenderness, and tense swelling of the distal digit with erythema. There may be a visible collection of pus or palpable fluctuance. Complications include deep ischemic necrosis, osteomyelitis, septic arthritis, and suppurative tenosynovitis. The differential diagnosis includes paronychia, herpetic whitlow, and hematoma following traumatic injury.

Management and Disposition

Incision and drainage is generally necessary to treat a felon. To ensure complete drainage of the abscess cavity, all affected compartments should be entered. The packing of the abscess space is made with a small, loose-fitting wick to facilitate drainage. Oral antibiotics directed against gram-positive organisms should be used for 10 days and the packing removed or replaced after 24 to 48 hours. Consider treatment for CA-MRSA in addition to standard coverage in highly endemic areas.

Pearls

1. Do not extend the incision proximal to the distal flexion crease.
2. Incisions should be made dorsal to the neurovascular bundle; the pincer surfaces (radial aspects of the index and long fingers and ulnar aspect of the thumb and small finger) should be avoided when possible.
3. "Hockey stick" and "fish mouth" incisions are associated with increased occurrence of unnecessary sequelae and are not recommended.
4. If there is radiographic evidence of osteomyelitis, or concern for tenosynovitis, consultation with a hand surgeon is required.



FIGURE 12.3 ■ **Felon.** Purulence, swelling, and erythema at the center of the palmar pad. (Photo contributor: Daniel L. Savitt, MD.)

GANGRENE

Clinical Summary

Gangrene denotes tissue with lost blood supply and undergoing necrosis. The term dry gangrene is used for tissues undergoing sterile ischemic coagulative necrosis. Patients with atherosclerotic disease and diabetes are at risk for the development of dry gangrene, usually as a result of embolization to the forefoot or toe. Wet gangrene is associated with bacterial proteolytic decomposition, and is characterized by its moist appearance, frequently with blistering.

Management and Disposition

Hospitalization is usually required. The treatment consists of amputation, debridement, and antibiotic therapy as needed. Wet gangrene requires emergent surgical consultation. Underlying vascular pathology must be evaluated by arteriography and corrected surgically or endovascularly. Patients with systemic toxicity should be resuscitated aggressively.



FIGURE 12.4 ■ **Dry Gangrene.** Dry gangrene of the toes showing the areas of total tissue death, appearing as black and lighter shades of discoloration of the skin demarcating areas of impending gangrene. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. Obtain radiographs to help rule out clostridial myonecrosis (gas gangrene, see related item) and osteomyelitis.
2. Aggressive surgical debridement is necessary for cure in most cases of wet gangrene.



FIGURE 12.5 ■ **Dry Gangrene.** Complete tissue death characterized by black, desiccated tissue, and lighter areas demarcating areas of impending gangrene. (Photo contributor: David Effron, MD.)



FIGURE 12.6 ■ **Wet Gangrene.** Note the moist appearance and blistering due to bacterial proteolytic decomposition of gangrenous tissue. (Photo contributor: Robert Tubbs, MD.)

Clinical Summary

This infection causes rapid necrosis and liquefaction of fascia, muscle, and tendon. Most involve Clostridium perfringens; Streptococcus pyogenes accounts for the majority of remaining cases. Myonecrosis is classically associated with trauma (including surgery) and diabetes. There is edematous bronze or purple discoloration, flaccid bullae with watery brown nonpurulent fluid, and a foul odor. The classic clinical presentation is pain out of proportion to physical findings. Low-grade fever and other systemic signs are also typically present, and may develop rapidly into shock.

Crepitance and appearance of gross pockets of air in the tissue are appreciated, but may not be present early. The incubation period for clostridia ranges between 1 and 4 days, but it can be as early as 6 hours. Decreased tissue oxygen tension along with wound contamination is required for the infection to progress. Crepitant cellulitis, synergistic necrotizing cellulitis, acute streptococcal hemolytic gangrene, and streptococcal myositis are some conditions that may be mistaken for clostridial myositis. Often, surgical exploration of the fascia and muscle is required to make the correct diagnosis.

Management and Disposition

Aggressive resuscitation should be initiated, and consideration given to packed red blood cell transfusion. Broad-spectrum antibiotics, especially clindamycin, in conjunction with penicillin G, are given urgently. Tetanus prophylaxis must not be overlooked. Surgical debridement or amputation is the mainstay of therapy. Hyperbaric oxygen may have a synergistic effect in preventing the progression of infection and toxin production.

Pearls

1. Shock and multiorgan failure may be rapidly progressive. Mortality is 80% to 90% if untreated, 10% to 25% when treated appropriately.
2. Clindamycin may improve survival by inhibiting toxin production.
3. Gram stain of gram-positive bacilli with a relative lack of leukocytes may rapidly confirm suspected clostridial myonecrosis.



FIGURE 12.7 ■ **Gas Gangrene.** A foot with significant areas of gangrene and sloughing skin. (Photo contributor: David Kaplan, MD.)



FIGURE 12.8 ■ **Gas Gangrene.** Radiograph of the foot in Fig. 12.7 exhibiting pockets of soft tissue gas tracking up the dorsal surface. (Photo contributor: Douglas Nilson, MD.)

NECROTIZING FASCIITIS

Clinical Summary

This uncommon, severe infection involves the subcutaneous soft tissues, including the superficial and deep fascial layers, with early skin sparing and late muscle involvement. It is most commonly seen in the lower extremities, abdominal wall, and perianal and groin area, as well as in postoperative wounds. It is commonly spread from a trauma site, surgical wound, abscess, or decubitus ulcer. Alcoholism, parenteral drug abuse, and diabetes are predisposing factors. Pain, tenderness, erythema, swelling, warmth, shiny skin, lymphangitis, and lymphadenitis are early findings. Later, there is rapid progression of bullae with clear pink or purple fluid and cutaneous necrosis. The skin becomes anesthetic, and subcutaneous gas may be present. Systemic toxicity may be manifest by fever, dehydration, leukocytosis, and frequently positive blood cultures. Type I includes anaerobic species (Bacteroides and Peptostreptococcus) and type II includes either group A streptococci alone or group A streptococci with S aureus.

Management and Disposition

Prompt diagnosis is critical; if made within 4 days from symptom onset, the mortality rate is reduced from ~50% to 12%. Initial treatment involves resuscitation with volume expansion, operative debridement, and prompt initiation of broad-spectrum antibiotics.

Pearls

1. Intravenous calcium may be necessary to reverse hypocalcemia from subcutaneous fat necrosis.
2. Radiographs may detect nonpalpable subcutaneous gas.
3. Hemolysis and disseminated intravascular coagulation (DIC) may be present.
4. Community-acquired MRSA has been implicated as a causative organism.



FIGURE 12.9 ■ **Necrotizing Fasciitis.** Markedly swollen, dusky, erythematous arm with necrotizing fasciitis in an IV drug user. (Photo contributor: Alexis Lawrence, MD.)



FIGURE 12.10 ■ **Necrotizing Fasciitis.** Radiograph of the forearm in Fig. 12.9 showing extensive subcutaneous gas. (Photo contributor: Alexis Lawrence, MD.)



FIGURE 12.11 ■ **Necrotizing Fasciitis.** Large cutaneous bullae on the leg of this patient with necrotizing fasciitis. Note the dark purple fluid in the bullae. (Photo contributor: Lawrence B. Stack, MD.)

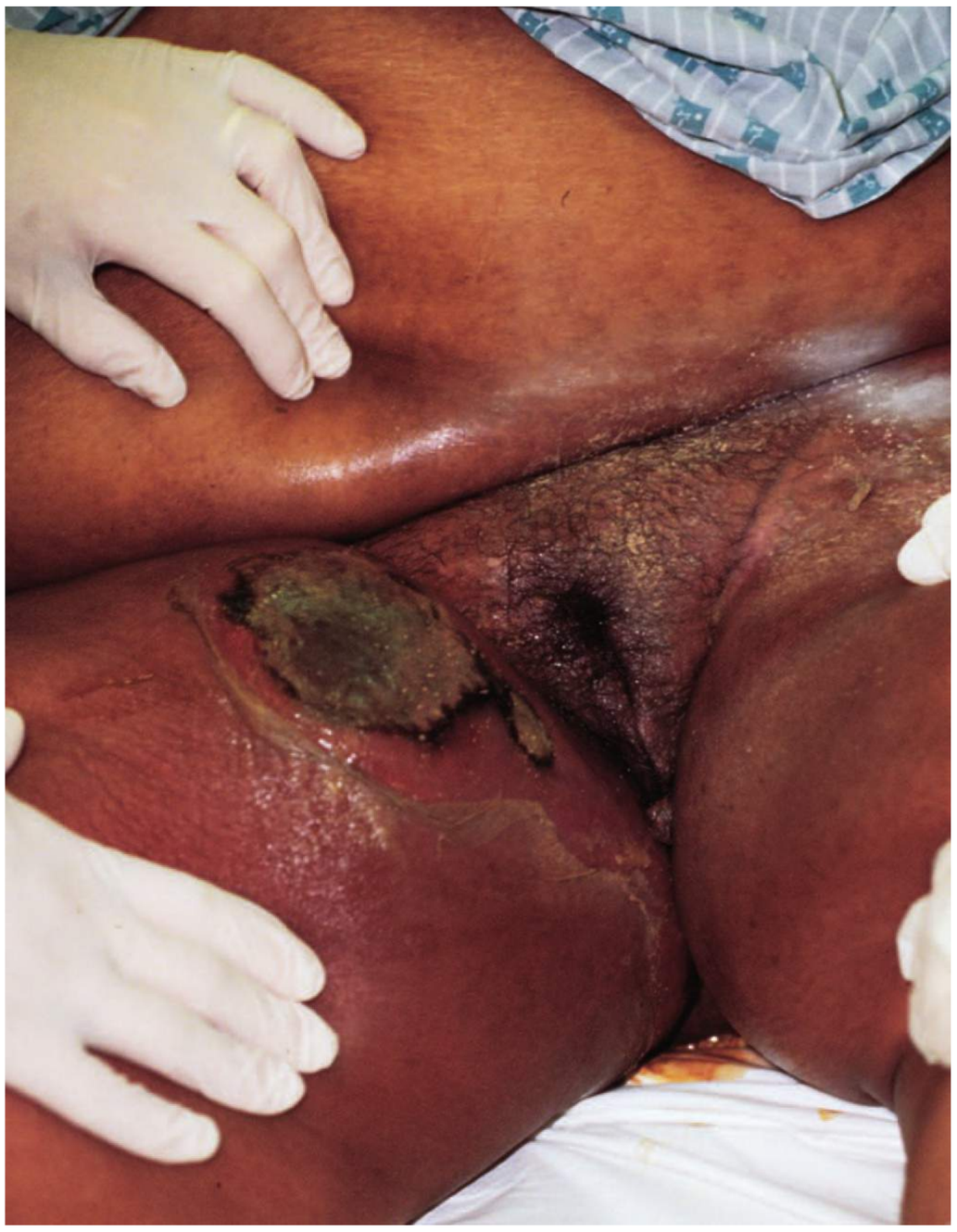


FIGURE 12.12 ■ **Necrotizing Fasciitis.** Necrotizing fasciitis with cutaneous necrosis in the inner thigh. (Photo contributor: Lawrence B. Stack, MD.)

CRYSTAL-INDUCED SYNOVITIS (GOUT AND PSEUDOGOUT)

Clinical Summary

Gout is an inflammatory disease characterized by deposition of sodium urate monohydrate crystals in cartilage, subchondral bone, and periarticular structures. An acute attack is characterized by sudden onset of monoarticular arthritis (commonly in the metatarsophalangeal joint of the great toe) with swelling, erythema, and tenderness. The deposits of crystals about the joint produce a chronic inflammatory response termed a tophus.

In pseudogout, calcium pyrophosphate dihydrate (rod- or rhombus-shaped, weakly birefringent) crystals are deposited. Although any joint may be involved, knees and wrists are most common. Presenting signs and symptoms are identical with gout, but formation of tophi is not seen. Low-grade fever, pain, and erythema are common to both entities.

Cellulitis and septic arthritis must be excluded. In the synovial fluid cell count, white blood cell count of 2000 to 50,000 with PMN predominance is expected (Table 1), although it may be significantly higher. The diagnosis is confirmed with urate or calcium pyrophosphate dihydrate crystals on polarized microscopy (see Microscopic chapter) coupled with negative Gram stain and cultures.

Management and Disposition

Nonsteroidal anti-inflammatories are used with excellent results acutely, along with joint immobilization and rest. Oral or intravenous (IV) colchicine is an alternative, but can cause nausea, vomiting, and diarrhea, as well as serious toxicities, including bone marrow suppression, neuropathy, myopathy, and death (particularly when given IV). Systemic and intra-articular corticosteroids and adrenocorticotropic hormone (ACTH) are alternatives. Allopurinol or probenecid is used in the chronic management of gout and plays no role in acute treatment.

Pearls

1. Serum urate may be normal in acute gout.
2. Acute attacks may be triggered by minor trauma, diuretic or salicylate use, alcohol, or diet.
3. Punched-out subchondral bone lesions may be seen on radiography in chronic tophaceous gout. Chondrocalcinosis may be seen in pseudogout.
4. Synovial fluid cell counts alone cannot be used to differentiate crystalline arthropathies from infectious etiologies.



FIGURE 12.13 ■ **Podagra.** Foot with erythematous base of great toe classic for gout ("podagra"). (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 12.14 ■ **Gout.** Markedly enlarged index finger proximal interphalangeal joint showing classic tophaceous gout. (Photo contributor: Robert Tubbs, MD.)



FIGURE 12.15 ■ **Pseudogout.** Markedly swollen left knee in a patient with an acute exacerbation of pseudogout. (Photo contributor: Robert Tubbs, MD.)

TABLE 12.1 ■ SYNOVIAL FLUID ANALYSIS

	Normal	Septic Arthritis	Gout	Pseudogout
Appearance	Clear	Purulent, turbid	Turbid	Turbid
WBC	<200	Usually >50,000	2000-50,000	2000-50,000
PMN	<25%	>75%	>50%	>50%
Special Tests/ Crystals	Normal "string sign"	+ Gram stain, culture positive	Negative birefringence, needle crystals	Positive birefringence, rhomboid crystals

INGROWN TOENAIL (ONYCHOCRYPTOSIS)

Clinical Summary

This condition is the result of impingement and puncture of the medial or lateral nail fold epithelium by the nail plate, allowing growth into the dermis. Tenderness and swelling of the nail fold is followed by granulation tissue causing sharp pain, erythema, and further swelling. If not promptly treated, the granulation tissue becomes epithelialized, preventing elevation of the nail above the medial or lateral nail groove. Often, there is secondary bacterial or fungal infection. Risk factors include cutting the nail too short, tightly fitting shoes, and trauma. The differential diagnosis includes paronychia, felon, and tumor.

Management and Disposition

Early treatment: Elevation of the nail out of fold and placement of gauze under it to prevent contact, in conjunction with warm soaks, is the initial therapy.

Late treatment: Surgical management involves removal of part of the nail and the inflamed tissue, and sometimes destruction of the involved nail matrix. The lateral nail is removed followed by packing of the paronychial fold with petroleum gauze or other nonadherent dressing. Follow-up by a podiatrist until growth of the nail plate is complete should be considered. The destruction of the nail matrix is required only in patients with recurrent infected ingrown toenails and is not part of routine emergency care.

Pearls

1. Ingrown toenail is most common in the great toe.
2. Use of antibiotics is not a substitute for surgical excision and will result in only transient improvement of symptoms.



FIGURE 12.16 ■ **Ingrown Toenail.** Ingrown toenail on the lateral aspect of the great toe with granulation tissue and erythema. (Photo contributor: Selim Suner, MD, MS.)

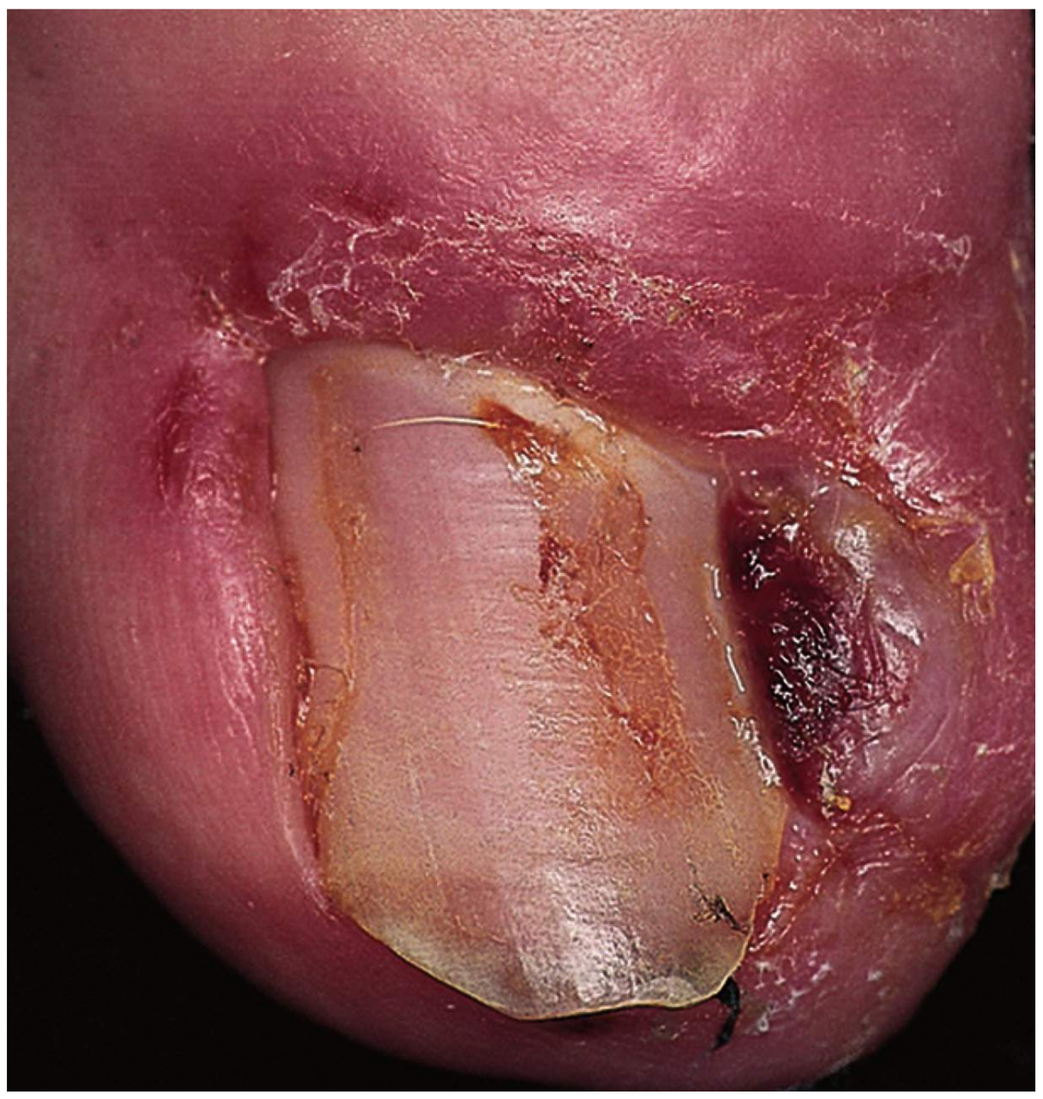


FIGURE 12.17 ■ **Ingrown Toenail.** An ingrown toenail on the medial aspect of the left great toe. (Photo contributor: Frank Birinyi, MD.)

Clinical Summary

Inflammation of lymphatic channels in the subcutaneous tissues is commonly caused by spread of local bacterial infection; Group A β-hemolytic streptococcal species are the most frequently implicated. Lymphangitis is characterized by red linear streaks extending, within 24 to 48 hours, from a primary site of infection (eg, abscess, cellulitis) to regional lymph nodes (eg, axilla, groin). The lymph nodes are often enlarged and tender. The differential diagnosis includes cellulitis, trauma, and superficial thrombophlebitis.

Management and Disposition

Rest, elevation, immobilization, and antibiotics are the initial treatment. Coverage for Streptococcus and Staphylococcus is appropriate. Toxic-appearing patients require admission for parenteral antibiotics. Any patient sent home with oral antibiotics should be followed up in 24 to 48 hours. Patients who subsequently do not show improvement require admission for parenteral antibiotic therapy. Consider treatment for CA-MRSA in addition to standard skin coverage in highly endemic areas.

Pearls

1. Consider Pasteurella multocida with cat and dog bites, Spirillum minus with rat bites, and Mycobacterium marinum in association with swimming pools and aquaria.
2. Chronic lymphangitis may be associated with mycotic, mycobacterial, and filarial infection.
3. Aspiration of the leading edge is generally not helpful for acute management.



FIGURE 12.18 ■ **Lymphangitis.** Severe lymphangitis extending from metacarpophalangeal wound up the arm from a "fight bite." The red streak extends from the hand to the axilla, extending along lymphatic channels. (Photo contributor: Selim Suner, MD, MS.)



FIGURE 12.19 ■ **Lymphangitis.** The red streak extends from ankle to groin along lymphatic channels. The site of infection was the great toe. (Photo contributor: Liudvikas Jagminas, MD.)



FIGURE 12.20 ■ **Lymphangitis.** Lymphangitis in the upper extremity, in this case from wrist to upper arm, commonly arises from nail biting. (Photo contributor: R. Jason Thurman, MD.)

LYMPHEDEMA

Clinical Summary

Lymphedema occurs from obstruction of lymphatic channels and is associated with malignancy, radiation, trauma, surgery, inflammation, infection, parasitic invasion, DVT, paralysis, renal insufficiency, heart failure, cirrhosis, and malnutrition. Lymphedema is characterized by painless pitting edema, fatigue, increase in limb size (particularly during the day), and presence of lymph vesicles. The skin becomes thickened and brown in the late stages.

Management and Disposition

Elevation, pneumatic compression boots and firm elastic stockings, maintenance of healthy skin, and avoidance of cellulitis and lymphangitis are the mainstays of symptomatic treatment. Treatment of the underlying disease may be curative.

Pearls

1. The dorsum of the toes and feet are always involved in lymphedema, unlike other causes of edema.
2. Careful examination for heart failure and screening for renal insufficiency should be completed for all patients.
3. Patients with chronic lymphedema are at risk for development of a rare secondary malignancy, lymphangiosarcoma, characterized by red-blue or purple macular or papular lesions.



FIGURE 12.21 ■ **Lymphedema.** Pitting edema is seen in a woman with lymphedema of the lower extremities. Note how the impression of the thumb remains on the foot in this patient with lymphedema. (Photo contributor: Selim Suner, MD, MS.)

Clinical Summary

Bursitis is an inflammatory reaction in a fluid-filled synovial sac, commonly over the subacromial, prepatellar, olecranon, or trochanteric bursa. It is associated with repetitive motion, trauma, or infection. The fluid collection may be bacterial (septic bursitis), gouty, or, most commonly, inflammatory. It produces pain, tenderness, swelling, warmth, and limited range of motion. It is critical to differentiate septic from benign inflammation.

Since bursitis does not involve the intra-articular space, signs and symptoms should be isolated to the bursal area. Typically, intra-articular involvement is associated with pain on minor range of motion, while the discomfort of bursitis occurs with skin and synovial sac stretching at extreme ranges of joint movement. When this differentiation is difficult, fluid aspiration and analysis for cell count, Gram stain, protein, glucose, and polarized microscopy (see Crystal-Induced Synovitis) may be helpful. Fluid with >50,000 cells per cubic millimeter, polymorphonuclear neutrophil predominance, increased protein, reduced glucose, and a positive Gram stain are associated with bacterial infection.

Management and Disposition

Rest, bulky compression dressings, and nonsteroidal anti-inflammatory drugs (NSAIDs) are initially used. Bursal injection of local anesthetics mixed with corticosteroids can be considered if septic bursitis has been ruled out, usually in patients who have failed treatment with NSAIDs. Reducing the effusion volume by aspiration may provide temporary relief, although it often recurs. Septic bursitis requires aspiration, gram-positive antibiotic coverage, and consideration of open incision and drainage by orthopedic surgery. Most patients can be treated as outpatients with follow-up.



FIGURE 12.22 ■ **Olecranon Bursitis.** Enlarged olecranon bursa in this patient with bursitis. (Photo contributor: Selim Suner, MD, MS.)

Pearls

1. Septic joint infections in patients with cancer, who are taking corticosteroids, or who are IV drug users may have lower synovial fluid leukocyte counts (<30,000/mm^3) than usual (>50,000/mm^3).
2. Bursal fluid from a septic bursitis typically has a lower nucleated cell count than septic joint fluid; lower limits of 2000/mm^3 have been proposed.
3. S aureus is the most common etiologic agent.

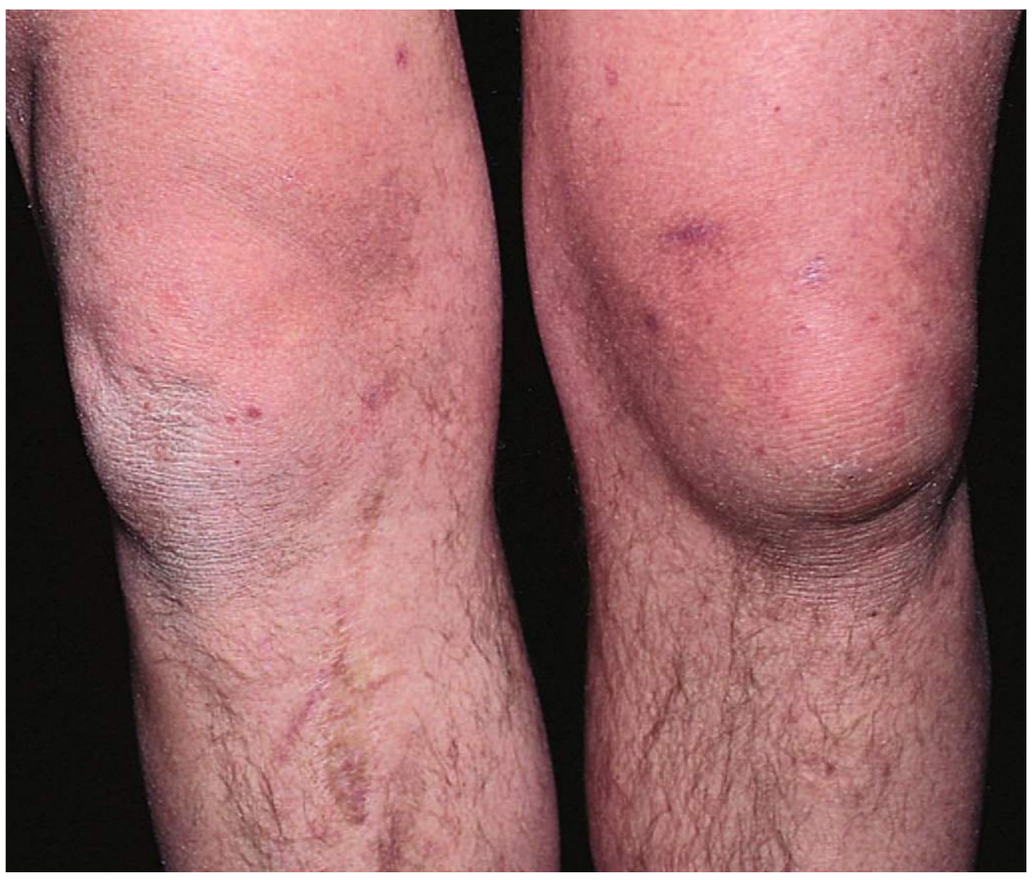


FIGURE 12.23 ■ **Prepatellar Bursitis.** Local bursal swelling is evident over the left knee. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 12.24 ■ **Septic Prepatellar Bursitis.** Erythematous, enlarged bursa in this patient with septic prepatellar bursitis. (Photo contributor: R. Jason Thurman, MD.)

PALMAR SPACE INFECTION

Clinical Summary

Palmar space infections occur within the deep fascial spaces of the hand. They commonly arise from puncture wounds, flexor tenosynovitis of the digits, or by hematogenous spread. The palm loses its concavity; tenderness, erythema, warmth, and fluctuance are evident. A thenar space infection is characterized by swelling over the thenar eminence and pain with thumb movement. With a midpalmar space infection, motion is limited and painful for the middle and ring fingers. Infection in the web spaces may involve both the palmar and dorsal sides, forming the classic hourglass-shaped (collar-button) abscess. Cellulitis, local traumatic injury, fractures, and soft tissue mass are included in the differential.

Management and Disposition

All deep space infections of the hand should be managed by a hand surgeon. Prompt incision and drainage in the operating room is necessary for the best outcome. Parenteral antibiotics against S aureus (including CA-MRSA) as well as anaerobes should be started urgently.

Pearls

1. Palmar space infections may cause swelling on the dorsal hand due to the dorsal location of hand lymphatics.
2. There may be minimal signs of swelling over the palmar surface.
3. Infections may be associated with compartment syndrome of the hand.



FIGURE 12.26 ■ **Palmar Space Infection.** Diffuse palmar and hand swelling after trauma. (Photo contributor: David Effron, MD.)



FIGURE 12.25 ■ **Thenar Space Infection.** Thenar space infection following injury to the thumb. In this palmar view, erythema and swelling in the right thenar area is evident. (Photo contributor: Richard Zienowicz, MD.)

Clinical Summary

Tenosynovitis, an inflammation of the tendon and surrounding synovial sheath, is characterized by pain and tenderness. Pyogenic flexor tenosynovitis is a serious tendon sheath infection resulting from puncture wounds, local extension, or hematogenous spread; it is characterized by the four cardinal Kanavel signs: finger held in mild flexion; fusiform digit swelling; tenderness along the entire tendon sheath, especially at the palmar surface of the metacarpophalangeal (MCP) joint; and severe pain with passive extension. Tenosynovitis may be complicated by fibrosis and adhesions leading to stiffness, loss of function, and tendon necrosis.

Inflammatory flexor tenosynovitis is typically due to rheumatoid arthritis, overuse, diabetes mellitus, or connective tissue disorders. The time course is typically more indolent than pyogenic flexor tenosynovitis, although the sequelae can be similar.

Intersection syndrome is tenosynovitis of the radial wrist extensors. It causes pain, swelling, and crepitus of these muscle bellies in the distal dorsoradial forearm.

Management and Disposition

It is difficult to distinguish infectious and noninfectious causes early (24-48 hours). Management of noninfectious includes immobilization and NSAIDs. Broad-spectrum parenteral antibiotics and emergent consultation with a hand surgeon for incision and drainage are mandated with pyogenic flexor tenosynovitis.

Pearls

1. S aureus is the most common organism, but Streptococcus as well as gram-negative and anaerobic organisms may occur.
2. The most specific sign of tenosynovitis is pain with passive digit extension.
3. Patients with immunocompromised states or recently administered antibiotics may not exhibit the classic tetrad of Kanavel signs.



FIGURE 12.28 ■ **de Quervain Tenosynovitis.** Swollen and markedly tender area over radial styloid in de Quervain tenosynovitis. (Photo contributor: Robert Tubbs, MD.)



FIGURE 12.27 ■ **Flexor Tenosynovitis.** Pyogenic flexor tenosynovitis of the fourth finger with fusiform swelling, tenderness along the flexor tendon sheath, and pain on flexion. (Photo contributor: Lawrence B. Stack, MD.)

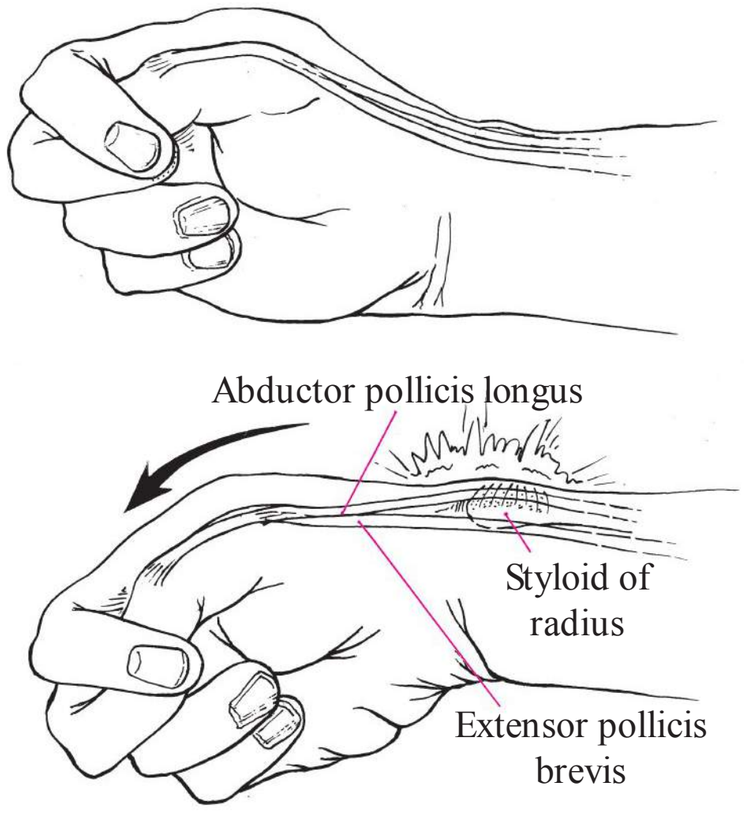


FIGURE 12.29 ■ **Finkelstein Test for de Quervain Tenosynovitis.** Pain over the radial styloid is elicited with ulnar deviation of the wrist as shown.



FIGURE 12.30 ■ **Tenosynovitis.** Pyogenic tenosynovitis of the middle finger with fusiform swelling, tenderness along the tendon sheath, and pain on movement. (Photo contributor: Edmond A. Hooker, MD, DrPH.)



FIGURE 12.31 ■ **Intersection Syndrome.** Discrete swelling at the intersection of muscles that connect to the thumb and underlying wrist tendons. (Photo contributor: Larry Mellick, MD, MS.)

Clinical Summary

Thrombophlebitis is superficial thrombosis and inflammation of veins or varicosities characterized by redness, tenderness, and palpable, indurated, cordlike venous segments. Common associations are IV insertion, irritant IV solutions, trauma, pregnancy, and recent postpartum states. There is little risk of embolism when associated with varicose veins or superficial veins distal to the popliteal fossa. However, pulmonary embolism can occur secondary to thrombus propagation to more proximal veins of the deep venous system, particularly with greater saphenous vein involvement. Lymphangitis, DVT, and cellulitis are in the differential. Septic superficial thrombophlebitis should be considered with a history of IV drug use. The patient should be evaluated for the possibility of DVT if no underlying cause of superficial thrombosis is elucidated.

Management and Disposition

Elevation with warm compresses, rest, and analgesia is sufficient treatment for uncomplicated superficial thrombophlebitis. Superficial thrombophlebitis of the saphenofemoral or iliofemoral system requires treatment as a DVT. Admission is warranted if there is extensive involvement, septic signs, progression of symptoms despite treatment, or severe inflammatory reactions.

Pearls

1. Thrombophlebitis of the greater saphenous vein may be confused with lymphangitis, since the lymphatic drainage from the leg runs along the vein.
2. The superficial femoral vein, despite its name, is considered a deep vein, and thrombosis involving this requires standard DVT treatment.
3. Anticoagulation may be considered for lower extremity thrombophlebitis involving the greater saphenous vein close to the femoral junction.
4. Thrombophlebitis of the upper extremity is unlikely to progress to DVT.
5. Suppurative thrombophlebitis should be suspected with high fevers, signs of extensive erythema or purulent drainage, or IV drug use.



FIGURE 12.33 ■ **Septic Thrombophlebitis.** Thrombophlebitis may be complicated by bacterial infection. Note the purulence associated with the erythematous thrombosed veins. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 12.32 ■ **Thrombophlebitis.** Thrombophlebitis of the superficial leg veins. The thrombosed veins are erythematous, close to the surface, and palpable. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 12.34 ■ **Thrombophlebitis.** Erythema and tenderness in the medial thigh of this patient with saphenous thrombophlebitis. This may be difficult to differentiate from a deep venous thrombosis by physical exam alone. (Photo contributor: Robert Tubbs, MD.)

PARONYCHIA

Clinical Summary

Paronychia is the most common hand infection and is characterized by inflammation and pus accumulation along a lateral nail fold. It may spread to involve the eponychium at the base of the nail and the opposite nail fold if untreated. S aureus is most frequently isolated, although the infection is generally mixed flora. Felon, dactylitis, herpetic whitlow, hydrofluoric acid burn, and traumatic injury should be considered in the differential.

Management and Disposition

If paronychia is recognized early, prior to frank abscess formation, warm soaks with or without oral antibiotics may be sufficient. After 2 to 3 days, there may be sufficient pus accumulation along the eponychial fold to warrant incision and drainage. After digital block, a #11 blade or 18-gauge needle is advanced parallel to the nail and under the eponychium at the site of maximal fluctuance. If pus has collected under the nail (subungual abscess), then a portion must be removed to provide drainage. Incisions should be packed open with gauze (removed in 24-48 hours). Oral antibiotics should be prescribed, and the patient reevaluated in 2 to 3 days.



FIGURE 12.35 ■ **Paronychia.** A paronychia involving one lateral fold and the eponychium. There is swelling, erythema, and tenderness on the dorsum of the distal phalanx. (Photo contributor: Frank Birinyi, MD.)

Pearls

1. Paronychia is associated with nail biting, manicure trauma, and foreign bodies.
2. Germinal matrix damage during nail plate excision may result in nail deformity.
3. It is important to distinguish a paronychia from herpetic whitlow, where incision and drainage is contraindicated.
4. Paronychia may occur in the toes as well as the fingers.



FIGURE 12.36 ■ **Paronychia.** A paronychia involving the medial fold and eponychium. (Photo contributor: Selim Suner, MD, MS.)



FIGURE 12.37 ■ **Paronychia.** Incision and drainage of a paronychia with considerable purulent drainage. (Photo contributor: Selim Suner, MD, MS.)

SUBCLAVIAN VEIN THROMBOSIS

Clinical Summary

Thrombosis of the subclavian vein (Paget-von Schrötter syndrome) is an uncommon condition, typically occurring in young patients following exercise and compression injury to the subclavian or axillary vein from a narrow thoracic outlet (effort thrombosis). Pain, tightness, and arm swelling are manifest within a day. Pitting edema develops in the fingers, hand, and forearm. There is no arterial insufficiency, and the pulses are palpable. This syndrome is separate from iatrogenic upper extremity thrombosis, generally as a result of vascular access catheters. There is a 15% risk of developing pulmonary embolism from thrombosis of upper extremity veins; however, large or fatal emboli are very rare. Ultrasound has become the screening test of choice. Superior vena cava syndrome, upper extremity trauma, heart failure, angioedema, and lymphatic obstruction are in the differential.

Management and Disposition

Treatment consists of hospital admission, elevation, local heat, analgesia, and anticoagulation with heparin for patients presenting with long-term thrombosis. In cases of acute thrombosis (within 5 days of symptom onset), thrombolysis with direct catheter infusion of thrombolytic agents or mechanical clot retrieval may be considered. Surgical thrombectomy has also been employed. Operative correction of anatomic abnormalities should be accomplished to prevent long-term morbidity.

Pearls

1. Swelling of the neck and face may signify thrombosis or compression of the superior vena cava.
2. The superficial veins in the upper extremity are often distended and do not collapse when the arm is elevated.
3. There is a greater incidence of subclavian vein thrombosis in men and in the right arm.
4. Ultrasound may be limited in visualizing nonocclusive mural thrombi, or those located in the proximal subclavian or innominate veins.



FIGURE 12.38 ■ **Subclavian Vein Thrombosis.** Left subclavian vein thrombosis manifested by swelling of the upper extremity. (Photo contributor: Frank Birinyi, MD.)

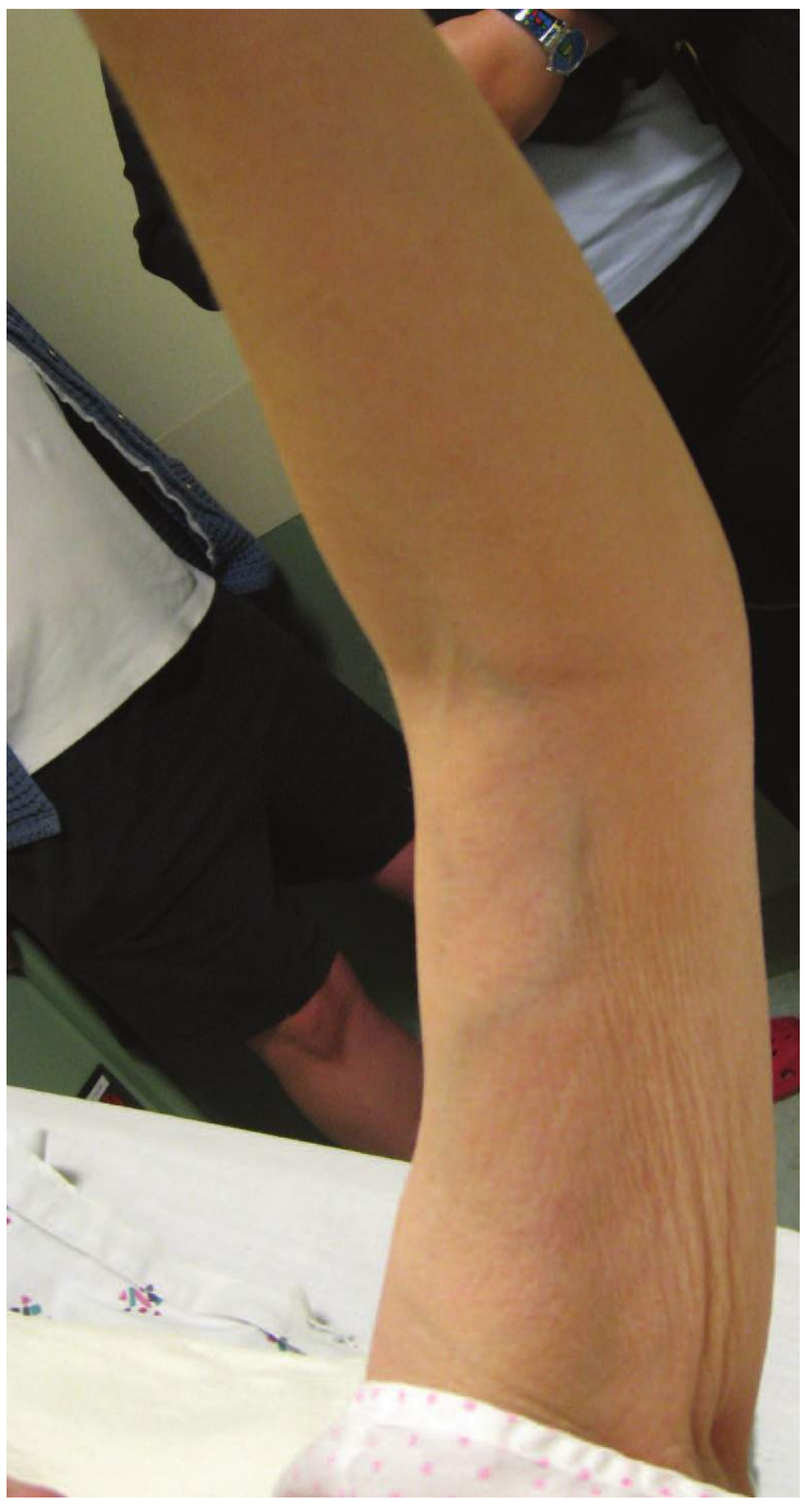


FIGURE 12.39 ■ **Subclavian Vein Thrombosis.** Diffuse arm swelling and dilated superficial veins. Note that the veins remain dilated even when the arm is elevated. (Photo contributor: Robert Tubbs, MD.)

CERVICAL RADICULOPATHY

Clinical Summary

Cervical radiculopathy is caused by compression of a nerve root by a laterally bulging or herniated intervertebral disk, osteoarthritis, or degenerative spondylosis. Pain results from injury to the nerve roots and nerves innervating the dura, ligaments, facet joints, and bone. Common clinical features include pain, paresthesia, and root signs (sensory loss, lower motor neuron muscle weakness, impaired reflexes, and trophic changes). The pain is sharp, stabbing, and worse with cough; it often radiates over the shoulder and down the arm. Frequently, there is numbness and tingling following a dermatomal distribution. Root signs may be found corresponding to anatomic distribution. MRI is the test of choice to distinguish cervical radiculopathy from disc and bone disease. Electromyelography studies may also be helpful in ruling out other disease processes. Trauma, myelopathy, plexopathy, neurofibromatosis, metastatic tumor infiltration of nerve roots, neoplasm, shingles, and central cord syndrome should be considered in the differential.

Management and Disposition

Emergency treatment includes pain control and referral to an orthopedic surgeon or neurosurgeon. Although management often requires narcotic analgesics, appropriate doses of NSAIDs should also be initiated in patients without contraindications. Since prolonged nerve root compression can lead to permanent deficits, immediate referral is necessary for progressive neurologic signs. Patients with intractable pain, progressive weakness, and myelopathy should be admitted.

Pearls

1. Most radiculopathies resulting from cervical disk disease are seen in the age group 30 to 60 years and in the C5 to C7 region.
2. Patients with acute cervical radiculopathy may present with their upper extremity supported by their head to counteract the cervical root distraction caused by the weight of their dependent extremity.
3. CT myelography may be the next most appropriate study in patients with a contraindication to MRI.

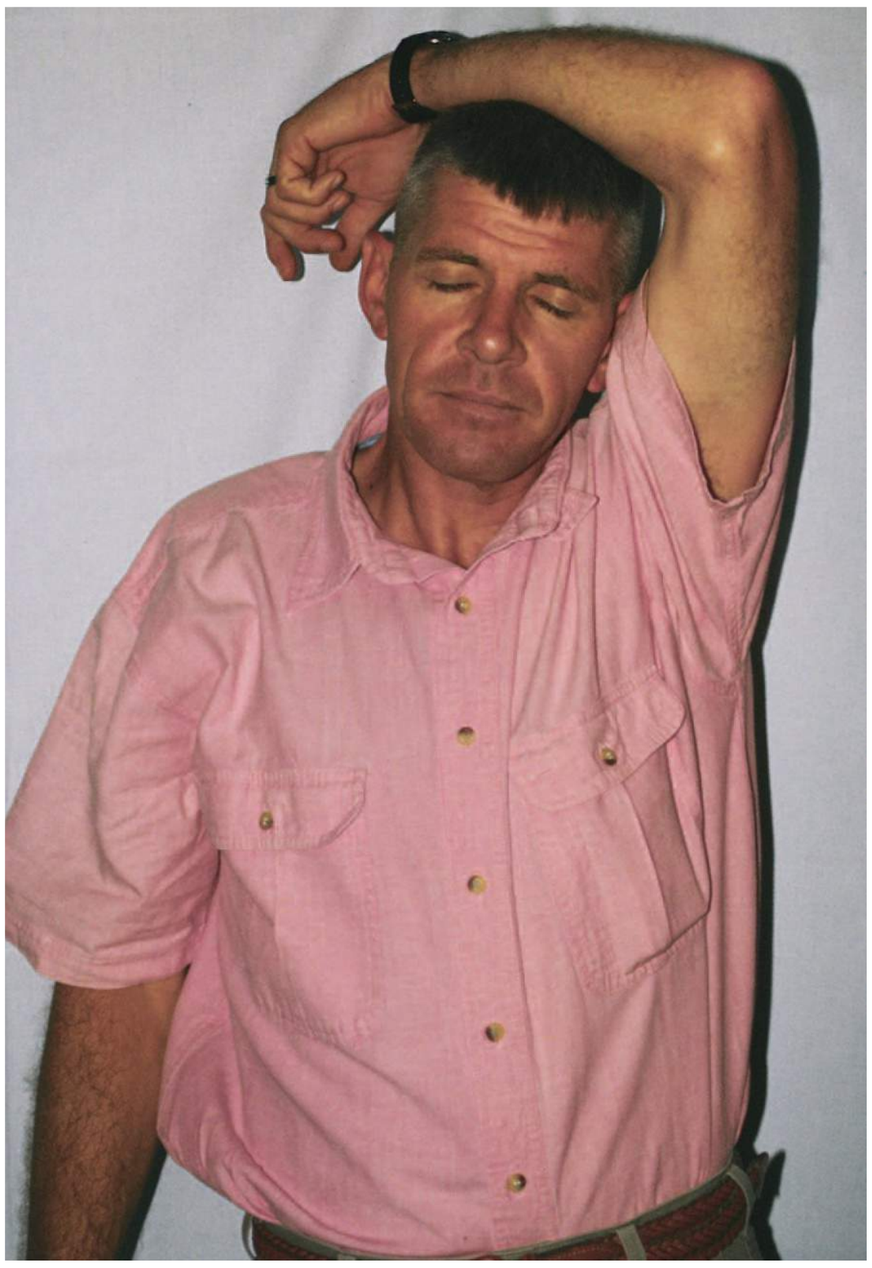


FIGURE 12.40 ■ **Cervical Radiculopathy.** This is the classic position of relief for cervical radicular pain. This patient presented with severe neck pain with radiation to the extremity. The only way the patient was able to get relief was by holding his arm over his head in the position shown. This patient has a C5-C6 herniated nucleus pulposus. (Photo contributor: Kevin J. Knoop, MD, MS.)

Clinical Summary

Digital clubbing is characterized by bulbous fusiform enlargement of the distal portion of a digit with loss of the angle between the proximal nail fold and the nail plate (Lovibond angle). The mechanism underlying clubbing is not known. It may be hereditary, idiopathic, or acquired, and is associated with multiple medical conditions including carcinoma, intrathoracic sepsis, bacterial endocarditis, cyanotic congenital heart disease, esophageal disorders, cirrhosis, inflammatory bowel disease, pulmonary disorders, atrial myxoma, multiple pregnancies, and pachydermoperiostosis. The incidence of clubbing with each of these conditions is variable. It may be reversible in certain disease processes.

Management and Disposition

Treatment of the underlying condition is indicated.

Pearls

1. Patients rarely recognize clubbing in their own fingers even if the condition is marked.
2. Pseudoclubbing is an overcurvature of the nails in both longitudinal and transverse axes, with preservation of the angle between the proximal nail fold and nail plate.



FIGURE 12.41 ■ **Clubbing.** Marked digital clubbing can be seen in this patient. Note the hyperemia in the skin folds around the nail. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 12.42 ■ **Clubbing.** Pronounced digital clubbing. Note the loss of angle between the proximal nail fold and the nail plate (Lovibond angle). (Photo contributor: Robert Tubbs, MD.)

PHLEGMASIA DOLENS

Clinical Summary

Phlegmasia alba dolens (painful white leg, or milk leg) is caused by massive thrombosis of the iliofemoral veins and characterized by pitting edema of the entire lower extremity, inguinal area tenderness, and a pale extremity secondary to arterial occlusion. Phlegmasia cerulea dolens (painful blue leg) arises from massive thrombosis of the lower extremity veins, including the perforators and collaterals, resulting in venous ischemia with a cool, painful, swollen, tense, and cyanotic-appearing lower extremity. Occasionally, there is bullae formation; compartment syndrome and gangrene may follow.



FIGURE 12.43 ■ **Phlegmasia Dolens.** Phlegmasia cerulea dolens of the left lower extremity. Note the bluish discoloration and swelling. (Photo contributor: Daniel L. Savitt, MD.)

The differential includes arterial insufficiency or thrombosis, aortic dissection, abdominal aortic aneurysm, cellulitis, and lymphedema. Doppler ultrasound, impedance plethysmography, and CT venography (most accurate for determining extent) are used in the diagnosis.

Management and Disposition

Systemic anticoagulation with heparin should be initiated immediately; consultation with vascular surgery or interventional radiology should be obtained emergently. There is a significant rate of pulmonary embolization in this condition. In addition, there is a high incidence of postphlebitic syndrome due to venous valvular incompetence. New endovascular techniques for pharmacomechanical clot dissolution are showing promising results for treating these patients.

Pearls

1. Pregnancy is one risk factor for phlegmasia alba dolens.
2. About 40% of patients with phlegmasia cerulea dolens have an underlying malignancy.
3. Hypotension may result from venous pooling of blood in the lower extremity and diminished venous return.



FIGURE 12.44 ■ **Phlegmasia Dolens.** Discoloration and edema secondary to phlegmasia cerulea dolens. (Photo contributor: Selim Suner, MD, MS.)

Clinical Summary

Deep venous thrombosis (DVT) is often associated with intrinsic coagulopathy, impaired fibrinolysis, recent surgery, or trauma. Other associated conditions include immobilization, increased estrogen (pregnancy, oral contraceptives) with smoking, malignancy, prior DVT, inflammatory disease processes, or coronary artery disease. Intravenous catheters are also a major cause of DVT, particularly in the upper extremity. Unilateral swelling and tenderness, classically in the calf and thigh, are characteristic. Doppler ultrasonography is the screening test of choice in most institutions. In selected low-risk patients, a quantitative d-dimer study may be used to rule out DVT. Cellulitis, lymphedema, heart failure, compartment syndrome, myositis, arthritis, and superficial phlebitis should also be considered in the differential.



FIGURE 12.46 ■ **Deep Venous Thrombosis.** Erythema and swelling in the left lower extremity. (Photo contributor: Selim Suner, MD, MS.)



FIGURE 12.45 ■ **Deep Venous Thrombosis.** This patient has some classic findings of left lower extremity DVT: swelling, erythema, pain, and tenderness. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 12.47 ■ **Ruptured Baker Cyst**. Comparison of this patient's ankles reveals circumferential swelling around the right side. MRI revealed a ruptured Baker cyst in the right popliteal fossa. Such a presentation may mimic acute lower extremity DVT. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Classic treatment is heparin anticoagulation and admission for warfarin loading. However, low-molecular weight heparin (LMWH) has been shown to be more efficacious for initial treatment. It has also allowed outpatient management in selected patients. In cases of large DVT, consultation with interventional radiology or vascular surgery may be indicated for endovascular thrombectomy. Although DVT in the calf and superficial veins of the lower extremity do not typically embolize, these thrombi can propagate into the deep venous system and may eventually lead to emboli. Serial diagnostic studies are performed to follow the course.

Pearls

1. A Baker cyst, herniation of the synovial membrane through the posterior knee capsule, may rupture, causing unilateral calf swelling similar to DVT.
2. Patients with unexplained DVT should be screened for occult malignancy.
3. Homan sign (calf pain during passive dorsiflexion) is unreliable in the diagnosis.
4. Patients with an unclear diagnosis of cellulitis should have an objective study to rule out DVT.
5. The superficial femoral vein is, despite the name, a part of the deep venous system; thrombosis there requires systemic anticoagulation.

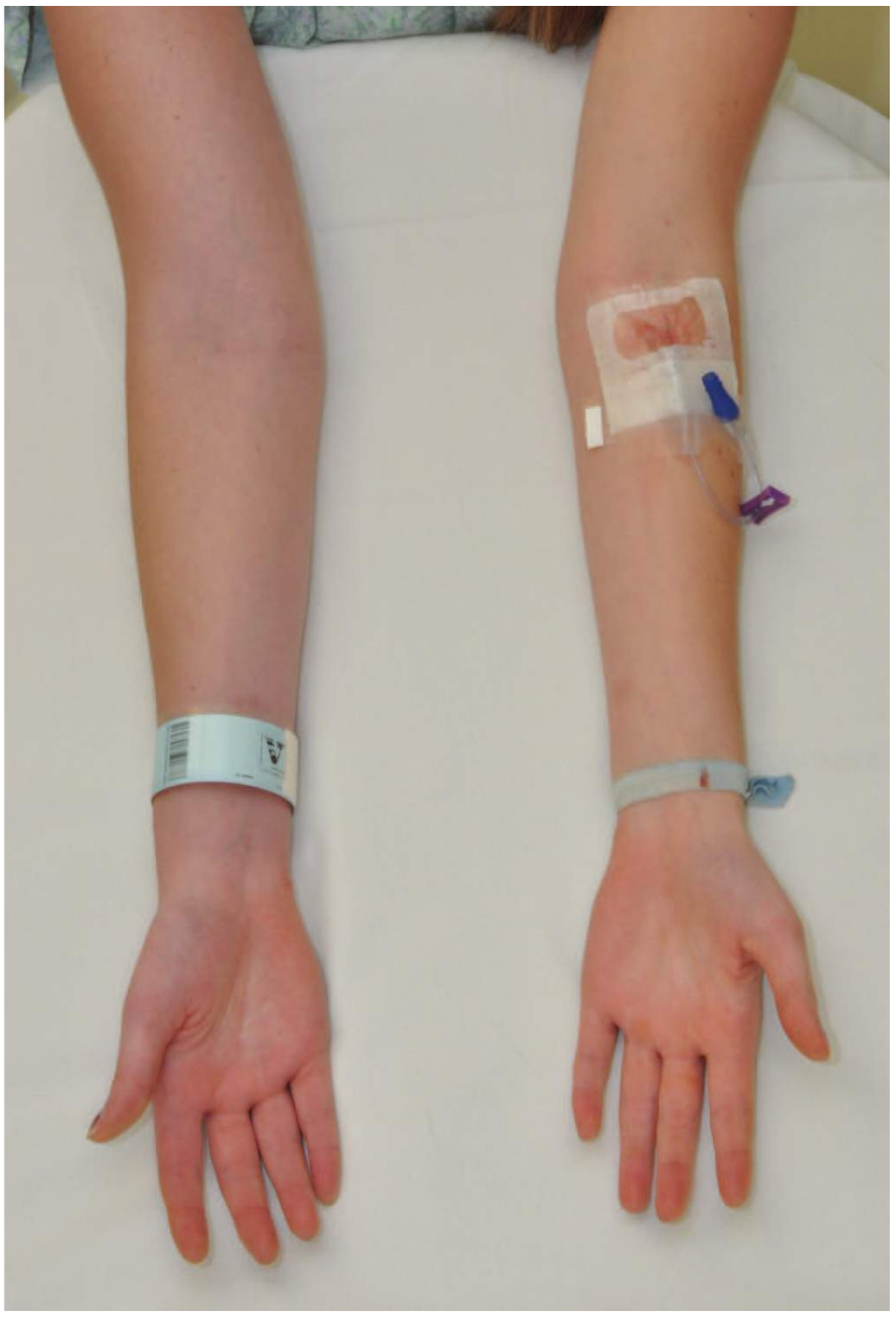


FIGURE 12.48 ■ **Deep Venous Thrombosis.** Swelling and erythema consistent with right upper extremity DVT. (Photo contributor: Hanyuan Shi.)

Clinical Summary

Dupuytren contracture results from shortening and fibrotic changes in the subcutaneous palm tissue and longitudinal bands of the palmar aponeurosis. It may begin as a nodule and then progress to contracture of a finger or fingers. Usually, this is noted at the MCP joint, but the proximal interphalangeal (PIP) or distal interphalangeal (DIP) joint may be involved.

Management and Disposition

The only effective treatment is surgery. Patient education and referral to a hand surgeon is recommended. Recurrence and development of a contracture in other areas may occur.



FIGURE 12.49 ■ **Dupuytren Contracture.** This chronic problem is seen at the most common site: the ring finger. (Photo contributor: Alan B. Storrow, MD.)

Pearls

1. The flexor tendons are not involved.
2. The ring and small fingers are the most commonly involved.
3. Alcoholic liver disease is associated with an increased risk of development.



FIGURE 12.50 ■ **Dupuytren Contracture.** Note the contracture at the fifth metacarpophalangeal joint. (Photo contributor: Vineet Mehan, MD.)

ACHILLES TENDONITIS

Clinical Summary

Achilles tendonitis refers to a spectrum of disease ranging from nonpainful nodules to painful swelling of the tendon and paratendon sheath. It most typically develops from overuse, usually after sudden changes in activity or training level. It often occurs in older recreational athletes, who are generally more sedentary and deconditioned. Multiple factors generally contribute to the condition, including inappropriate footwear, training on poor surfaces, or prolonged running or jumping. High-risk factors include anatomic abnormalities, such as cavus feet, tibia vara, and heel or forefoot varus deformities. A tight Achilles tendon may develop in women who frequently wear high-heeled shoes or in patients who wear boots with high heels.

The most common area of pain is typically the area 2 to 6 cm proximal to the insertion site. This is due to a relative paucity of blood vessels in that region, making it more susceptible to inflammation and degeneration from repetitive microtrauma. Pain and tenderness increase with dorsiflexion of the foot. In some cases, a tendon friction rub may be palpable.



FIGURE 12.51 ■ **Achilles Tendonitis.** Note the significant erythema and thickening of the Achilles tendon on the patient's left compared to the unaffected right side. (Photo contributor: Kevin J. Knoop, MD, MS.)

Management and Disposition

Careful examination should be performed to distinguish between Achilles tendonitis and tendon rupture. The Thompson test and palpation of the tendon for gaps or discontinuity should be performed. Any patients with suspicion of partial or complete tendon rupture should be splinted and urgently referred to orthopedic surgery. In cases of tendonitis, gentle progressive stretching and lengthening exercises are helpful. In athletes, a reduction in activity is recommended. Use of gel heel inserts may be helpful in the short term, by cushioning and raising the heel, thereby decreasing tendon excursion. Use of ice and NSAIDs are useful in reducing pain and inflammation. Referral to an orthopedic surgeon for physical therapy is generally indicated in refractory cases.

Pearls

1. Fluoroquinolone use has been reported to increase the risk of Achilles tendonitis and possible tendon rupture. Patients presenting with pain, who are currently taking a fluoroquinolone, should have alternative antibiotic therapy considered.
2. Corticosteroid injection for Achilles tendonitis is very controversial, and should not be performed in the emergency department.
3. Bilateral tendon involvement, especially at the insertion site, suggests a systemic inflammatory condition such as ankylosing spondylitis, reactive arthritis (Reiter syndrome), or psoriatic arthritis.

GANGLION (SYNOVIAL) CYST

Clinical Summary

Ganglion (synovial) cysts are a cystic swelling overlying a joint or tendon sheath. They are the most common soft-tissue tumors of the hand and wrist, although they can arise over any joint. The etiology is currently debated; the most commonly accepted theory is the cyst forms secondary to mucoid degeneration of collagen and connective tissues. Other theories postulate a traumatic origin. It is unclear whether repetitive motion leads to causation, although it does appear to provoke symptoms and possibly lead to cyst enlargement. They may occur in any age, although the majority arise between the second and fourth decades of life.

Ganglion cysts are composed of collagen fiber walls with clear, highly viscous mucin content. They may be unilocular or multilocular. The most common presentations include swelling over or in close proximity to a joint, as well as pain, limitation of motion, weakness, and paresthesias. They are rarely greater than 2 cm in diameter. The most common location is dorsally over the scapholunate ligament of the wrist (60-70%), with the volar wrist the next most common site (20%).

Management and Disposition

Ganglion cysts may spontaneously regress; therefore, treatment is generally reserved for symptomatic lesions. Treatments include both nonsurgical and surgical options. The most common nonsurgical option is cyst aspiration, generally using a 16-gauge needle, followed by steroid injection. For recurrent lesions, referral to a hand surgeon for excision is warranted.

Pearls

1. Transillumination may aid in differentiating a ganglion cyst from a solid tumor; ganglia transilluminate, while solid lesions do not.
2. Patients with chronic dorsal wrist pain of unknown etiology should be screened for occult ganglion cysts.
3. MRI or ultrasound may be useful in detecting occult ganglion cysts.



FIGURE 12.52 ■ **Ganglion Cyst.** This large ganglion cyst is located over the scapholunate ligament, the most common location for this entity. (Photo contributor: Lawrence B. Stack, MD.)

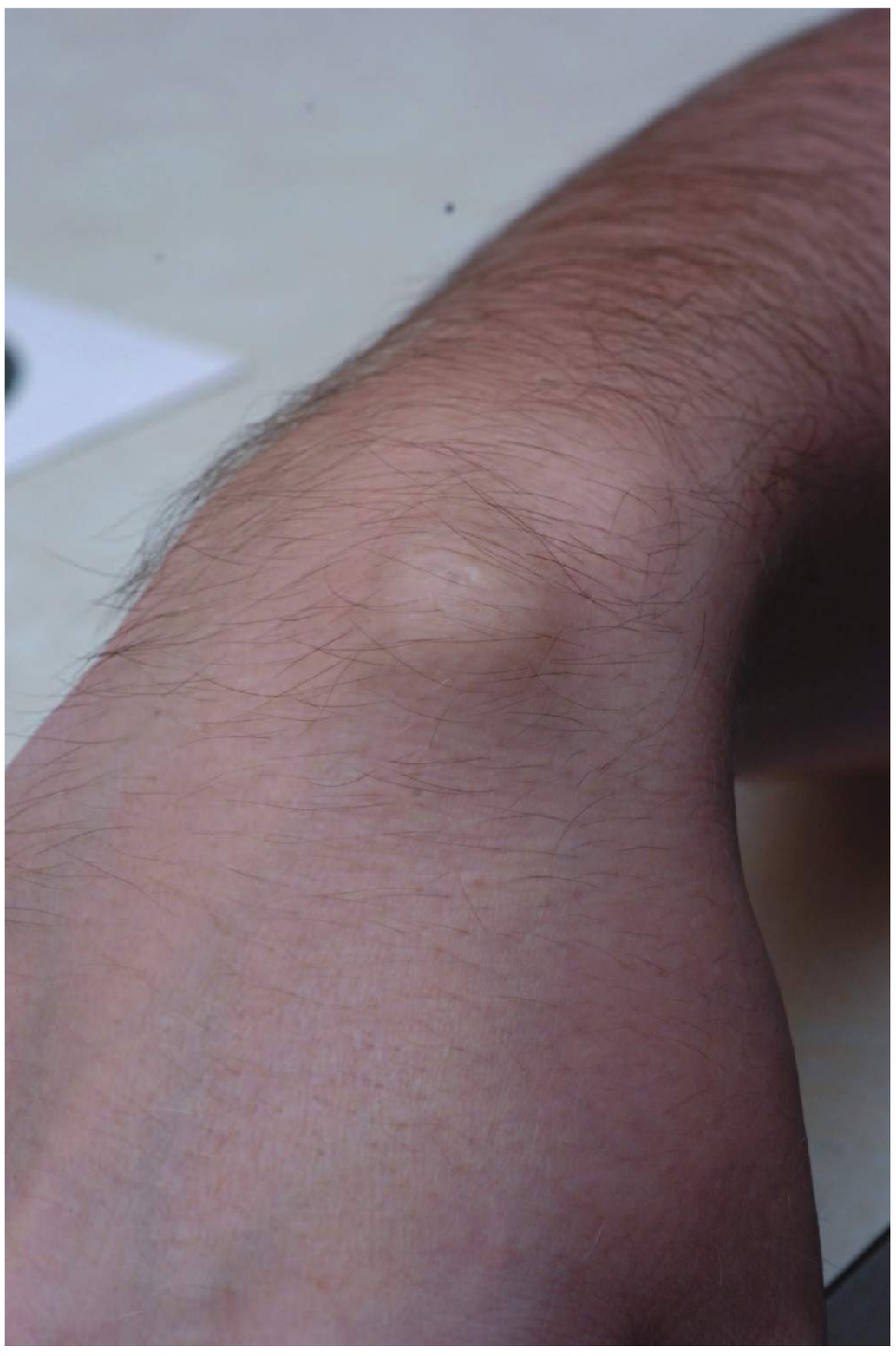


FIGURE 12.53 ■ **Ganglion Cyst.** Classic example of a ganglion cyst located over the scapholunate ligament of the wrist. (Photo contributor: Lawrence B. Stack, MD.)

RAYNAUD DISEASE

Clinical Summary

Raynaud disease refers to reversible ischemia of peripheral arterioles, usually in response to cold exposure or emotional stress. In general, it is more prevalent in women and most often affects the fingers, although the toes, face, ears, nose, and nipples may be involved.

A typical episode usually starts suddenly with the onset of cold digits associated with sharply demarcated, blue or white, color changes. With rewarming, erythema develops due to a reactive hyperemia. The vasospasm may last for several hours, but usually resolves with removal of the initial stimulus.

In general, it is called Raynaud disease, or primary Raynaud phenomenon, if symptoms occur without evidence of any other associated disease process. In contrast, secondary Raynaud phenomenon occurs in association with a related disease process, such as systemic lupus erythematosus or scleroderma.

Management and Disposition

Treatment involves removal of the inciting stimulus. As many cases are induced by going from a warm to a cold environment, active rewarming with warm water soaks or placing in the axilla are generally effective. Sympathetic stimulation may also trigger an episode, so calming patients who are anxious, or removing them from stressful situations, may be efficacious.

A thorough history and physical examination should be performed with careful attention paid to signs and symptoms of connective tissue disorders. Acrocyanosis (Crocq disease) is a circulatory disorder in which the hands, and less commonly the feet, are persistently cold and blue; some forms are related to Raynaud phenomenon. Counseling patients about methods for reducing the frequency and duration of attacks is helpful, including avoiding sudden cold exposure, minimizing stress, keeping the digits warm, avoiding cigarette smoking, and avoiding sympathomimetic drugs. Referral for primary care follow-up is recommended for all patients.

Pearls

1. A history of cool skin and sharply demarcated color changes is essential for diagnosis.
2. Routine ordering of blood tests, such as erythrocyte sedimentation rate (ESR) or antinuclear antibody (ANA), is not recommended unless other symptoms of connective tissue disorders are present. In these cases, close primary care or rheumatology follow-up is recommended.



FIGURE 12.55 ■ **Raynaud Disease.** A patient with sharply demarcated color changes characteristic of an acute attack of Raynaud phenomenon. (Photo contributor: Katherine Farmer, MD.)



FIGURE 12.54 ■ **Raynaud Disease.** A patient with sharply demarcated color changes consistent with an acute attack of Raynaud phenomenon due to cold exposure. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 12.56 ■ **Acrocyanosis.** Persistently blue and cold fingers. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

An embolus refers to a foreign body in a blood vessel, carried by the blood to a site distant from its origin. Most emboli result from a detached piece of thrombus, often originating from left ventricular thrombus following myocardial infarction, or from atrial fibrillation. Other sources include atheroemboli from ruptured plaque, tumor, or foreign bodies such as venous or arterial catheters or guidewires.

Acute arterial embolization usually results in distal tissue infarction. Most embolic occlusion occurs at the branch points of arteries, due to the generally abrupt change in diameter at these bifurcation points. The most frequent site of arterial embolism is the bifurcation of the common femoral artery, accounting for 35% to 50%. Patients generally present with some or all of the "six Ps": pain, pallor, pulselessness, paresthesias, poikilothermia, and paralysis.

The general predictors of degree of ischemic insult include the amount of collateral circulation, as well as the size of the involved vessel and obstructing embolus. Generally, patients with longstanding peripheral vascular disease have a greater amount of collateral circulation, and are able to tolerate an acute occlusion better than a patient with normal arteries.

Management and Disposition

Acute arterial embolus is a surgical emergency. Immediate initiation of IV heparin is indicated to prevent further clot propagation. Prompt consultation with a vascular surgeon is imperative, as the rate of limb salvage drastically decreases after 4 to 6 hours.

In clear-cut cases of acute arterial embolism, the treatment is generally Fogarty catheter embolectomy without prior angiography. In these cases, a preoperative angiography only prolongs the limb's ischemic time and decreases the chance of salvage. Ambiguous cases, where it is difficult to distinguish between acute embolic occlusion and in situ thrombosis, may benefit from preoperative angiography. Emergent surgical intervention may aggravate thrombosis in the case of in situ thrombus formation.

Pearls

1. Acute arterial embolus generally presents with sudden onset of severe pain. In contrast, in situ thrombosis tends to have a more subacute presentation.
2. Aortic dissection may mimic acute arterial embolus. Involvement of multiple sites suggests dissection.
3. Intra-arterial thrombolytic therapy for acute arterial embolus remains controversial.



FIGURE 12.57 ■ **Arterial Embolus.** Note right foot pallor, consistent with an acute arterial embolus, in this case secondary to femoral artery occlusion. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 12.58 ■ **Arterial Embolus.** Pallor in the right hand secondary to arterial embolus, likely from atrial fibrillation. (Photo contributor: Lawrence B. Stack, MD.)

Chapter 13

CUTANEOUS CONDITIONS

J. Matthew Hardin



Kerion. (Photo contributor: Alan B. Storrow, MD.)

The author acknowledges Christopher R. Sartori, Michael B. Brooks, and Sean P. Collins for portions of this chapter written for the first and second editions.

STEVENS-JOHNSON SYNDROME/TOXIC EPIDERMAL NECROLYSIS

Clinical Summary

Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are two ends of a continuum of life-threatening, reactive diseases. Generally, SJS involves epidermal detachment of less than 10% body surface area (BSA) and TEN greater than 30%; TEN and SJS overlap between 10% and 30%. Two or more mucosal sites are usually affected. The overall mortality of SJS is 1% to 5%, while that of TEN approaches 30%.

SJS/TEN begins with a nonspecific prodrome of upper respiratory tract symptoms, fever, fatigue, myalgia, headache, and mucous membrane and skin sensitivities. A rash appears 1 to 3 days later with mucosal lesions first (erythema, erosions, and hemorrhagic crusting) and a simultaneous or lagging, generalized, dusky, erythematous rash. Bullae form, large sheets of epidermis separate from the dermis, and the involved skin is exquisitely tender to palpation. The Nikolsky sign is present when lateral pressure on unblistered skin causes the epidermis to slide off. Progression of involved skin can occur over a single day or slowly evolve over 14 days. In addition to the generalized "skin failure," life-threatening sepsis, respiratory failure, metabolic derangements, and gastrointestinal hemorrhage may occur. These serious complications may be compounded by underlying comorbidities.

With a few exceptions, SJS/TEN results from a drug exposure, generally within 7 to 21 days previous to the prodrome. Sulfonamide antibiotics, aromatic anticonvulsants (phenytoin, phenobarbital, and carbamazepine), penicillins, nonsteroidal anti-inflammatory drugs (NSAIDs), allopurinol, lamotrigine, and antiretrovirals are common causes, although over 200 medications, including over-the-counter (pseudoephedrine) and herbal remedies, have been implicated. Mycoplasma pneumoniae and vaccinations have also been associated with SJS/TEN.

Management and Disposition

Stop the offending medication (sometimes requiring withholding all). Assess airway status and secure admission to a burn intensive care unit. SJS/TEN patients require careful attention to fluid management; burn protocols may overestimate fluid requirements and contribute to pulmonary edema (resuscitation should maintain urine output at ~0.5-1 mL/kg/h). Dress denuded skin with gauze bandage rolls and moisten with normal saline (transition to antibacterial solution in the burn unit). Emergently consult dermatology and ophthalmology. Meticulous supportive care is the foundation of treatment. With dermatology and burn unit consultation, consider intravenous immune globulins (IVIg) within 72 hours.

Pearls

1. The only intervention proven to decrease mortality is to stop the offending medication.
2. If a patient notes continued skin pain, even in areas of normal skin, expect additional sloughing.
3. High-risk groups to develop TEN include cancer/hematologic malignancies, HIV/AIDS, immunosuppression, slow acetylator genotypes, anticonvulsant use associated with radiotherapy, and specific human leukocyte antigen alleles (HLA-B*1502 associated with carbamazepine and HLA-B*5801 associated with allopurinol).
4. Infections and pulmonary edema/acute respiratory distress syndrome (ARDS) are the leading causes of death in TEN patients.



FIGURE 13.1 ■ **Stevens-Johnson Syndrome.** Moderate hemorrhagic crusting on the lips and target-like macules on the palms and fingers—similar to erythema multiforme. (Photo contributor: Alan B. Storrow, MD.)

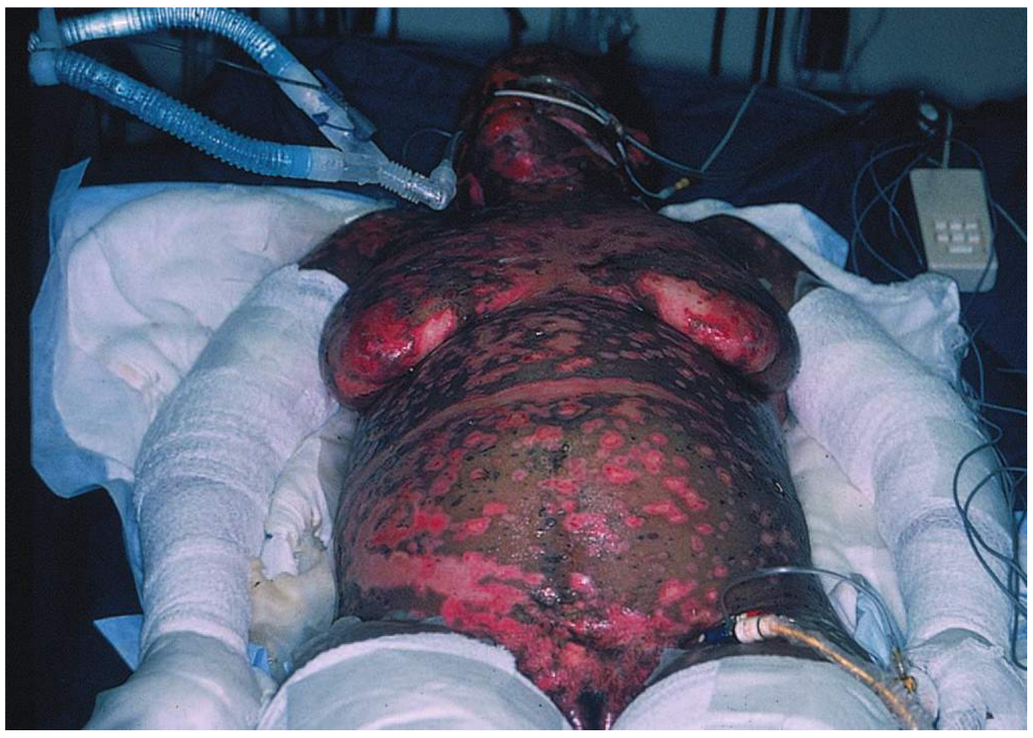


FIGURE 13.2 ■ **Toxic Epidermal Necrolysis.** Widespread desquamation. (Photo contributor: James J. Nordlund, MD.)



FIGURE 13.3 ■ **Toxic Epidermal Necrolysis.** Extensive epidermal desquamation. In darker skinned patients, the initial macules/patches may be hyperpigmented and erythema difficult to appreciate. (Photo contributor: Keith Batts, MD.)



FIGURE 13.4 ■ **Bullous SJS/TEN.** A bullous form of TEN. (Photo contributor: J. Matthew Hardin, MD.)

ERYTHEMA MULTIFORME

Clinical Summary

Erythema multiforme (EM) begins with symmetric, erythematous, sharply defined extremity or trunk macules, and evolves into a "targetoid" or "bull's eye" morphology (a flat, dusky, central area with two concentric, erythematous rings). Bullae may appear in the central dusky area (bullous EM). The mucous membranes, typically oral, may become involved and raise concern for SJS. The typical targetoid lesions allow a diagnosis to be made clinically (bullae, purpura, and mucosal involvement should prompt a dermatology consultation). The rash usually persists for 1 to 4 weeks.

Herpes simplex virus (HSV; frequently labialis) is strongly associated but may not be clinically apparent. Other viruses, bacteria (M pneumoniae, Chlamydia, Salmonella, Mycobacterium), and fungi (Histoplasma capsulatum, dermatophytes) are also associated. Medications account for <10%; NSAIDs, sulfonamides, antiepileptics, allopurinol, and antibiotics are responsible for the majority. Physical factors such as trauma, ultraviolet light exposure, and cold have been reported to elicit EM.

Management and Disposition

Prevention of HSV recurrences is essential. Antivirals administered after lesions present have minimal clinical impact, but patients should be referred for future prophylaxis consideration. Use of facial sunscreens and lip balms may help prevent UVB-induced recurrences. Systemic steroids are discouraged but can be considered in atypical presentations. With the distinctive clinical findings and no systemic symptoms, patients may be discharged home. Systemic symptoms and atypical presentations require admission and dermatologic consultation.

Pearls

1. EM does not progress to TEN.
2. Reassure patients that the lesions will completely resolve without scarring. Hypopigmentation and hyperpigmentation may occur but should resolve over months.
3. Eye involvement requires a slit-lamp examination and ophthalmologic consultation to exclude active HSV infection.
4. Patients with immunosuppression are more prone to recurrent and prolonged episodes.
5. The dusky centers help differentiate EM from typical exanthematous drug reactions (no dusky centers) and urticaria (normal central zone skin).



FIGURE 13.5 ■ **Erythema Multiforme.** Wrist, hand, and fingers with typical central dusky centers surrounded by the concentric "bull's eye" rings. (Reproduced with permission from Weinberg S, Prose NS, Kristal L. Color Atlas of Pediatric Dermatology. 4th ed. New York, NY: McGraw-Hill; 2008: Fig. 13-9.)



FIGURE 13.6 ■ **Erythema Multiforme.** Symmetric distribution of targetoid macules and plaques. The dusky central zone is more obvious on the left waistline lesions. (Photo contributor: Michael Redman, PA-C.)



FIGURE 13.7 ■ **Bullous Erythema Multiforme.** Atypical targetoid appearance (only two rings apparent) and bulla formation. (Photo contributor: J. Matthew Hardin, MD.)

DRUG ERUPTIONS

Clinical Summary

Exanthematous drug eruptions present 7 to 14 days after a new medication but may appear sooner with rechallenge. A symmetric, erythematous, morbilliform, blanching, eruption is most frequently encountered. Typically, pruritus and low-grade fever are present. The macules and papules usually become confluent and may progress to an exfoliative dermatitis (rarely to erythroderma). The eruption peaks in a few days and, if the culprit medication is stopped, completely resolves over 7 to 14 days.

Acute generalized exanthematous pustulosis (AGEP), a type of drug eruption, presents 1 to 5 days after starting a new medication (typically, a β-lactam or a macrolide antibiotic). A high fever is usually noted with neutrophilia (90% of patients) and eosinophilia (30%). The rash begins on the face and intertriginous areas with edematous erythema studded with nonfollicular, <5 mm pustules. Within hours, it becomes generalized with larger flaccid, flat pustules. Over the next 2 to 4 days, widespread superficial desquamation occurs. Mucosal sites, especially oral mucosa, can be involved. With the cessation of the offending medication, the rash slowly resolves over 2 weeks.

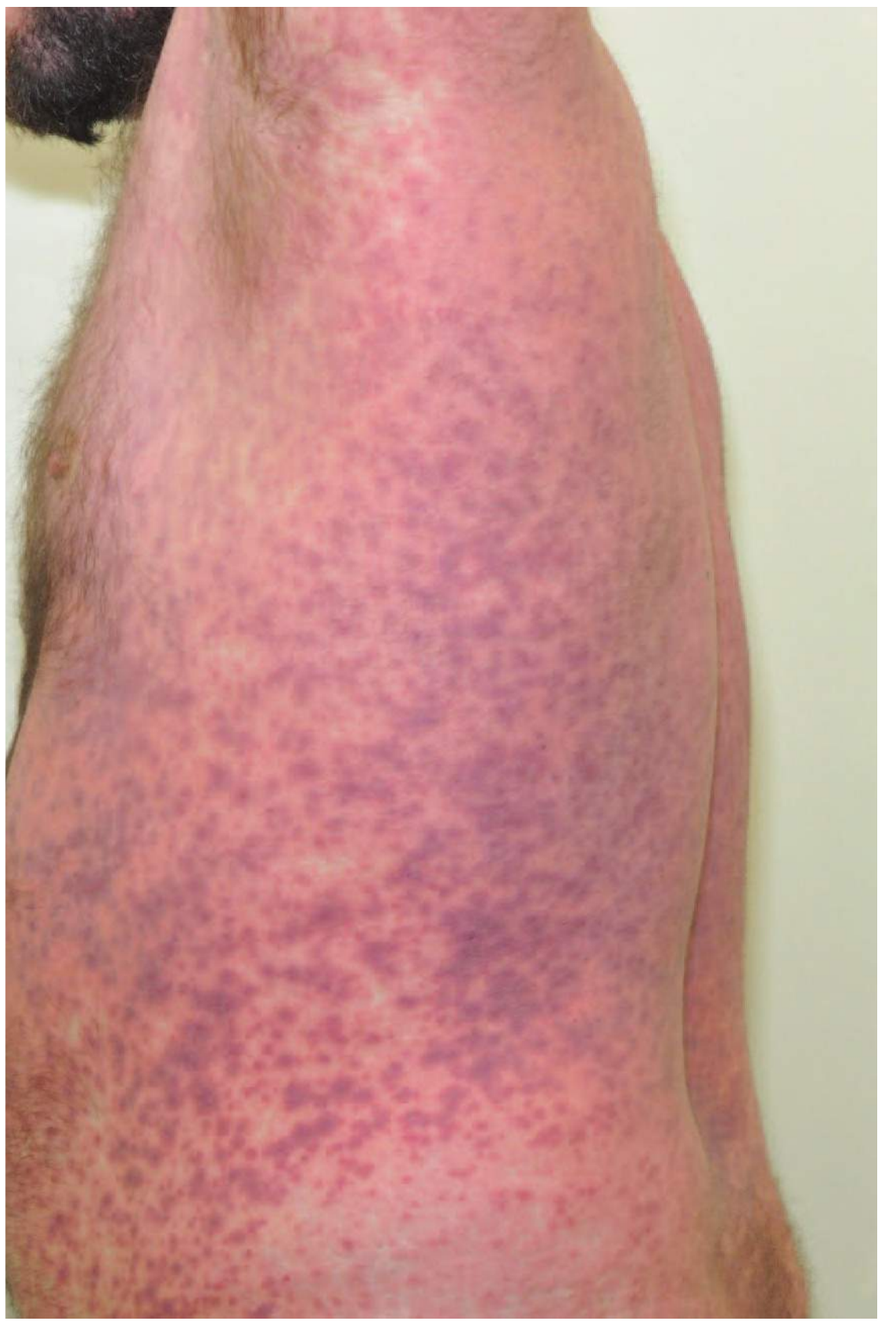


FIGURE 13.8 ■ **Exanthematous Drug Eruption.** Coalescing macules and papules—typically, this is a symmetric exanthem. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

While exanthematous drug eruptions may resolve despite the medication's continued use, cessation of the causative agent is paramount. Symptomatic management includes antihistamines and topical corticosteroids.

The appearance of AGEP, with pustules, fever, and neutrophilia, is difficult to distinguish from an infectious etiology. Wound and blood cultures should be obtained early. Consult dermatology to help differentiate from pustular psoriasis, cellulitis, EM, bullous diseases, SJS, and TEN. Treatment consists of supportive care. The large surface area of desquamation makes secondary infection a major concern.



FIGURE 13.9 ■ **Acute Generalized Exanthematous Pustulosis.** Note the large, flaccid pustule (sterile) and surrounding smaller pustules. The smaller pustules are typical of the initial AGEP presentation. These will eventually slough off and leave a superficial, erythematous erosion. (Photo contributor: J. Matthew Hardin, MD.)

Pearls

1. Exanthematous drug eruptions are usually symmetric and pruritic as opposed to viral eruptions, which are usually asymmetric and asymptomatic.
2. Mononucleosis patients taking amoxicillin or AIDS patients taking sulfa drugs frequently experience this reaction (augmented by viral infections).
3. The desquamation seen in AGEP is much more superficial than the full-thickness desquamation seen in SJS or TEN.

Clinical Summary

Fixed drug eruptions (FDEs) appear 7 to 14 days after first exposure. The lesions can appear anywhere, including mucous membranes, but are most common on the face, lips, hands, feet, and genitalia. Single or multiple annular, edematous, well-demarcated plaques are typical. A central vesicle, bulla, or erosion may occur. After stopping the offending medication, the lesion(s) fade over several days. Residual hyperpigmentation is common. Within 24 hours of reexposure to the culprit medication, the exact rash reappears. The most common offending medications are sulfonamides, NSAIDs, barbiturates, tetracyclines, and carbamazepine.

Management and Disposition

Identify all potential medications (prescription, herbal, and over-the-counter) and stop the offending drug. Symptomatic treatment with antihistamines and analgesics is sufficient. Refer to dermatology for further evaluation.

Pearls

1. Without a thorough history including medication use, FDEs are difficult to identify.
2. Pseudoephedrine, a common over-the-counter medication, is a frequent cause of FDEs.
3. Warn patients that hyperpigmentation is expected and may not completely resolve.



FIGURE 13.10 ■ **Fixed Drug Eruption.** This red to violaceous, pruritic, sharply demarcated patch is a cutaneous reaction to a drug. Repeated exposure will cause a similar reaction in the same location. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)



FIGURE 13.11 ■ **Fixed Drug Eruption.** Recurring reaction to acetaminophen. (Photo contributor: J. Matthew Hardin, MD.)

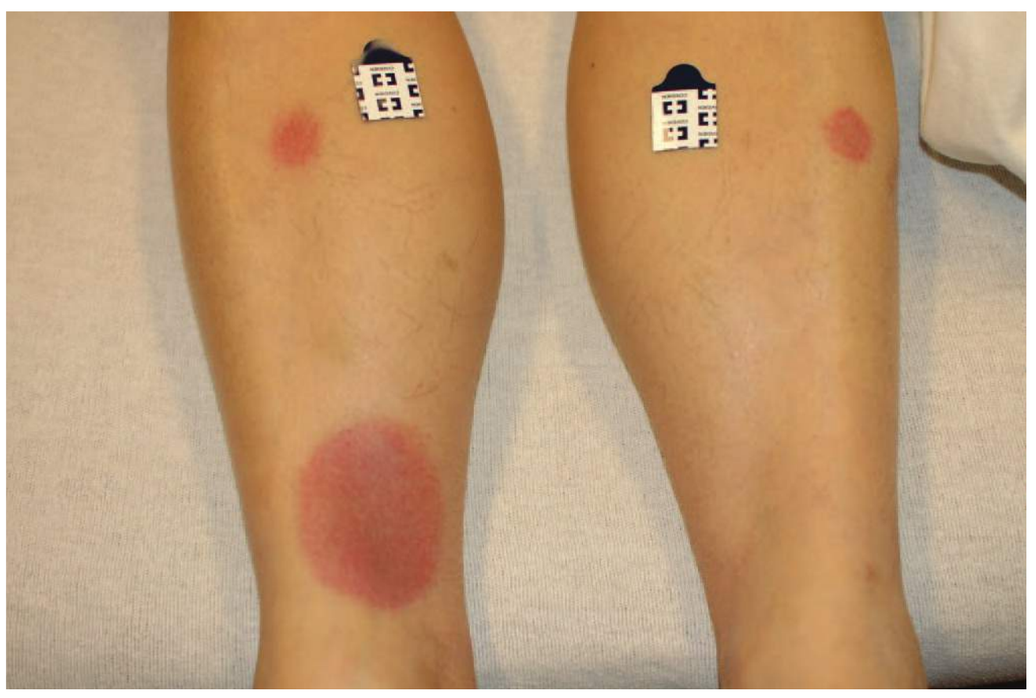


FIGURE 13.12 ■ **Fixed Drug Eruption.** Recurring reaction to sulfamethoxazole and trimethoprim. (Photo contributor: Edmond A. Hooker, MD, DrPH.)

DISSECTING CELLULITIS OF THE SCALP

Clinical Summary

Dissecting cellulitis of the scalp occurs predominately in young black males. This condition, along with acne conglobata, hidradenitis suppurativa (HS), and pilonidal cysts, is the "follicular occlusion tetrad." Multiple, fluctuant abscesses on the scalp vertex and occiput are present. Sinus tracts form between the abscesses and purulent; foul-smelling discharge may be present. Over time, extensive scarring alopecia can result.

Management and Disposition

Exclusion of a secondary infection is paramount. Incision and drainage of new, rapidly forming abscesses may be helpful; obtain cultures if this is attempted. Antibiotics help prevent further abscesses; the tetracyclines help decrease the inflammatory component. Since this is an inflammatory condition, antibiotics alone are not adequate. Refer the patient to a dermatologist for biopsy and further treatment.

Pearls

1. Consider this diagnosis when presented with recurrent scalp abscesses and draining sinus tracts unresponsive to antibiotics.
2. Early diagnosis and referral can prevent further scarring and alopecia.
3. Most patients are young males; the psychological impact can be significant.

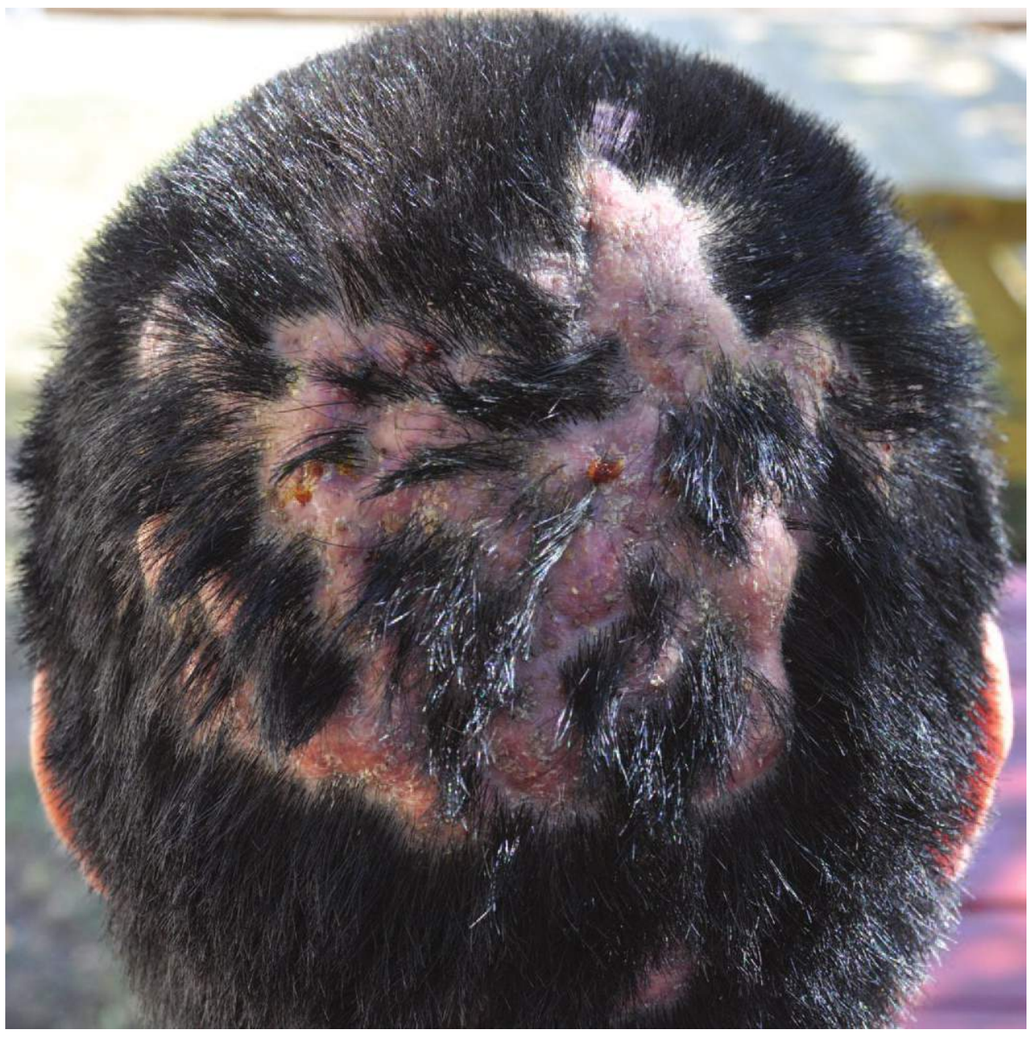


FIGURE 13.13 ■ **Dissecting Cellulitis of the Scalp.** Many active sinus tracts and alopecia in a young Hispanic man. (Photo contributor: Richard P. Usatine, MD. Used with permission. From Usatine RP, Smith MA, Mayeaux EJ, Chumley HS. The Color Atlas of Family Medicine. 2nd ed. McGraw-Hill; 2013: Fig. 189-7.)



FIGURE 13.14 ■ **Dissecting Cellulitis of the Scalp.** Inactive but with disfiguring scars on the entire scalp. Early referral to a dermatologist may prevent this stage. (Photo contributor: J. Matthew Hardin, MD.)

Clinical Summary

HS most commonly affects obese, postpubertal females. This unremitting disease involves hair-bearing intertriginous sites such as the axillae, inguinal folds, gluteal fold, perianal area, and inframammary folds. Individual lesions begin with erythematous nodules that become tender and fluctuant. The sterile abscess ruptures with a suppurative discharge and eventual sinus tract formation or fistulae. This is a primary inflammatory response to the follicle and is not infectious; however, secondary infections are common. Severe hypertrophic scar formation may be dramatic and disfiguring. In addition, chronic discharge is difficult to control and may become malodorous. The differential diagnosis includes bacterial furunculosis, granuloma inguinale, mycetoma, tuberculosis, and fistulas associated with inflammatory bowel disease.

Management and Disposition

HS should be identified and local or systemic infection ruled out. Topical and systemic antibiotics help improve lesions, especially if a secondary infection is suspected. Oral doxycycline or minocycline, with topical clindamycin, is common first-line agents. If possible, incision and drainage should be avoided as this can induce chronic sinus tract formation and worse scarring. Referral to a dermatologist for chronic management and alternative treatments is indicated.

Pearls

1. HS is not a primary infectious process but rather inflammatory. This often leads to delayed diagnosis, worse skin involvement, and permanent scarring.
2. One hallmark of hidradenitis is the "double comedones," a blackhead with two or greater surface openings with superficial communication.
3. Perianal involvement should prompt gastroenterology referral to rule out inflammatory bowel disease.



FIGURE 13.15 ■ **Hidradenitis Suppurativa.** Typical lesions present in the axilla. Note longitudinal scarring and multiple ostia. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.16 ■ **Hidiradenitis Suppurativa.** Chronic lesions in the inframammary folds. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.17 ■ **Hidradenitis Suppurativa.** Chronic lesions in the inguinal folds. (Photo contributor: J. Matthew Hardin, MD.)

PYOGENIC GRANULOMA

Clinical Summary

Pyogenic granuloma presents as an eruptive, friable papule over weeks. They are frequently located on the extremities, face, or at trauma sites. Children are most commonly affected but they can occur at any age. The lesion will bleed with very little trauma. If the papule is not completely removed, it will likely recur at the same site. Pregnant women have a higher incidence of pyogenic granulomas (common on the gingiva).

Management and Disposition

Most emergency department (ED) presentations of pyogenic granuloma will be due to prolonged, often brisk, bleeding. Silver nitrate applied to the papule base is usually effective (avoid on the face to prevent permanent staining). Refer patients to a dermatologist for possible biopsy and definitive treatment.

Pearls

1. An association with isotretinoin, indinavir, epidermal growth factor receptor agents, and capecitabine has been described.
2. Approximately one-third of these benign lesions follow some form of minor trauma.
3. Refer patients for biopsy and histology to exclude other vascular tumors.



FIGURE 13.18 ■ **Pyogenic Granuloma.** Note the violaceous color and multilobulated nodule. The hyperpigmented patches on either side are secondary to a bandage. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.20 ■ **Pyogenic Granuloma.** Friable papule with frequent bleeding. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 13.19 ■ **Pyogenic Granuloma.** A moist, violaceous, vascular nodule formed at the site of an injury. Note that the nodule is demarcated by a thin rim of friable epidermis. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.21 ■ **Pyogenic Granuloma.** Note the violaceous color and multilobulated nodule. (Photo contributor: J. Matthew Hardin, MD.)

PYODERMA GANGRENOSUM

Clinical Summary

Pyoderma gangrenosum (PG) is an inflammatory condition of all ages but is most common among 20- to 50-year-old females. Lesions can be located anywhere (most commonly on the lower extremities) and begin as a papulopustule surrounded by erythema. This pustule erodes to form a necrotic ulcer. Similar satellite pustules and ulcers form around the original lesion and eventually coalesce into a large ulcer. The surrounding border is "rolled," due to the convex elevation, and has a violaceous hue. The ulcers are exquisitely tender to movement and palpation. On the extremities, the ulcers can rapidly involve muscles and tendons. Ostomy sites are a common location and make care very difficult.

Half of cases are idiopathic; the other half are associated with inflammatory bowel disease, hematologic diseases (leukemia, myelodysplasia, monoclonal gammopathy), and the arthritides. Since diagnosis is based on examination, dermatopathology, and exclusion of other causes, it is difficult to confirm and delayed treatment is common. The most concerning diagnosis to exclude is infection due to bacteria, mycobacteria, fungi, syphilis, or amebiasis. Loxosceles spider bites and large vessel vasculitis are also considerations.

Management and Disposition

Appropriate cultures should be obtained. Broad-spectrum antibiotics are indicated for secondary infections. Consult dermatology for biopsy, tissue culture, and initiation of immunosuppressant therapy.

Pearls

1. With the number of ulcers seen in the ED, early diagnosis of PG is difficult and requires a high level of suspicion.
2. PG is often not diagnosed until late-stage ulcer formation and after multiple failed skin grafts. Without immunosuppressant therapy, skin grafting is unlikely to succeed.
3. Half of PG cases have associated diseases; each case needs a thorough specialty evaluation.



FIGURE 13.22 ■ **Pyoderma Gangrenosum.** Rolled and violaceous borders. This lesion can rapidly enlarge and become secondarily infected. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.23 ■ **Pyoderma Gangrenosum.** Early ulcer formation at the site of an ileostomy dressing. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Seborrheic dermatitis represents a spectrum ranging from localized lesions to generalized exfoliative erythroderma. All ages are affected. Lesions have an erythematous base with a yellow, greasy scale. The scalp, external auditory canal, postauricular, eyebrows, eyelids, face (especially the nasolabial folds), axillae, umbilicus, presternal chest, and groin are common locations. Infants can have lesions at the above sites, but focal and confluent lesions are most common on the scalp and called "cradle cap."

Management and Disposition

Seborrheic dermatitis is a lifelong disease and has no cure; management is directed at control. Localized cases are effectively treated with topical 2% ketoconazole. Scalp involvement can be treated with selenium sulfide, ketoconazole, or zinc pyrithione shampoos. Parents of affected infants should be reassured that infantile seborrheic dermatitis is self-limited. Refer to a dermatologist for confirmation of diagnosis and chronic care.

Pearls

1. New-onset, severe, or prolonged seborrheic dermatitis may indicate a new HIV infection or immunosuppression.
2. Although there is no cure, reassure adult patients that the rash can be well controlled with topical medications.
3. In infants, seborrheic dermatitis can appear indistinguishable from Langerhans cell histiocytosis. Always have a clear discharge plan to follow up with a pediatrician or dermatologist.



FIGURE 13.24 ■ **Seborrheic Dermatitis.** Erythema and yellow-orange scales and crust on the scalp of an infant ("cradle cap"). Eczematous lesions are also present on the arms and trunk. (Used with permission from Wolff K, Johnson RA, Suurmond D. Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology. 5th ed. New York, NY: McGraw-Hill; 2005: 51.)



FIGURE 13.25 ■ **Seborrheic Dermatitis.** Intense erythema on the forehead and cheeks. (Photo contributor: David Effron, MD.)



FIGURE 13.26 ■ **Seborrheic Dermatitis.** Typical erythematous, scaling patches on the pinna and conchal bowl. Note also similar lesions on the anterior and posterior hairlines. (Photo contributor: J. Matthew Hardin, MD.)

PSORIASIS

Clinical Summary

Psoriasis has many forms. The most common is chronic plaque psoriasis with stable, symmetric lesions on the trunk and extremities, especially the elbows and knees. Lesions are well-defined, erythematous plaques with silvery scales. Inverse psoriasis represents a form that involves the intertriginous areas and, due to the moist environment, the silvery scale is absent. Guttate psoriasis, common in children and young adults, presents with an abrupt eruption of 2- to 5-mm erythematous scaly papules on the trunk and extremities. A preceding respiratory infection, usually streptococcal pharyngitis, can be a precipitant. Pustular forms of psoriasis can present as localized (nail bed, finger, palms, or soles) or generalized. It is characterized by erythema and "lakes of pus." Triggers for pustular psoriasis include steroid withdrawal (as in patients with chronic obstructive pulmonary disease [COPD] and asthma exacerbations), pregnancy, infections, and topical irritants.

Management and Disposition

ED management should ensure there is no infectious etiology or systemic symptoms. Localized psoriasis typically responds to topical glucocorticoids, although the chronicity and variety of other management options, including phototherapy, should prompt referral to dermatology. Pustular forms may be challenging and require admission. Guttate psoriasis may respond to antistreptococcal antibiotics. Obtain emergent consultation with a dermatologist for patients with generalized presentations and referrals for localized disease.

Pearls

1. Medication-induced psoriasis is associated with β-blockers, lithium, interferon, and antimalarials.
2. Patients with psoriasis have a higher incidence of coronary artery disease, obesity, tobacco use, and alcoholism.



FIGURE 13.27 ■ **Psoriasis.** Annular, well-defined plaque on the shin. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.28 ■ **Psoriasis.** Classic silvery scale associated with longstanding lesions. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.29 ■ **Psoriasis.** Note the erythematous plaques with diffuse fissuring in this case of palmar psoriasis. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.31 ■ **Psoriasis.** Erythematous plaques on the forehead and scaling in the scalp. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.30 ■ **Psoriasis.** Erythematous plaques on the upper arm and hand. (Photo contributor: J. Matthew Hardin, MD.)

PITYRIASIS ROSEA

Clinical Summary

The first sign of pityriasis rosea (PR) is usually a well-demarcated, salmon-colored macule that evolves into a larger patch (1-4 cm) with peripheral scaling ("herald patch"). Over 1 to 2 weeks, generalized, bilateral, and symmetric macules and plaques appear along cleavage lines. The macules have a peripheral collarette of fine scaling (termed "Christmas tree" pattern). Most patients will have severe itching associated with the generalized eruption. The lesions slowly resolve over 6 to 8 weeks. Atypical presentations in children include inverse PR (presentation on the face, axillae, and/or inguinal areas) and papular PR (peripheral scaling papules with central, hyperpigmented plaques). A viral etiology is postulated due to seasonal variation and case clustering.



FIGURE 13.32 ■ **Pityriasis Rosea Herald Patch.** The herald patch of PR, a well-demarcated salmon-colored macule with scales, frequently precedes the generalized phase by 1 to 2 weeks. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

PR is both benign and self-limited. Pruritus can be treated with oral antihistamines, topical steroids, and oatmeal baths.

Pearls

1. Patients often will not describe the herald patch unless specifically asked.
2. In patients with risk factors for syphilis and HIV, appropriate screening tests should be performed.
3. Patients should be warned of the possible extended course of PR and given appropriate antihistamines and follow-up.
4. Atypical presentations are seen in dark-skinned individuals.



FIGURE 13.33 ■ **Pityriasis Rosea.** An exanthematous, papulosquamous eruption, with the long axis of the oval papules following the lines of cleavage in a Christmas tree–like eruption. (Photo contributor: David Effron, MD.)



FIGURE 13.34 ■ **Pityriasis Rosea.** A more subtle Christmas tree–like eruption. (Photo contributor: Kevin J. Knoop, MD.)

ATOPIC DERMATITIS

Clinical Summary

Atopic dermatitis presents in three overlapping stages: infantile, childhood, and adult. Infantile begins after 2 months of age and is symmetrically distributed on the cheeks, scalp, neck, forehead, and extensor surfaces of the extremities. The lesions begin as erythema or papules, but, with persistent itching and rubbing, they become thin plaques, exudative and crusted. Childhood atopic dermatitis presents with flexural involvement. Other areas frequently involved are the face, neck, and trunk. The scratching induces plaque lichenification and potential for secondary infection. Adult atopic dermatitis is less specific but can present with a childhood-like distribution, papular lesions that coalesce into plaques, and chronic hand dermatitis. Atopic dermatitis can become a generalized exfoliative erythroderma. Differential diagnoses include seborrheic dermatitis, psoriasis, irritant or allergic contact dermatitis, nummular eczema, and scabies.

Management and Disposition

If a severe flare, suprainfection, punched-out lesions (eczema herpeticum), or a generalized exfoliative erythroderma is present, an ED dermatologic consultation is indicated. Mild cases can be sent home with referral. Patients (caregivers) should avoid soaps, detergents, or any personal products with fragrances. After bathing, pat dry the skin and smear a thin film of petrolatum or mild corticosteroid over the affected areas. Wearing damp pajamas (100% prewashed cotton) after application of emollients/mild corticosteroids can help rapidly heal the skin.

Pearls

1. Atopic dermatitis is often called the "itch that rashes" since pruritus precedes clinical disease.
2. If dispensing corticosteroids, use appropriate classes for the affected site and patient age. In addition, specific instructions should be written for the patient.
3. Frequent relapses are common and require an astute clinician to differentiate associated complications.



FIGURE 13.35 ■ **Atopic Dermatitis.** A typical localization of atopic dermatitis in children is the region around the mouth, with lichenification and fissuring and crusting. (Used with permission from Wolff K, Johnson RA, Saavedra AP. Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology. 7th ed. McGraw-Hill; 2013: Fig. 2-14.)

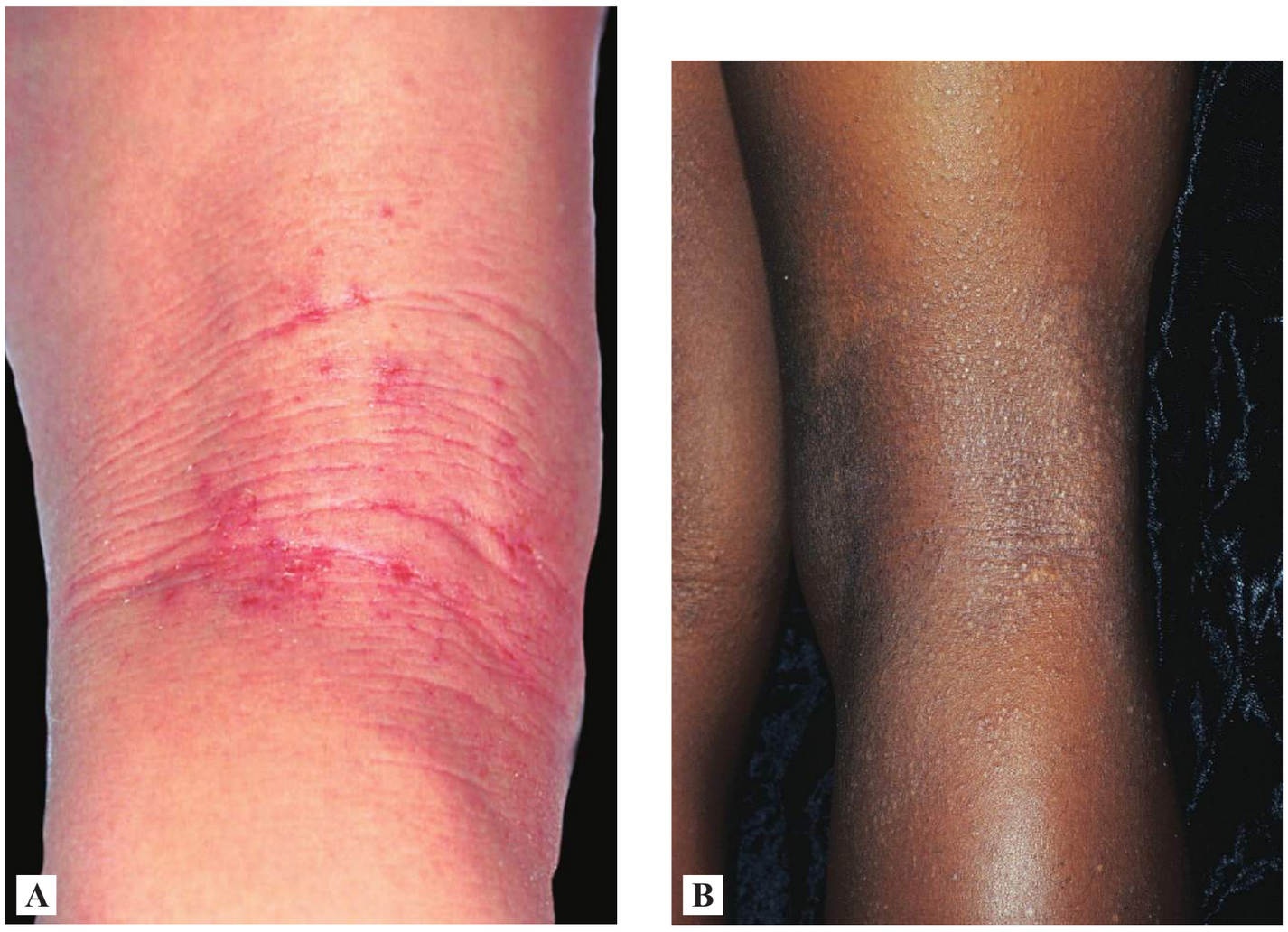


FIGURE 13.36 ■ **Atopic Dermatitis.** (**A**) One of the hallmarks of atopic dermatitis is lichenification in the flexural regions. Note the thickening of the skin with exaggerated skin lines and erosions. (**B**) Atopic dermatitis in black child. Pruritic follicular papules on posterior leg. Follicular eczema pattern is more common in African and Asian children. (Used with permission from Wolff K, Johnson RA, Saavedra AP. Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology. 7th ed. McGraw-Hill; 2013: Fig. 2-15 A and B.)



FIGURE 13.37 ■ **Atopic Dermatitis.** Lichenfied plaques, erosions, and fissures are characteristic of adult atopic dermatitis. (Photo contributor: James J. Nordlund, MD.)

NUMMULAR/XEROTIC ECZEMA

Clinical Summary

Nummular eczema is characterized by an erythematous, edematous, vesicular, and crusted extremity plaque. The lesions enlarge by forming satellite, peripheral papulovesicles that coalesce with the original lesion. Pruritus is the dominant symptom.

Xerotic eczema (also called winter itch, eczema craquele, and asteatotic eczema) presents on the anterior shins, extensor arms, and flanks. The lesions are erythematous patches with fine, cracked fissures and adherent scaling. The edema and exudate present in nummular eczema is absent. Pruritus can be severe. This is a common finding in the winter and in the elderly.

Management and Disposition

Treatment of nummular eczema consists of mid- to high-potency topical steroids under occlusion. Prevention of secondary infection is important as patients frequently cannot resist scratching.

Xerotic eczema is treated with topical emollients (petrolatum), three to four applications per day. Topical steroids may be required for areas with inflammation.



FIGURE 13.38 ■ **Eczema.** Nummular eczema of the wrist. Note the satellite lesions on the periphery. (Photo contributor: J. Matthew Hardin, MD.)

Pearls

1. Nummular eczema should be considered with lesions unresponsive to antibiotics and pruritus as the dominant feature.
2. Both of these entities are associated with significant pruritus and secondary infections, especially in the young and elderly.



FIGURE 13.39 ■ **Eczema.** Nummular eczema on the extensor surface of the forearms and elbows. Thickened, scaly lesions resemble psoriatic plaques. A biopsy was performed to confirm the diagnosis of nummular eczema. (Photo contributor: Richard P. Usatine, MD. Used with permission. From Usatine RP, Smith MA, Mayeaux EJ, Chumley HS. The Color Atlas of Family Medicine. 2nd ed. McGraw-Hill; 2013: Fig. 148-6.)

Clinical Summary

Dyshidrotic eczema (also called pompholyx or acute vesiculobullous hand eczema) is the abrupt appearance of deep-seated, 1- to 2-mm vesicles on the sides of the fingers, palms, and soles. The vesicles are extremely pruritic, may coalesce into larger bullae, and may rupture to become dry or fissured. The outbreak usually resolves over a few weeks unless secondary infection develops. The differential includes bullous tinea, id reaction, scabies infestation, or allergic contact dermatitis.

Management and Disposition

Treatment includes a high-potency topical steroid and prevention of secondary infection. Refer to a dermatologist for long-term treatment; this is often a chronic condition with significant disability.

Pearls

1. The initial lesions resemble tapioca pudding.
2. These lesions wax and wane and are often exacerbated by stressful events.



FIGURE 13.40 ■ **Dyshidrotic Eczema.** In most cases dyshidrotic eczematous dermatitis starts with tapioca-like vesicles on the lateral aspects of the fingers. (Photo contributor: Richard P. Usatine, MD. Used with permission. From Usatine RP, Smith MA, Mayeaux EJ, Chumley HS. The Color Atlas of Family Medicine. 2nd ed. McGraw-Hill; 2013: Fig. 147-7.)



FIGURE 13.41 ■ **Dyshidrotic Eczema.** The vesicles show confluence and spread to the palm but also to the wrist and dorsal aspect of the hand when the eruption progresses. (Used with permission from Wolff K, Johnson RA, Suurmond D. Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology. 5th ed. New York, NY: McGraw-Hill; 2005:45.)

ID REACTION (DISSEMINATED ECZEMA)

Clinical Summary

Id reactions are seen in response to a variety of disorders, including fungal infection (tinea capitis and tinea pedis), scabies infestation, pediculosis capitus, molluscum contagiosum, bacterial and mycobacterial infections, and arthropod bites. The rash appears days to weeks after the instigating rash and consists of erythematous papules (sometimes crusted at the apices) as well as eczematous patches and plaques. The rash can be local to the instigating lesions/rash, distant, or generalized. The id reaction usually presents on the extremities, commonly on the sides of fingers, but may occur on the face and trunk. Pruritus is intense. The id reaction will not demonstrate infectious organisms and may not respond to topical steroids.

Management and Disposition

Recognition and treatment of the initial infection or infestation is curative. Refer to a dermatologist for follow-up to ensure diagnosis and resolution.

Pearls

1. Repeated ED evaluation for fungal infection or eczematous rash should prompt further investigation for a distant, untreated, or occult fungal infection.
2. Id reactions are intensely pruritic; make sure secondary bacterial infections do not develop from excoriations.
3. Recurrences are common, especially if the primary source is not treated adequately.



FIGURE 13.42 ■ **Id Reaction.** Bullous tinea pedis causing id reaction on the fingers. (Used with permission from Wolff K, Johnson RA, Suurmond D. Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology. 5th ed. New York, NY: McGraw-Hill; 2005: 48.)



FIGURE 13.43 ■ **Id Reaction.** Id reaction to tinea pedis. Erythematous, partially dried up, pruritic vesicles on the foot. (Used with permission from Goldsmith LA, Katz SI, Gilchrest BA, Paller AS, Leffell DJ, Wolff K. Fitzpatrick's Dermatology in General Medicine. 8th ed. McGraw-Hill; 2012: Fig. 16-7.)

Clinical Summary

Stasis dermatitis results from venous insufficiency, is characteristically distributed on the distal tibia above the medial malleolus, and appears early with erythematous patches. These can progress to scaling as well as eczematous and weeping plaques. Patients will often have light brown pigmentation distributed on the lower third of the extremity due to microvasculature blood extravasation (hemosiderin deposition secondary to increased superficial capillary pressure). Varicose veins are usually present, although they are often difficult to visualize in obese patients. Patients with heart failure, cirrhosis, and nephrotic syndrome are at increased risk due to a chronic edematous state.

Management and Disposition

Referral to a primary care physician should be initiated to address the underlying etiology (eg, valvular insufficiency, thromboembolic disease, chronic edematous state). Daily elevation of the extremities and compression hose use should be encouraged. Emollients and mid-potency topical steroids help decrease the pruritus and promote healing.

Pearls

1. Differentiation of stasis dermatitis and early cellulitis can be extremely difficult. Clinical history and deviation from previous presentations may be helpful. Follow-up for reevaluation should be obtained.
2. Due to the chronic and repetitive nature of stasis dermatitis, patients use many over-the-counter products. These may contribute to allergic and irritant contact dermatitis.
3. An associated contact dermatitis may initiate an "autosensitization" rash—presenting with erythematous patches on the legs and arms. Stop nonessential topical medications or other products.



FIGURE 13.44 ■ **Stasis Dermatitis.** Erythematous patches and mild scaling in a patient with chronic venous insufficiency. Note location above the ankles. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 13.45 ■ **Stasis Dermatitis.** An example of stasis dermatitis showing erythematous, scaly, and oozing patches over the lower leg. Several stasis ulcers are also present. (Used with permission from Fauci AS, Braunwald E, Kasper DL, et al. Harrison's Principles of Internal Medicine. 17th ed. New York, NY: McGraw-Hill; 2008: 315.)

LIVEDO RETICULARIS

Clinical Summary

Livedo reticularis is a reactive macular, reticulated (lace-like) patch of nonpalpable cutaneous vasodilatation due to vasospasm, vessel wall damage, and intraluminal pathology. It occurs in many systemic diseases including connective tissue disorders, vasculitis, polycythemia vera, cold agglutinins, hypercoagulability diseases, thrombotic thrombocytopenic purpura, embolic disease, decompression sickness, and infections/sepsis. It has also been associated with medications such as amantadine, quinine, and quinidine. Physiologic livedo reticularis is a response to cold temperatures and is a common finding in infants, children, and adults prone to acrocyanosis.

Management and Disposition

Management is dependent on treating the underlying disorder. Patients without an acute medical condition can be referred to dermatology.

Pearls

1. Physiologic livedo reticularis improves or disappears with warming, whereas secondary causes usually do not.
2. Patchy, nonsymmetric distribution of livedo reticularis should elicit concern for more serious underlying diseases.



FIGURE 13.46 ■ **Livedo Reticularis.** A netlike, arborizing pattern on the posterior thighs and buttocks defined by violaceous, erythematous streaks resembling lightning. The skin within the erythematous areas is normally pale. (Used with permission from Wolff K, Johnson RA, Saavedra AP. Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology. 7th ed. McGraw-Hill; 2013: Fig. 14-42.)



FIGURE 13.47 ■ **Livedo Reticularis.** Reticulated (lacelike) branching erythema symmetrically distributed over the lower extremities (Photo contributor: James J. Nordlund, MD.)

VASCULITIS (LEUKOCYTOCLASTIC VASCULITIS)

Clinical Summary

Leukocytoclastic vasculitis (LCV) represents the deposition of immune complexes in small blood vessels with subsequent vessel damage and blood extravasation. Nonblanching, purpuric macules and erythematous papules frequently coalesce into plaques ("palpable purpura"). The lower extremities and dependent areas of the back and buttocks are most frequently involved. Pruritus can be significant. Vesicles, ulcers, and necrosis may be seen within the purpuric lesions. A crop of lesions appear over a few days and usually resolve with hyperpigmentation over 4 to 6 weeks. Associated symptoms may include fever, arthralgias, myalgias, and malaise.

LCV is associated with many chronic diseases (connective tissue diseases, malignancies, and inflammatory bowel disease), medications (cephalosporins, penicillins, sulfonamides, minocycline, thiazides, allopurinol, phenytoin, NSAIDs, oral contraceptives, antithyroid agents), infections (group A β-hemolytic streptococci, mycobacterium leprae, viral hepatitis, HIV), and idiopathic subtypes (Henoch-Schönlein purpura [HSP], acute hemorrhagic edema of childhood, urticarial vasculitis).

HSP is a unique form of LCV with palpable purpura of the lower extremities and buttocks. Occasionally, the lesions may be found on the upper extremities, trunk, and face. A recent respiratory infection, arthralgias, abdominal pain, and hematuria are commonly noted. Renal vasculitis can occur in up to 40%, but chronic renal impairment occurs in only 2%. Adults have a tendency to have necrotic lesions (rare in children), as well as a higher incidence of renal impairment.

Urticarial vasculitis presents with burning and painful urticaria. The lesions are distinguished from common urticaria by their persistence (lasting over 24 hours), associated purpura, and resolution with hyperpigmentation. Urticarial vasculitis can be seen with connective tissue disease, viral infections, medications (NSAIDs, fluoxetine, cocaine, and others), and malignancies.

Management and Disposition

Recognition that a diverse group of diseases can trigger LCV is the first step. Evaluation for systemic symptoms (fever or other signs of infection, hematuria, gastrointestinal bleeding, and neurologic symptoms) requiring admission and appropriate consultation should be undertaken. Most cases are self-limited and only require supportive care (rest, elevation, antihistamines, and analgesics). Systemic symptoms require admission and consideration of corticosteroids and other immunosuppressants. Dermatologic referral for mild LCV cases is indicated and, if systemic symptoms present, consultation may help expedite the diagnosis. HSP and urticarial vasculitis require dermatologic consultation as well as appropriate specialty consultations.

Pearls

1. LCV affects all ages and has equal incidence in males and females. The etiologies are often idiopathic, but scrutinize each patient for associated diseases and medicine exposures.
2. Renal involvement associated with HSP is more common in adults and lesions above the waist.



FIGURE 13.48 ■ **Leukocytoclastic Vasculitis.** Lower extremities with erythematous papules and dorsal foot with erythematous plaques. If you were to run your finger across these lesions, they would be raised and not blanch. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 13.49 ■ **Leukocytoclastic Vasculitis.** Acute necrotic LCV. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.51 ■ **Henoch-Schönlein Pupura.** Note the classic acral distribution of HSP (both upper and lower extremities—raising concern for renal involvement). (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 13.50 ■ **Leukocytoclastic Vasculitis.** Note the erythematous papules beginning to coalesce. These would not blanch with pressure. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.52 ■ **Urticarial Vasculitis.** Urticarial plaques present for over 24 hours. (Photo contributor: J. Matthew Hardin, MD.)

IDIOPATHIC THROMBOCYTOPENIC PURPURA

Clinical Summary

Idiopathic thrombocytopenic purpura (ITP) occurs as a result of platelet injury and destruction (via IgG autoantibodies to the platelet surface). Pinpoint, red, nonblanching petechiae or nonpalpable purpura and ecchymoses are found on the skin and mucous membranes, either spontaneously (platelets <10,000/mm^3) or at the site of minimal trauma (platelets <40,000/mm^3). Petechiae are commonly found on dependent areas. Gingival bleeding, melena, hematochezia, menorrhagia, and severe intracranial hemorrhages may also occur in conjunction with the thrombocytopenia. The acute form affects children (peak incidence 2-4-year-olds, equal gender distribution) after a viral illness and completely resolves in 2 months. The chronic form occurs most often in adults, with women outnumbering men 3:1. Other causes of thrombocytopenia include infections (HIV, hepatitis), medications (heparin, quinidine/quinine, sulfonamides), and alcohol.

Management and Disposition

Consult hematology for medical management guidance. Treatment can include IV corticosteroids, IVIg, or IV Rho (D) immune globulin alone or in combination. If life-threatening hemorrhage is present, IV methylprednisolone and IVIg should be initiated along with platelet transfusions. For adult patients with longstanding, recalcitrant disease, splenectomy may be recommended.

Pearls

1. Isolated thrombocytopenia is the hallmark finding—WBC, hemoglobin, and coagulation levels should be normal (if no major hemorrhage).
2. Patients with advanced age, comorbidities, and prior history of hemorrhage have a high risk of associated bleeding (eg, central nervous system). Keep a low threshold for imaging and insist on appropriate disposition.
3. IV Rho (D) immune globulin is only used in nonsplenectomized, Rh positive patients. Associated intravascular hemolysis can be seen and should be monitored in the ED if IV Rho (D) immune globulin used.
4. The acute form of ITP has an excellent prognosis (90% spontaneous remission), whereas the course of chronic ITP is one of varying severity with little hope of remission.



FIGURE 13.53 ■ **Idiopathic Thrombocytopenic Purpura.** This thrombocytopenic patient with splenomegaly has pinpoint, nonblanching, nonpalpable petechiae. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 13.54 ■ **Idiopathic Thrombocytopenic Purpura.** Nonpalpable purpura in ITP. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Primary (idiopathic) thrombotic thrombocytopenic purpura (TTP) is characterized by the following pentad of symptoms: (1) microangiopathic hemolytic anemia (with characteristic schistocytes on the peripheral blood smear and a reticulocytosis), (2) thrombocytopenia with platelet counts ranging from 5000 to 100,000/μL, (3) renal abnormalities (including insufficiency, azotemia, proteinuria, or hematuria), (4) fever, and (5) neurologic abnormalities (including headache, mental status changes, cranial nerve palsies, seizures, or coma). Clinically, patients will have petechial rash and may have pallor and jaundice secondary to hemolytic anemia and thrombocytopenia.

Secondary TTP-hemolytic uremic syndrome (TTP-HUS) is associated with hemorrhagic colitis (Escherichia coli O157:H7 and Shigella toxin mediated), pregnancy, medications (cyclosporine, tacrolimus, ticlopidine, gemcitabine, mitomycin C, and others), metastatic carcinoma, autoimmune connective tissue disease, and antiphospholipid syndrome.

Management and Disposition

The cornerstone of primary TTP therapy is total plasma exchange transfusion (plasmapheresis) with fresh frozen plasma. Some patients can be treated with simple plasma infusions alone until transferred to site with total plasma exchange transfusion. Patients recalcitrant to standard therapy may be treated with immunosuppressives (systemic corticosteroids, vincristine, or rituximab) and splenectomy.

Patients with TTP-HUS require supportive care with close attention to fluid balance (especially diarrheal dehydration). Signs of renal failure require emergent nephrology consultation for hemodialysis. Total plasma exchange is reserved for recalcitrant cases in children (majority improve with supportive care alone). Antibiotic use should be avoided due to an associated 17-fold increase in disease progression (presumably by bacterial release of toxin). Avoid antimotility medications with diarrheal disease.

Pearls

1. Since only 20% to 30% of patients have the typical symptom pentad, diagnosis based on partial findings is challenging.
2. Platelet transfusions should be avoided unless there is life-threatening hemorrhage; platelet transfusions can worsen the thrombotic process leading to myocardial infarction and cerebrovascular infarctions.
3. Disseminated intravascular coagulation and the pregnancy-associated HELLP (hemolysis, elevated liver enzymes, low platelet count) syndrome can present with similar findings to primary TTP and TTP-HUS.



FIGURE 13.55 ■ **Thrombotic Thrombocytopenic Purpura.** Bleeding at initial presentation is seen in about 30% to 40% of patients with TTP. (Photo contributor: James J. Nordlund, MD.)

URTICARIA AND DERMATOGRAPHISM

Clinical Summary

Acute urticaria develops over days to weeks and presents with transient wheals. Generally, acute urticaria resolves within six weeks, whereas chronic urticaria lasts longer. Common triggers include medications (penicillin, aspirin, NSAIDs), foods (chocolate, shellfish, nuts, eggs, milk, and others) and infections (streptococcal, hepatitis B and C, mononucleosis, and helminths), and physical factors (exercise, pressure, cold, vibratory and solar-induced urticaria).

Dermatographism is the production of linear urticarial lesions and surrounding erythematous flare after stroking the skin. Simple dermatographism, seen in up to 5% of the population, is considered an exaggerated physiologic response to friction. In contrast, symptomatic dermatographism presents without a previous history. Wheals are typically seen with scratching and friction from tight clothing. Children and young adults are commonly affected and, fortunately, no associated systemic disease, autoimmunity or food allergy exists. Symptomatic dermatographism can persist for several years.

Management and Disposition

Triggers of urticaria should be investigated and, if present, stopped. H1 and H2 blockers usually help. Systemic steroids and epinephrine are used for severe reactions and anaphylaxis.

Pearls

1. More than 50% of chronic urticaria is idiopathic.
2. Wheals present for over 24 hours and in the same location are concerning for urticarial vasculitis (see related item) and should prompt dermatology consultation. Draw a circle around the lesions and have the patient monitor changes.



FIGURE 13.56 ■ **Acute Urticaria.** Note the classic raised plaques on the lower extremity of this patient with urticaria. (Photo contributor: James J. Nordlund, MD.)

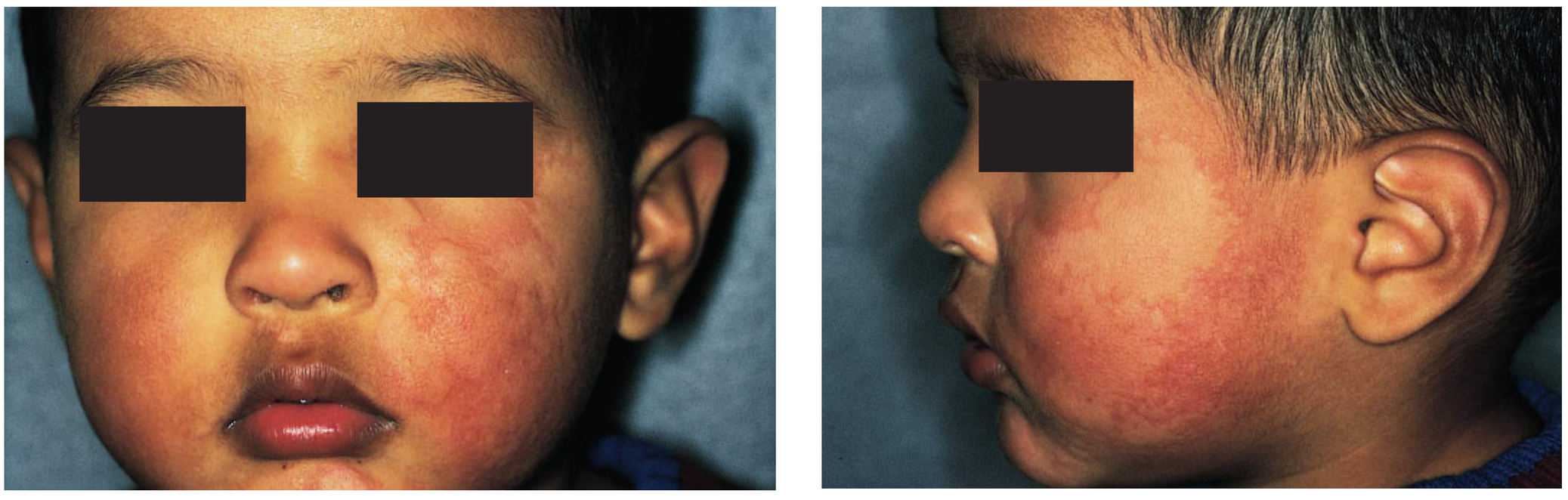


FIGURE 13.57 ■ **Acute Urticaria.** Preschool child with annular, raised pruritic lesions with central clearing and tense edema. The lesions disappeared approximately 5 minutes after presentation. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 13.58 ■ **Cold Urticaria.** Plaque formation after placement of an ice cube on the skin confirms cold urticaria. (Photo contributor: James J. Nordlund, MD.)



FIGURE 13.59 ■ **Dermatographism.** Note the linear wheal with surrounding erythematous flare after scratching and stroking. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.60 ■ **Dermatographism.** Urticaria 5 minutes after scratching. (Used with permission from Wolff K, Johnson RA, Saavedra AP. Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology. 7th ed. McGraw-Hill; 2013: Fig. 14-9.)

ALLERGIC CONTACT DERMATITIS

Clinical Summary

Allergic contact dermatitis occurs after previously sensitized skin is rechallenged with the same allergen. It represents a delayed-type hypersensitivity reaction. Papules and vesicles first develop; they can become a generalized morbilliform eruption (autosensitization). Pruritus is a dominant feature. The most common causes of allergic contact dermatitis are nickel, toxicodendrons (poison ivy, poison oak, and poison sumac), neomycin, fragrances, balsam of Peru (common in perfumes), formaldehyde, bacitracin, and rubber compounds.

Management and Disposition

Identification of the causative agent and prevention of further contact is critical. Supportive care is given with antihistamines and topical corticosteroids. Systemic corticosteroids may be needed for generalized eruptions. Refer patients to dermatology for further evaluation and possible patch testing.

Pearls

1. Obtaining a complete history of any and all exposures is critical. Pay particular attention to personal hygiene products.
2. Toxicodendron allergic contact dermatitis (poison ivy, poison sumac, poison oak) requires a minimum 3-week taper of oral prednisone—a shorter course allows the reappearance of the rash.
3. Bacitracin and neomycin cause 9% and 10% of allergic contact dermatitis cases, respectively.



FIGURE 13.62 ■ **Contact Dermatitis.** The erythematous, edematous base of the eruption corresponds to the posterior watch surface. Superimposed on the erythematous base are multiple vesicles with exudate and crust. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)



FIGURE 13.61 ■ **Contact Dermatitis.** Erythematous eruption in a waist-band distribution (elastic allergy from underwear). (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.63 ■ **Contact Dermatitis.** Allergy to tattoo pigment. Note erythema, scale, and erosions contained within the tattoo image. (Photo contributor: J. Matthew Hardin, MD.)

Clinical Summary

Erythema nodosum (EN) can present at any age but is most common in young, adult females. The most typical presentation is bilateral, erythematous, subcutaneous, tender nodules on the pretibial and lateral lower extremities (usually spares the posterior calves). Rarely, the nodules can be found on the thighs, upper extremities, and face. Concomitant symptoms often include lower extremity edema and arthralgias. Systemic symptoms can occur and include fever, headache, and gastrointestinal complaints. Generally, the nodules resolve over days to weeks with flattening and a change in color to a blue-green (like a deep bruise). There is no ulceration and the skin slowly returns to normal. Recurrence occurs in up to one-third of cases.

Multiple etiologies can present with EN—although over one-third of cases are idiopathic. Infectious causes include streptococcal, tuberculosis, Yersinia, Salmonella, Shigella, coccidioidomycosis, histoplasmosis, sporotrichosis, blastomycosis, and toxoplasmosis. EN has also been associated with pregnancy, sarcoidosis, and inflammatory bowel disease. Oral contraceptives, sulfonamides, bromides, and iodides are known to be common causative agents, among many others.

Management and Disposition

With the many etiologies of EN, it is critical to exclude and treat an infectious, systemic, or medication cause. Supportive care with elevation of the extremity, rest, and NSAIDs is prudent. Recurrences do occur and should prompt a further workup for occult infection or persistent medication. Referral to a dermatologist for a biopsy may help confirm the diagnosis.



FIGURE 13.64 ■ **Erythema Nodosum, Acute.** Note the pretibial, erythematous, and subcutaneous nodules. (Photo contributor: Gianina Best, MD.)

Pearls

1. The patient's history is very helpful in determining possible etiologies. A complete medication, travel, and past medical history must be performed.
2. Systemic glucocorticoids can be considered, but only when the etiology is clearly known and infectious agents are excluded.
3. EN is considered a good prognostic sign in sarcoidosis and pregnancy-associated coccidioidomycosis.



FIGURE 13.65 ■ **Erythema Nodosum, Resolving.** Note the bruise-like appearance of the resolving phase. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.66 ■ **Erythema Nodosum.** Tender subcutaneous nodules with overlying erythematous and hyperpigmented macules—indicating a new crop of lesions. (Photo contributor: J. Matthew Hardin, MD.)

ERYSIPELAS

Clinical Summary

Erysipelas is a group A streptococcal cellulitis (groups G, B, C, and D can be implicated) involving the skin. The most commonly affected sites are the face and the lower extremities. Sudden onset of fever, chills, and malaise is followed by the appearance of an erythematous, edematous, and painful plaque. The characteristic border is sharp and elevated. This nonpitting edematous plaque is different from typical cellulitis due to dilation of the superficial lymphatics. Regional lymphadenopathy and lymphangitis may be present. Patients with chronic lymphedema are prone to repeated infections.

Management and Disposition

All infections require rest, elevation, and antibiotics. Mild presentations may be treated on an outpatient basis with oral penicillins. More severe illness or toxicity requires hospitalization and IV antibiotics.



FIGURE 13.67 ■ **Erysipelas.** Sharply demarcated and elevated erythema. (Photo contributor: David Effron, MD.)

Pearls

1. The sharp elevated border is diagnostic of erysipelas.
2. A similar shiny, erythematous (although more violaceous) plaque on the face of a febrile child may be caused by Haemophilus influenza type B in nonimmunized or immunosuppressed individuals.



FIGURE 13.68 ■ **Erysipelas.** Note the well-demarcated, edematous, erythematous, shiny plaque. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)

Clinical Summary

Infective endocarditis (IE) is characterized by infection of the endocardium (including the valves, mural endocardium, or septal defect) manifested by a number of cutaneous findings. Causes of IE are diverse and can be broadly categorized as (1) acute and subacute native valve IE, (2) early and late prosthetic valve IE, (3) IV drug abuse IE, and (4) iatrogenic IE. The latter encompasses IE associated with recent hospital admission or a healthcare procedure causing bacteremia or endocardial damage. Overall, Staphylococcus aureus is the most common causative organism but many other bacteria and fungi are implicated. Janeway lesions (septic emboli forming microabscesses) are nontender, small, erythematous (occasionally with central hemorrhage) macules on the palms or soles. Osler nodes (immune complex deposition resulting in small vessel vasculitis) consist of transient, tender, purplish nodules on the pulp of the fingers and toes. Subungual splinter hemorrhages are black, linear discolorations beneath the conjunctiva and nail plate. Murmurs, retinal hemorrhages, septic arthritis, and significant embolic episodes such as pulmonary embolism or cerebral vascular embolism may also be present.

Management and Disposition

Empiric IV antibiotic therapy is the mainstay of treatment. Obtain blood cultures prior to antibiotic administration. Consult cardiology for emergent, echocardiographic assessment of the cardiac valves. Consult infectious disease for empiric antibiotic recommendations. Emergent surgical intervention may be required for valvular dysfunction or associated abscess.

Pearls

1. Acute bacterial endocarditis presents with fever (over 90% of patients) and a toxic patient—this contrasts with a more insidious presentation of subacute bacterial endocarditis.
2. Previous episode of IE, congenital heart disease, IV drug abuse, prosthetic heart valves, recent medical procedures, and cardiac transplants with valvulopathies are risk factors.
3. Although splinter hemorrhages in the nail bed have long been associated with IE, the most common causes are trauma, psoriasis, and onychomycosis. Proximal nail bed splinter hemorrhages have a higher likelihood of systemic disease (including IE, drug reactions, vasculitis, antiphospholipid antibody syndrome, and trichinosis).



FIGURE 13.69 ■ **Janeway Lesions.** Peripheral embolization to the sole, resulting in a cluster of nontender, erythematous macules known as Janeway lesions. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)



FIGURE 13.70 ■ **Janeway Lesion.** Embolization to the hand in a patient with infectious endocarditis. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 13.71 ■ **Osler Nodes.** Tender, subcutaneous, violaceous nodules in the pulp of the fingers known as Osler nodes. (Photo contributor: Armed Forces Institute of Pathology, Bethesda, MD.)

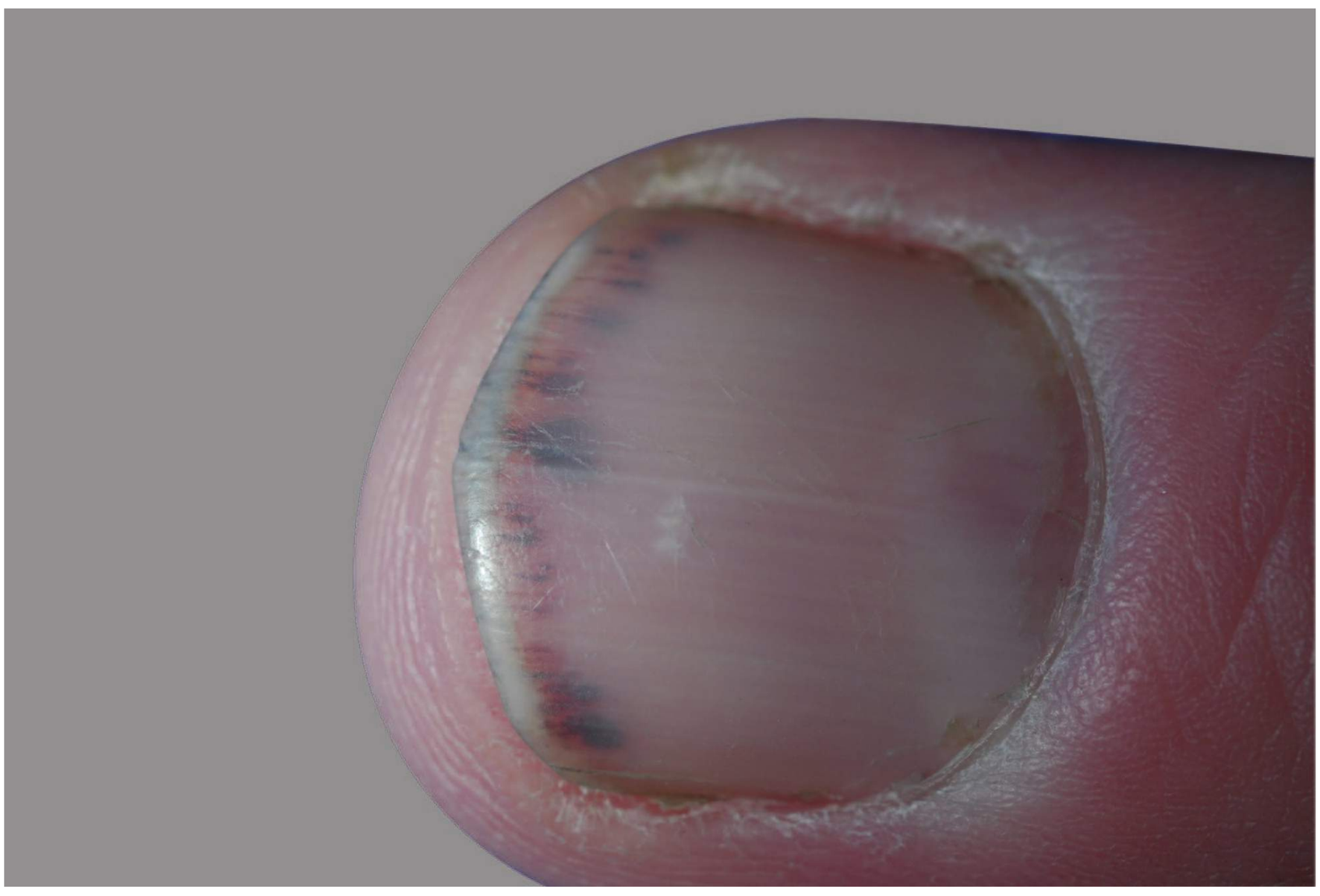


FIGURE 13.72 ■ **Splinter Hemorrhages.** Note the splinter hemorrhages along the proximal and distal aspect of the nail plate—emboli from subacute bacterial endocarditis. (Photo contributor: R. Jason Thurman, MD.)

Clinical Summary

Borrelia burgdorferi is the tick-borne spirochete responsible for Lyme disease, and erythema chronicum migrans (ECM) is the pathognomonic rash. ECM typically presents 1 to 2 weeks after the bite. The initial prodromal symptoms of fever, myalgias, arthralgias, and headache are followed by a macule or papule progressing to a plaque at the bite site. This plaque expands (usually to 5 cm or larger) its red, raised border as it clears centrally, leading to an annular appearance ("bull's eye"). The plaque may burn and is rarely pruritic. Less frequently, secondary ECM-like lesions can appear due to multiple bites or spirochetemia. ECM is seen in 60% to 90% of patients and represents the early localized stage.

Management and Disposition

The duration and choice of antibiotic depends on the clinical features (presence of ECM, early disseminated disease with mild or severe symptoms, cranial nerve palsies, heart block, meningitis, and radiculopathy). Doxycycline is the drug of choice for adults and children over 8 years of age. Pregnant or lactating females and children younger than 8 years of age should be treated with amoxicillin. Patients with minimal symptoms may be treated and followed up on an outpatient basis. Those patients with significant toxicity (severe disseminated rash, systemic symptoms, meningitis, radiculopathy, or third-degree heart block) require admission, supportive care, and IV antibiotics.

Pearls

1. Over 50% of untreated ECM can progress to an asymmetric, episodic, oligoarticular arthritis weeks to months after the initial infection.
2. Facial nerve palsies are the most common neurologic manifestation of untreated Lyme disease.
3. Early serology may not demonstrate elevated anti-Borrelia antibodies and, if not followed up, one may miss the diagnosis.



FIGURE 13.73 ■ **Erythema Chronicum.** The eruption of Lyme disease forms at the site of the tick bite. The initial papule forms into an expanding oval of erythema. There is central "bull's eye" clearing as the erythema progresses. (Photo contributor: James Gathany, Public Health Image Library, US Centers for Disease Control and Prevention.)



FIGURE 13.74 ■ **Erythema Chronicum.** An atypical erythematous patch of erythema chronicum—always have a high level of suspicion. (Photo contributor: David Effron, MD.)

ROCKY MOUNTAIN SPOTTED FEVER

Clinical Summary

Rickettsia rickettsia, the causative organism of Rocky Mountain spotted fever (RMSF), is transmitted by the bite of an infected tick. Fever, headache, rigors, abdominal pain, myalgias, and malaise occur 2 to 14 days after inoculation. Three to five days after the onset of symptoms, the rash begins with erythematous, blanching macules on the distal extremities (wrists and ankles). This is followed by centripetal spread to the trunk and to the palms and soles. The lesions evolve into papules and petechia. Without treatment, RMSF has a 25% mortality; delayed diagnosis and delayed antimicrobial treatment results in 3% to 4% mortality.

Management and Disposition

Prompt initiation of doxycycline is recommended for adults and children. Consultation with an infectious disease specialist should be initiated to help guide treatment—this is especially critical for pregnant females. Mildly ill patients may be treated with oral antibiotics on an outpatient basis as long as close follow-up can be confirmed. More severely ill patients require admission.



FIGURE 13.76 ■ **Rocky Mountain Spotted Fever.** Early erythematous and hemorrhagic macules and papules appeared initially on the ankles of an adolescent. (Used with permission from Wolff K, Johnson RA, Saavedra AP. Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology. 7th ed. McGraw-Hill; 2013: Fig. 25-49.)



FIGURE 13.75 ■ **Rocky Mountain Spotted Fever.** These erythematous macular lesions will evolve into a petechial rash that will spread centrally. (Photo contributor: Daniel Noltkamper, MD.)

Pearls

1. Typical extremity lesions in a severely ill patient should be treated as RMSF until proven otherwise. Treatment should not be delayed for laboratory confirmation.
2. Most cases occur between April and October, with the highest US incidence occurring in the Southeast and South-Central states (not Rocky Mountain states).
3. Forty percent of patients do not recall the inciting tick bite.
4. Up to 15% of cases present without any cutaneous manifestations. Patients with darker skin types may have less obvious cutaneous findings.
5. In pediatric patients less than 9 years old, the recommended course of doxycycline has a negligible effect on permanent tooth discoloration. Previously recommended chloramphenicol has a higher mortality rate than doxycycline.

DISSEMINATED GONOCOCCUS

Clinical Summary

Disseminated gonococcus (gonococcemia) is a systemic infection that presents in 1% to 3% of untreated mucosal (urethral, endocervical, pharyngeal, and rectal) gonorrhea infections. The hematogenous dissemination of Neisseria gonorrhea results in fever, arthralgias, and scattered pustules. The initial lesion is an erythematous macule that evolves into a papule and hemorrhagic pustule. Petechial or purpuric macules can be seen as well. These lesions are few in number, asymmetric, painful, and predominantly located on the distal extremities. The spectrum of disease varies from skin lesions alone to associated tenosynovitis, septic arthritis, endocarditis, and meningitis.

Management and Disposition

Therapy consists of IV or intramuscular ceftriaxone until symptoms either improve or resolve, followed by an additional 7 days of orally administered cefixime. Hospitalization is recommended for initial treatment. Dermatology consultation may help establish the diagnosis.

Pearls

1. Pregnant or menstruating females have a higher risk of disseminated gonococcemia.
2. Disseminated gonococcemia is the most common cause of septic arthritis in young, sexually active adults.



FIGURE 13.77 ■ **Disseminated Gonococcus.** Vesiculopustule on the hand of a patient with disseminated gonococcus. (Photo contributor: Stephan E. Russ, MD.)



FIGURE 13.78 ■ **Disseminated Gonococcus.** Erythematous macules of disseminated gonococcus—these will evolve into hemorrhagic pustules. (Photo contributor: David Effron, MD.)

HOT TUB FOLLICULITIS/PSEUDOMONAS HOT-FOOT SYNDROME

Clinical Summary

Hot tub folliculitis is a pruritic, pustular eruption confined to the hair follicle and is secondary to a cutaneous infection with Pseudomonas aeruginosa. Patients have a history of hot tub, whirlpool, or swimming pool exposure within 24 to 72 hours of the eruption. The lesions have a predilection for areas covered with bathing suits. Headache, sore throat, earache, and fever may accompany the pustules (but do not necessarily indicate systemic disease).

Pseudomonas hot-foot syndrome is related to hot tub folliculitis; patients report wading in a swimming pool (later discovered to have elevated P aeruginosa concentrations). The weight-bearing aspects of the soles have multiple 1 to 2 cm erythematous nodules. Unlike hot tub folliculitis, these lesions are exquisitely painful and not associated with other symptoms. Like hot tub folliculitis, the lesions resolve spontaneously without treatment.



FIGURE 13.79 ■ **Hot Tub Folliculitis.** Note the pustules localized to the hair follicles of the trunk and proximal extremity. (Photo contributor: Jeffrey S. Gibson, MD.)

Management and Disposition

The folliculitis usually involutes in 1 to 2 weeks without treatment; however, oral ciprofloxacin or topical gentamicin may decrease recovery time. In addition, the implicated source of exposure must be decontaminated to avoid reexposure. Patients with immunosuppression, widespread lesions, or concern for systemic involvement should be treated with oral ciprofloxacin.

Pearls

1. Ensure patient is aware of the cause and insist on decontamination of the water source.
2. Hot tub folliculitis may also result from contact with depilatory agents.



FIGURE 13.80 ■ **Hot Tub Folliculitis.** Folliculitis from Pseudomonas aeruginosa in a hot tub. (Photo contributor: Richard P. Usatine, MD. Used with permission. From Usatine RP, Smith MA, Mayeaux EJ, Chumley HS. The Color Atlas of Family Medicine. 2nd ed. McGraw-Hill; 2013: Fig. 117-1.)

ECTHYMA GANGRENOSUM

Clinical Summary

Ecthyma gangrenosum is a cutaneous manifestation of P aeruginosa septicemia occurring in an immunocompromised or neutropenic patient. Patients have a toxic appearance with fever, abnormal vital signs, and altered consciousness. The initially erythematous macules develop bullae or pustules surrounded by violaceous halos. Bullae become hemorrhagic and rupture (pustules breakdown), becoming necrotic ulcers with central black or gray eschars. The time course from macule to eschar can occur within 12 to 24 hours. The lesions primarily affect the anogenital areas (over 50%) but may appear anywhere on the body. Ecthyma gangrenosum is seen in up to 13% of patients with P aeruginosa septicemia and rarely in nonbacteremic patients.

Management and Disposition

Ecthyma gangrenosum is associated with life-threatening P aeruginosa sepsis and a high mortality. Appropriate supportive care measures and rapid initiation of antipseudomonal antibiotics are required. Dermatology consultation for diagnosis confirmation and infectious disease consultation for antibiotic guidance are helpful. Patients with oncologists should consult the oncology service and follow oncology protocols.

Pearls

1. Patients with ecthyma gangrenosum are severely immunosuppressed and many other bacteria and fungi can cause similar lesions. Discuss additional antibiotics and antifungal medications with the oncology and infectious disease services.
2. Without examining the entire patient, these lesions can be completely missed.
3. Multiple lesions, delayed diagnosis, and delayed institution of appropriate antibiotics portend a poor prognosis.



FIGURE 13.82 ■ Pseudomonas aeruginosa **Infected Ulcerations.** Note the green colored crusting. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.81 ■ **Ecthyma Gangrenosum.** A typical hemorrhagic bulla of ecthyma gangrenosum secondary to pseudomonal sepsis. (Photo contributor: James Mensching, DO.)

Clinical Summary

Primary or recurrent HSV infection presents as grouped vesicles on an erythematous base. Recurrent HSV is typically less severe than the primary infection. A prodrome is frequently noted with fever, malaise, anorexia, and regional lymphadenopathy. The vesicles progress to pustules and crusted erosions. They heal over 2 to 3 weeks. Mucocutaneous involvement of the mouth and lips are the most common sites. Herpetic whitlow is a painful HSV infection of a distal finger seen primarily in children and adolescents. Herpes gladiatorum spreads via direct skin-to-skin contact in sports such as rugby and wrestling.

Management and Disposition

Oral antivirals in addition to analgesics and antipyretics are useful. To be most effective, antivirals should be started within 72 hours of the eruption or during the typical prodrome. Immunocompromised patients may require admission and IV antivirals. Dermatology consultation is recommended for complicated presentations.

Pearls

1. Wear protective gloves; herpetic infections are an occupational hazard in the medical and dental professions.
2. Wrestlers (or any skin-to-skin contact sport) with vesicles and ulcers may not participate in organized sports until completely healed.
3. Recurrent, same-site HSV infections on the fingers should alert the clinician to consider herpetic whitlow. These infections are often misdiagnosed as cellulitis, blistering dactylitis, and paronychia.

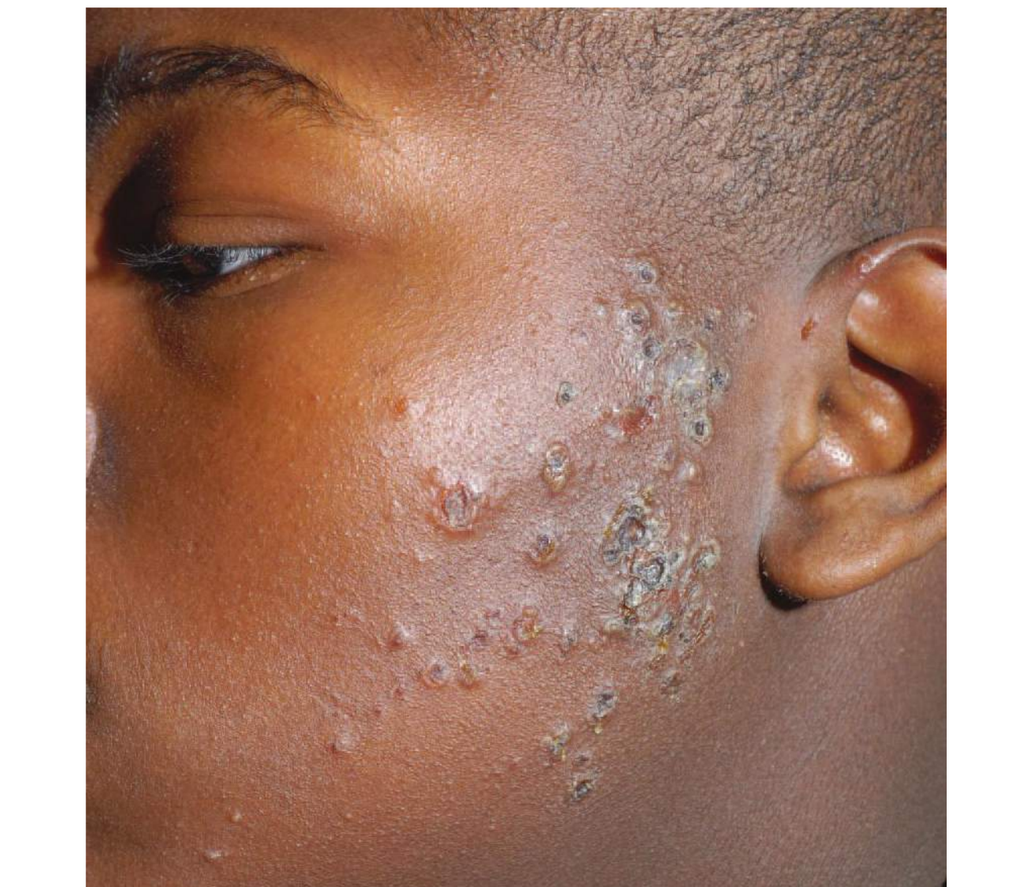


FIGURE 13.84 ■ **Herpetic Whitlow.** Vesicles on an erythematous base. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 13.83 ■ **Herpetic Whitlow.** Painful, grouped, confluent vesicles and an erythematous base on the distal finger. (Photo contributor: Selim Suner, MD, MS.)

FIGURE 13.85 ■ **Herpes Gladiatorum.** Primary herpes gladiatorum in a wrestler. Note the varied stages of lesions, from vesicular to dried crusts. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Herpes zoster is a dermatomal, unilateral reactivation of the varicella zoster virus. All ages, including infants, can be affected. The eruption may occur anywhere on the body but most commonly on the face and trunk. Pruritus, pain, tenderness, and dysesthesias may present 4 to 5 days prior to an eruption composed of umbilicated, grouped vesicles on an erythematous, edematous base. The vesicles may become purulent or hemorrhagic. Occasionally, nerve involvement may occur without cutaneous involvement. Rare presentations involve multiple dermatomes or cross midline. Ophthalmic zoster (see related item) involves the nasociliary branch of the fifth cranial nerve and presents with vesicles on the nose and cornea (Hutchinson sign). Ramsay Hunt syndrome (see related item) is a herpes zoster infection of the geniculate ganglion with tinnitus, decreased hearing, facial palsy, and vesicles on the tympanic membrane, pinna, and ear canal.

Management and Disposition

Uncomplicated cases of herpes zoster can be managed with supportive care, especially pain control. However, acyclovir, famciclovir, or valacyclovir hastens the healing, decreases the pain, and decreases postherpetic neuralgia incidence and symptoms, if started within 72 hours of vesicle appearance (still helpful even after 7 days). Gabapentin decreases postherpetic neuralgia pain.

Admission to the hospital for IV acyclovir is usually reserved for complicated cases involving multiple dermatomal distributions, involvement of the ophthalmic branch of the trigeminal nerve, disseminated disease, or immunocompromised patients. Herpes zoster keratitis requires emergent ophthalmologic consultation to avoid any potential scarring or vision loss.

Pearls

1. The nonimmune or immunocompromised should avoid lesional contact from the prodrome until reepithelialization, since the crusts can contain the varicella zoster virus.
2. Herpes zoster during pregnancy confers no risk to the mother or unborn baby (as opposed to primary varicella virus infection).
3. Postherpetic neuralgia affects 10% to 20% of patients and is more common with advancing age, family history, and immunosuppression.
4. Immunocompromised patients can have unusual presentations with diffuse cutaneous and visceral involvement.



FIGURE 13.86 ■ **Herpes Zoster.** Dermatomal crusted erosions within an erythematous patch. (Photo contributor: Selim Suner, MD, MS.)



FIGURE 13.87 ■ **Herpes Zoster.** Dermatomal distribution of vesicles and crusted erosions. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)

DERMATOPHYTE INFECTIONS

Clinical Summary

Tinea corporis includes all dermatophyte infections excluding the scalp, face, hands, feet, and groin. The dermatophytosis is pruritic and consists of a well-circumscribed scaly plaque with a slightly elevated border and central clearing. This annular configuration is most commonly found on the trunk and neck. Skin scrapings examined with a KOH preparation demonstrate hyphae.

Tinea faciale (dermatophyte infection of the facial skin) commonly appears as a well-circumscribed scaling and erythematous patch. Tinea manuum (hands) presents with long-term scaling of the palms. Tinea cruris, or "jock itch," is a pruritic dermatophytosis of the intertriginous areas, usually, but not always, sparing the penis and scrotum. The scaly, erythematous plaque spreads peripherally with well-defined borders.

Tinea pedis, or "athlete's foot," consists of erythema and scaling of the sole and interdigital spaces, frequently with maceration, vesiculation, and fissure formation. The toenails may also be affected (tinea unguium).

Tinea capitis (scalp) presents as a pruritic, erythematous, scaly plaque. This may develop into a delayed-type hypersensitivity reaction, where the initial erythematous, scaly plaque becomes boggy with inflamed, purulent nodules and plaques (kerion). The hair follicle is frequently destroyed by the inflammatory process in a kerion, leading to a scarring alopecia.



FIGURE 13.89 ■ **Tinea Faciale.** Note the sharply marginated, annular plaques with central clearing. KOH preparation is positive. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)



FIGURE 13.88 ■ **Tinea Corporis, Ringworm.** A well-defined, annular, pruritic plaque with a raised, scaly border and central clearing. KOH preparation is positive. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)

Management and Disposition

Systemic antifungals are required to treat tinea capitis and tinea unguium infections. Due to the long-term treatment requirement and associated side effects, referral to a dermatologist is recommended. Topical antifungal medications are used for tinea corporis, tinea faciale, tinea cruris, and tinea pedis infections. Rarely, systemic antifungals may be required.

Pearls

1. The scale is usually located at the leading edge of erythema and provides the best yield for positive scrapings seen on KOH examination (see related item for slide preparation).
2. Macerated areas may become secondarily infected by bacteria. Discuss the importance of good wound care.
3. When using topical antifungals, it is important to treat for one to two weeks beyond the point of clinical cure to ensure success.



FIGURE 13.90 ■ **Tinea Manuum and Tinea Pedis.** The involvement of both the hands and feet are common. (Photo contributor: James J. Nordlund, MD.)



FIGURE 13.91 ■ **Tinea Cruris.** Erythematous plaque with accentuated and well-defined border. The scale may not be appreciated in this anatomic site. (Photo contributor: James J. Nordlund, MD.)



FIGURE 13.92 ■ **Tinea Pedis.** The interdigital spaces are characteristically involved. (Photo contributor: James J. Nordlund, MD.)



FIGURE 13.93 ■ **Tinea Capitis.** Multiple, well-defined, scaly plaques on the occiput. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.94 ■ **Kerion.** Occipital boggy swelling with hair loss consistent with kerion. (Photo contributor: Anne W. Lucky, MD.)

TINEA (PITYRIASIS) VERSICOLOR

Clinical Summary

Tinea versicolor, or pityriasis versicolor, is a chronic, superficial fungal infection that involves the trunk and extremities with rare facial involvement. The fungus is part of normal skin flora. Finely scaling brown macules are present in fair-skinned patients, whereas scaly hypopigmented macules are often noted in dark-skinned patients.

Management and Disposition

Treatment consists of short applications of selenium sulfide lotion, topical antifungal creams, or topical ketoconazole. Resistant cases require referral and consideration of oral antifungal.

Pearls

1. Tinea versicolor is more common in adolescents and young adults.
2. Clinically active areas or areas colonized with the fungus may be identified by orange fluorescence noted on the Wood lamp examination.
3. Normal pigmentation may take months to return.



FIGURE 13.95 ■ **Tinea Versicolor.** Multiple, small-to-medium-sized, well-demarcated hypopigmented macules on the back of a tanned individual with white skin. (Used with permission from Wolff K, Johnson RA, Saavedra AP. Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology. 7th ed. McGraw-Hill; 2013: Fig. 26-19.)



FIGURE 13.96 ■ **Tinea Versicolor.** An example of hypopigmented areas on dark skin. (Photo contributor: James J. Nordlund, MD.)

Clinical Summary

Onychomycosis is an invasion of the nails by any fungus. Four clinical subtypes are described. Distal subungual presents as discolorations of the free edge of the nail with hyperkeratosis leading to subungual accumulation of friable keratinaceous debris. White superficial consists of sharply outlined white areas on the nail plate which leave the surface friable. Proximal subungual presents as discolorations which start proximally at the nail fold. Candidal onychomycosis encompasses the entire nail plate, leaving the surface rough and friable.

Management and Disposition

Oral antifungals are required to eradicate the fungus. Refer to a dermatologist or primary care physician.

Pearls

1. All that causes the nail plate to separate from the nail bed is not necessarily fungus. Psoriasis, lichen planus, and various other nail dystrophies, such as distal onycholysis (caused by excessive water exposure or drugs) must be differentiated from this fungal infection.
2. Treatment requires proper monitoring and long-term follow-up.



FIGURE 13.97 ■ **Onychomycosis.** Invasion of the nail bed by fungus. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 13.98 ■ **Onychomycosis.** Note that multiple nail beds have been invaded by the fungus, leading to chronic hyperkeratosis and subungual accumulation of friable keratinaceous debris. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)



FIGURE 13.99 ■ **Onychomycosis.** Multiple nail beds with dystrophy and yellow subungual debris. (Photo contributor: J. Matthew Hardin, MD.)

INTERTRIGO

Clinical Summary

Intertrigo is a dermatitis occurring on opposed surfaces of skin, such as the creases of the neck, folds of the groin and armpit, or a panniculus. It is characterized by a tender, red patch or plaque with a moist, macerated surface. The juxtaposed skin surfaces create a chronic friction and this can easily become suprainfected with candida, fungal, or bacterial infections.

Management and Disposition

Local care, empiric topical antifungal treatment, and good personal hygiene are recommended.

Pearls

1. The nature of the intertriginous areas make them a high risk for secondary infection.
2. Potent topical corticosteroids should be avoided because of the high atrophy risk.



FIGURE 13.100 ■ **Intertrigo of the Panniculus.** In this dramatic case, the weight of the pannus creates constant friction on the abdominal wall. This leads to erythema and tissue breakdown. This patient also had fever, suggesting secondary infection. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Human scabies is caused by Sarcoptes scabiei var. hominis, a mite that lives within the layers of the epidermis. Transmission of scabies occurs after direct skin contact with an infected individual or possibly from infested clothing and bedding. The female mite burrows into the individual's skin and deposits two to three eggs daily. Fecal pellets (scybala) are also deposited in the burrow and may be responsible for the localized pruritus. Nocturnal pruritus is characteristic of scabies. The pink-white, slightly elevated burrows are typically found in the web spaces of the hands and feet, penis, buttocks, scrotum, and extensor surfaces of the elbows and knees. Crusted, or Norwegian, scabies seen in immunosuppressed or debilitated patients (see HIV chapter) present with asymptomatic acral crusting, but can occur anywhere.

Management and Disposition

Topical 5% permethrin cream is commonly used to treat scabies. Apply from the neck to the toes overnight (8-12 hours) and then wash off. Repeat treatment in 7 days. Bedding, towels, and clothing should be washed in hot water and dried on high heat (an alternative is to place items into a plastic bag for 10 days).

Pearls

1. Intimate contacts and all family members in the same household should be treated.
2. Patients often experience "postscabietic itch" which can last for 2 to 4 weeks. This is not a new infestation but rather an immunologic reaction to the dead mite. Confirm completion of an appropriate treatment.



FIGURE 13.102 ■ **Scabies.** Burrows and erosions in the web space of the hand. (Photo contributor: David Effron, MD.)



FIGURE 13.101 ■ **Scabies.** Burrows and erosions from itching on the feet of a patient with scabies. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 13.103 ■ **Scabies.** Extensive burrows and erosions in an infant. (Photo contributor: David Effron, MD.)

JAUNDICE

Clinical Summary

Jaundice is a light yellowing of the skin, mucous membranes, and sclera; it is generally detectable when bilirubin levels are about 3.0 mg/dL. Many patients may not be aware of the faint yellowing and present with seemingly unrelated symptoms. Be aware that up to 50% of patients with jaundice will have pruritus. The most important diagnoses to rule out are: hemolytic anemias, viral hepatitis, chronic alcohol abuse, autoimmune hepatitis, medications, primary biliary cirrhosis, primary sclerosing cholangitis, cholelithiasis, surgical strictures, and obstructive malignancies. Acetaminophen, penicillins, and oral contraceptives are some of the more common medications associated with jaundice.

Management and Disposition

As the etiology of jaundice is broad, a thorough history focusing on associated symptoms (fever, pruritus, vomiting, hematochezia, melena, and abdominal pain), previous surgical procedures, and medication history (including over-the-counter medications) is essential. Physical findings of fever, abdominal tenderness, and hepatomegaly should be sought. Workup of jaundiced patients should include white blood cell count and differential, liver function tests including bilirubin levels, hepatitis viral screening, and imaging studies.

Pearls

1. Patients who consume large amounts of β-carotene (found in squash and carrots) may have mild yellowing of their skin (especially palms and soles) but will lack scleral icterus or elevations in bilirubin.
2. Women starting oral contraceptives may experience cholestasis in the first few months that may cause jaundice.



FIGURE 13.105 ■ **Jaundice, Scleral Icterus.** Yellowing of the sclera in a patient with liver disease. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 13.104 ■ **Jaundice.** Mild palmar jaundice in a dark-skinned patient. (Photo contributor: Kevin J. Knoop, MD, MS.)

Clinical Summary

Melasma is commonly seen on the face of young adult females. It consists of symmetric, well-defined, light to dark brown patches. The most common sites are on the malar cheek, lateral forehead, upper cutaneous lip, and mandible. Factors associated with accentuation of melasma include sunlight exposure, pregnancy (often called "the mask of pregnancy"), and oral contraceptives.

Management and Disposition

Most patients will be concerned about accentuation of their previously imperceptible melasma. Rule out pregnancy in new onset or worsening melasma. Essential to any treatment of melasma is strict sun avoidance. Refer patients to a dermatologist.

Pearls

1. Melasma is common in young females with darker skin types but all races can be afflicted.
2. Sun exposure on other parts of the body can cause accentuation of facial melasma.
3. Contrasted with the hyperpigmentation of melasma, Addison disease is diffuse hyperpigmentation with accentuation in sun-exposed areas.



FIGURE 13.106 ■ **Melasma.** Well-demarcated, hyperpigmented patch seen on the cheek. (Photo contributor: J. Matthew Hardin, MD.)

ABDOMINAL STRIAE (STRIAE ATROPHICAE)

Clinical Summary

Abdominal striae are linear, depressed, pink or bluish scar-like lesions that may later become silver or white. They are caused by weakening of the elastic cutaneous tissues from chronic stretching. They most commonly occur on the abdomen but are also seen on the buttocks, breasts, axilla, and thighs. Striae are commonly seen in obesity, pregnancy, rapid growth associated with puberty, Cushing syndrome, and chronic topical corticosteroid treatment.

Management and Disposition

This finding seldom presents as a condition requiring acute treatment; thus, attention is directed to determining and treating the underlying cause.

Pearls

1. Recent striae with moon facies, hypertension, renal calculi, osteoporosis, and psychiatric disorders are suggestive of Cushing syndrome.
2. The striae color (red/purple) caused by pregnancy typically fades with time, unlike striae associated with Cushing syndrome.
3. Inappropriate use of topical corticosteroids can result in permanent striae.

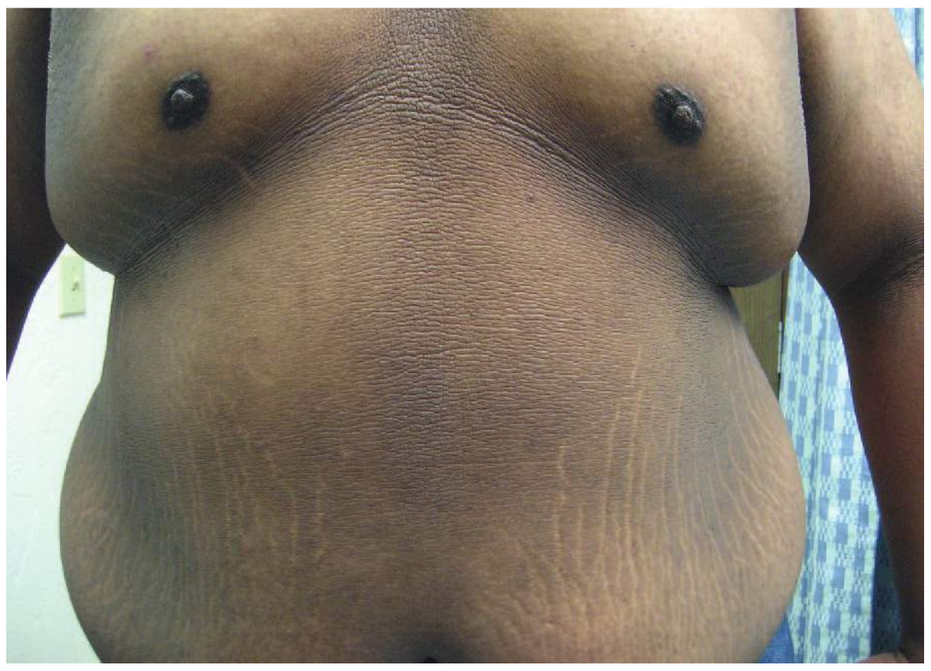


FIGURE 13.108 ■ **Abdominal Striae.** Obese patient with lower lateral wall striae. Also noted is thickened, hyperpigmented, abdominal skin, typical of acanthosis nigricans (associated with diabetes). (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.107 ■ **Abdominal Striae.** These striae are seen in a patient with recent weight gain, moon facies, and altered mental status. The patient was diagnosed with Cushing syndrome.

Clinical Summary

Vitiligo is an acquired loss of pigmentation that commonly involves the backs of the hands, face, and body folds. There is a positive family history in 30% of the cases. Initially the disease is limited, but it then slowly progresses over years. Vitiligo is secondary to absence of epidermal melanocytes, which may be due to an autoimmune phenomenon against melanocytes. Approximately half of these cases begin in patients less than 20 years of age.

Management and Disposition

Refer patients to a dermatologist for further workup and long-term treatment.

Pearls

1. Vitiligo can occur at sites of trauma (Koebner phenomenon).
2. Wood lamp examination helps identify hypopigmented areas in patients with light complexions.
3. Tinea versicolor, in contrast, has a scale and positive KOH preparation.



FIGURE 13.109 ■ **Vitiligo.** Well-defined, hypopigmented areas characteristic of vitiligo. (Photo contributor: James J. Nordlund, MD.)



FIGURE 13.110 ■ **Vitiligo.** Almost complete depigmentation of the hands. (Used with permission from Fauci AS, Braunwald E, Kasper DL, et al. Harrison's Principles of Internal Medicine. 17th ed. New York, NY: McGraw-Hill; 2008: 312.)

UREMIC FROST

Clinical Summary

Uremic frost is a classic manifestation of chronic renal failure; it is rarely seen today. It develops as a result of accumulation of urea in sweat. In advanced uremia, the accumulation of urea in sweat may reach such a critical level that, upon its evaporation, a fine white powder is left on the skin surface. Associated hyperkalemia may also be present owing to concurrent renal failure.

Management and Disposition

Treatment of the underlying condition that resulted in the patient's uremia may prevent further accumulation of uremic frost. Typically, urgent dialysis is indicated.

Pearls

1. Although rare today, this condition may be seen in noncompliant patients or environmental stressors, including inadequate air conditioning.
2. For patients presenting with altered mental status, attention to the airway, oxygenation, and rapid assessment and treatment of associated metabolic disorders, such as hyperkalemia, is paramount.



FIGURE 13.111 ■ **Uremic Frost.** Note the fine white powder on the skin of this patient with end-stage renal disease. (Photo contributor: Alan B. Storrow, MD.)

Clinical Summary

Autoimmune bullous diseases are uncommon but have dramatic presentations. Bullous pemphigoid (BP) results from autoantibodies to the epidermal basement membrane and creates tense bullae, frequently located on the proximal extremities. In pemphigus vulgaris (PV), the autoantibodies are directed against the epidermal keratinocytes. This results in flaccid bullae (more superficial bullae that easily slough off to form erosions). PV delicate bullae and subsequent erosions commonly present in the pharynx, scalp, and trunk. Paraneoplastic pemphigus presents with severe oral ulcerations (similar to SJS/TEN; see related item) and resolves with treatment of the associated malignancy.

Management and Disposition

Considering an autoimmune bullous disease in the differential is the first step. Admission with early dermatologic consultation for histologic and immunofluorescent studies should be considered. Systemic corticosteroids and immunosuppressant therapy are required for control. Patients with significant BSA involvement should be treated in a burn unit.

Pearls

1. Oral erosions and ulcerations should always raise the suspicion of autoimmune bullous diseases.
2. The high morbidity and mortality of this disease is now significantly lower due to modern steroid-sparing immunosuppressants and wound care.
3. Always consider medication history and signs of underlying malignancies when evaluating a patient with extensive bullae or erosions.



FIGURE 13.112 ■ **Bullous Pemphigoid.** Tense blister formation among confluent erosions and plaques. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 13.113 ■ **Bullous Pemphigoid.** Tense bullae on in the distal extremities are a common presentation of BP. (Photo contributor: Selim Suner, MD, MS.)



FIGURE 13.114 ■ **Pemphigus Vulgaris.** Multiple flaccid bullae now de-roofed and at risk for secondary infection. (Photo contributor: J. Matthew Hardin, MD.)

SYSTEMIC LUPUS ERYTHEMATOSUS

Clinical Summary

Systemic lupus erythematosus (SLE) has four cutaneous manifestations: malar rash, discoid rash, photosensitivity, and oral ulcers. Eighty percent of patients have skin findings at some point of the disease. The malar "butterfly" rash presents with erythema and mild edema over the bridge of the nose and malar cheeks. Similar erythematous patches may be seen on the ears, neck, and chest. Discoid lesions can appear at any site but are usually found above the neck, including the scalp. They are characterized as annular, erythematous macules, or plaques that eventually become atrophic and scarred.

Management and Disposition

Urgent referral to a dermatologist or rheumatologist is essential for early diagnosis and prevention of other systemic manifestations.

Pearls

1. Consider any suspicious rash on the face a manifestation of SLE and refer to a dermatologist early.
2. The malar rash resolves without scarring, whereas discoid lesions (primarily on the face, ears, and scalp) result in permanent scarring.
3. Not all photoexacerbated rashes are sunburns.



FIGURE 13.115 ■ **Butterfly Rash of SLE.** Erythematous malar rash with slight edema and minimal scaling ("butterfly pattern"). (Used with permission from Wolff K, Johnson RA, Suurmond D. Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology. 5th ed. New York, NY: McGraw-Hill; 2005: 385.)



FIGURE 13.116 ■ **Butterfly Rash of SLE.** The appearance of the malar "butterfly" rash in a dark-skinned individual. Note mild scale on the cheeks and frontal thinning hairline. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 13.117 ■ **Discoid Lupus.** Well-defined scaly patches with atrophy. (Photo contributor: Kristin L. Stevens, MD.)

Clinical Summary

Sweet syndrome, also known as acute febrile neutrophilic dermatosis, is characterized by fever, peripheral neutrophilia, and a nonvasculitic neutrophilic cutaneous eruption. Sweet syndrome can occur at any age but most commonly from 30 to 60 years old. Typically, the lesions are tender, well-demarcated erythematous plaques with an edematous periphery (pseudovesiculation). Some progress to ulcerations and hemorrhagic crusting. Lesions can occur anywhere on the body, but most frequently on the upper extremities, neck, and face. Variants have been isolated to the face (erysipelas-like) and the dorsal hands. The plaques generally cause a burning pain and are nonpruritic.

Sweet syndrome is associated with preceding upper respiratory infections, vaccinations, medications, malignancies, inflammatory bowel disease, autoimmune connective tissue diseases, and pregnancy.

Management and Disposition

The associated diseases need to be considered and appropriate consultation is warranted. A dermatologist should be consulted for diagnosis confirmation and further evaluation. Management includes prednisone tapered over 4 to 6 weeks.

Pearls

1. Unless a specific infection is identified (Streptococcus, Yersinia, or Staphylococcus), antibiotics do not improve Sweet syndrome.
2. Malignancies associated with Sweet syndrome account for 20%; acute myeloid leukemia is the most common hematologic malignancy.
3. Although preceding infections can cause this rash, the lesions are not infectious.



FIGURE 13.119 ■ **Sweet Syndrome.** Less obvious erythematous plaques on the foot. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.118 ■ **Sweet Syndrome.** This young woman complained of sudden onset of fever and painful skin lesions. Her WBC was 22,000 with neutrophilia. This constellation of symptoms suggests Sweet syndrome, and was confirmed by histology. There is a hint of peripheral vesiculation on some lesions. (Photo contributor: Lawrence B. Stack, MD.)

PORPHYRIA CUTANEA TARDA

Clinical Summary

Porphyrias are associated with enzymatic defects in heme biosynthesis. Porphyria cutanea tarda (PCT) is the most common type. Patients have photosensitivity, skin fragility, and characteristic lesions on sun-exposed sites (most common on the dorsal hands and forearms). Typically, the skin is easily traumatized with blisters, erosions, superficial scars, milia, and hypertrichosis. PCT does not cause the life-threatening neurologic attacks associated with the acute porphyrias.

PCT may be induced by ethanol, estrogens, oral contraceptives, infections (Hepatitis C and HIV), polychlorinated hydrocarbons, dialysis, and iron overload. Pseudoporphyria, clinically indistinguishable from PCT, is associated with dialysis and certain medications (NSAIDs, furosemide, hydrochlorothiazides, and amiodarone, among others).

Management and Disposition

Laboratory examination may begin in the ED with blood chemistries, porphyrin studies (24-hour urinary porphyrins can be ordered), and referral to a dermatologist. Long-term treatment includes phlebotomy and antimalarials (which can induce fatal hepatotoxicity in PCT patients). Discontinue any medications that might initiate PCT or pseudoporphyria.

Pearls

1. Consider PCT in a patient with fragile skin with erosions and scarring, but only on sun-exposed skin.
2. PCT does not have acute attacks of abdominal pain, neurologic deficits, psychosis, or autonomic dysfunction. Variegate porphyria, common in South Africans with Dutch ancestry, does present with the typical skin lesions and acute attacks.
3. Examination of the urine with a Wood lamp may reveal orange-red fluorescence.
4. Always consider medications (both prescribed and over-the-counter medications) when considering PCT.



FIGURE 13.120 ■ **Porphyria Cutanea Tarda.** Blisters and erosions of PCT. (Photo contributor: Selim Suner, MD, MS.)



FIGURE 13.121 ■ **Porphyria Cutanea Tarda.** The easily traumatized skin and erosions of PCT. (Photo contributor: Alan B. Storrow, MD.)

SQUAMOUS CELL CARCINOMA

Clinical Summary

Squamous cell carcinoma (SCC) is the second most common skin cancer. It is associated with a higher incidence in males, increased age, chronic sun exposure, immunosuppressive treatment, and chronic burns or scars. Initially, SCC presents with erythematous macules that develop into firm papules and plaques. Most are located on the sun-exposed sites of the head, neck, and upper extremities.

Management and Disposition

After ensuring a secondary infection is not present, prompt outpatient dermatologic referral is indicated.

Pearls

1. There is a higher risk of metastasis with SCC versus basal cell carcinoma (although still very low). As with basal cell carcinoma, metastatic potential is higher on the ears, periocular area, nose, and lips—do not miss the opportunity to refer patients with questionable lesions!
2. Any persistent nodule, plaque, or ulcer should be examined for SCC.

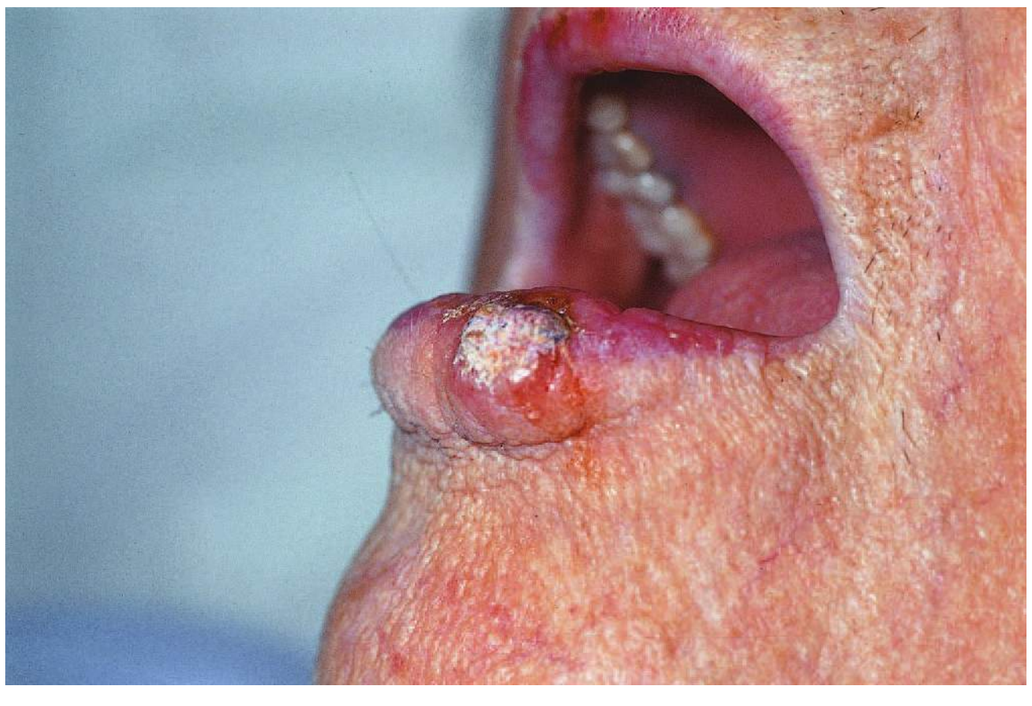


FIGURE 13.122 ■ **Squamous Cell Carcinoma.** The lower lip is exposed to more sunlight and is therefore involved more frequently. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.124 ■ **Squamous Cell Carcinoma.** This nodule has a central keratogenous core. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.123 ■ **Squamous Cell Carcinoma.** This nodule with central ulceration slowly developed over 1 year. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.125 ■ **Squamous Cell Carcinoma.** Ulcerated nodule on the forehead. (Photo contributor: J. Matthew Hardin, MD.)

BASAL CELL CARCINOMA

Clinical Summary

Basal cell carcinoma (BCC) is the most common nonmelanoma skin cancer. BCC can present anywhere but is most common on sun-exposed areas. The typical lesion begins as a pearly papule with telangiectasias (nodular BCC). The lesion may ulcerate and bleed. Other forms of BCC include superficial BCC (pink, scaly plaque with pearly border), pigmented BCC (appears as a nodular or superficial BCC with dark brown to black center), and morpheaform BCC (appears as a rapidly expanding scar).

Management and Disposition

After ensuring a secondary infection is not present, prompt outpatient dermatologic referral is indicated.

Pearls

1. The metastatic potential of BCC is very low (0.1%) but higher on the ears, periocular area, nose and lips. Do not miss the opportunity to refer patients with questionable lesions.
2. BCC is not rare in darker skinned persons (a common misperception).



FIGURE 13.126 ■ **Basal Cell Carcinoma.** Nodular basal cell carcinoma consists of a firm, centrally ulcerated (rodent ulcer) nodule with a raised, pearly, telangiectatic border. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)



FIGURE 13.127 ■ **Pigmented Basal Cell Carcinoma.** Translucent, brownish-black, flat papule, easily confused with melanoma. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)



FIGURE 13.128 ■ **Basal Cell Carcinoma.** A superficial basal cell carcinoma is frequently disregarded. Note the flat, erythematous, scaly plaque with its elevated, irregular border. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)



FIGURE 13.129 ■ **Basal Cell Carcinoma.** This chronic, erythematous papule and crusted ulcer appeared 2 years prior to presentation. (Photo contributor: J. Matthew Hardin, MD.)

Clinical Summary

Melanoma is a potentially fatal cutaneous tumor derived from epidermal melanocytes. Any age can be affected, but the peak incidence is 20- to 45-year-old patients (much younger than basal cell or SCCs). The most significant risk factor is a primary relative with melanoma. Evaluation of any pigmented lesion should include the ABCDE rule (A for asymmetry, B for irregular borders, C for color variegation, D for diameter greater than 6 mm, and E for elevation and evolving). Any lesion with these characteristics is considered suspicious for melanoma.

Management and Disposition

Prompt outpatient dermatologic referral is indicated. Simply acknowledging suspicious lesions seen during emergency care may encourage earlier follow-up.

Pearls

1. The palms, soles, and subungual sites are most common with dark-skinned individuals.
2. Melanoma can occur in sites not exposed to the sun (genitalia/buttocks). In the course of an exam, note worrisome lesions and refer.
3. Any growing pigmented or nonpigmented lesion should be referred to dermatology.
4. Most patients will not have new moles after 35 years old. A new mole in this setting should be evaluated by dermatology.



FIGURE 13.131 ■ **Nodular Melanoma.** This melanoma has progressed to an exophytic tumor, which was deeply invasive histopathologically. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brooke Army Medical Center, San Antonio, TX.)



FIGURE 13.130 ■ **Melanoma.** This lesion demonstrates asymmetry, color variegation, and a diameter greater than 6 mm. (Photo contributor: J. Matthew Hardin, MD.)



FIGURE 13.132 ■ **Melanoma.** Slow-growing, pigmented plaque on the arch of the foot. (Photo contributor: J. Matthew Hardin, MD.)

LIP LICKER'S DERMATITIS

Clinical Summary

Lip licker's dermatitis is caused by repeated exposure of the vermillion border and the cutaneous lips to saliva. Children and young adults are more commonly affected but older adults, including the elderly, can be afflicted. Irritants in topical lip preparations can initiate this process or can worsen a presentation. Winter is the most common time to see lip licker's dermatitis due to the constant exposure to cold air and low humidity but presentations occur throughout the year.

Lip licker's dermatitis typically starts with red papules that slowly progress into an erythematous, thin plaque. As the cycle of licking continues, the area becomes confluent circumferentially around the vermillion border and the cutaneous lips. The skin begins to break down with fissuring and scaling and the area can become very painful. The rash does not extend beyond the tongue's reach on the cutaneous lips.

Management and Disposition

Treatment is focused on identifying and preventing repetitive lip licking. Many children are not aware that they are contributing to this rash and care should be taken to not blame them for this predicament (especially younger children). Use of 100% petrolatum (Vaseline) applied all day (any time the area becomes dry or the patient desires to lick) will help. Stop the use of any other topical products. The addition of 1% hydrocortisone ointment may hasten the resolution.

Pearls

1. Never approach a young child blaming them for their condition; it is best to use indirect questioning to make them aware of the cause ("Do you think you ever lick your lips?").
2. The use of over-the-counter lip preparations or flavored lip products may cause or exacerbate the condition.
3. Lip licker's dermatitis never ends until the lip licking behavior ends!



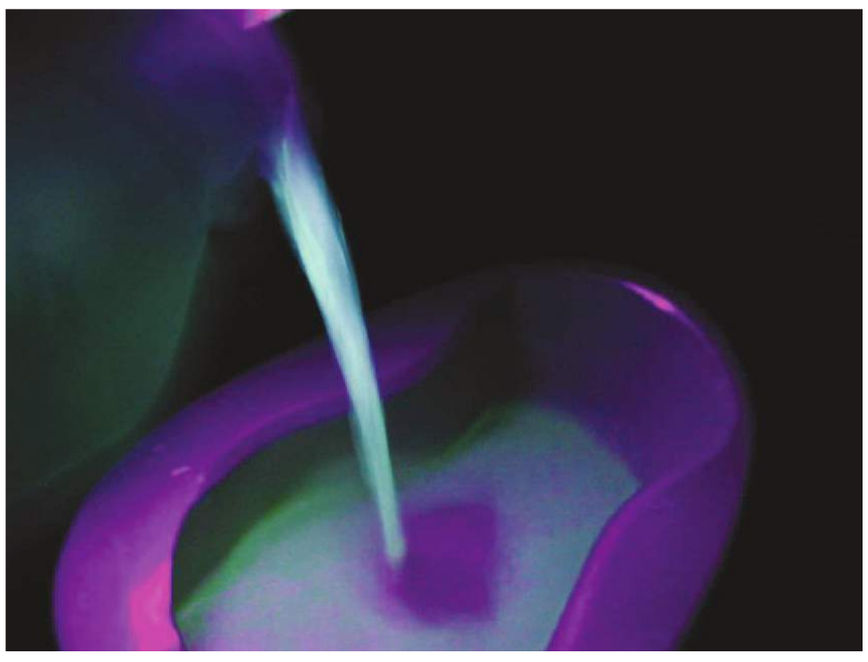
FIGURE 13.133 ■ **Lip Licker's Dermatitis.** Circumoral dryness and pain. (Photo contributor Aubrey Mowery, MPH, CHES.)

PART 2

Specialty Areas







Chapter 14

PEDIATRIC CONDITIONS

Brad Sobolewski
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Fifth Disease. Toddler with the classic slapped-cheek appearance of fifth disease caused by parvovirus B19. Note the lacy reticular macular rash on the shoulder and upper extremity. (Photo contributor: Anne W. Lucky, MD.)

ERYTHEMA TOXICUM NEONATORUM

Clinical Summary

Erythema toxicum neonatorum is a benign, self-limited vesicopustular lesion of unknown etiology that occurs in up to 70% of term newborns. It is characterized by discrete, small, erythematous macules or patches up to 2 to 3 cm in diameter with 1 to 3 mm firm pale yellow or white papules or pustules in the center. The trunk and proximal extremities are predominantly involved. This rash usually appears within the first 24 to 72 hours of life but may be present at birth. The distinctive feature of erythema toxicum is its evanescence, with each individual lesion usually disappearing within 5 to 7 days. New lesions may occur in a waxing and waning fashion. The diagnosis is usually made based on the clinical appearance of the rash in an otherwise well-appearing neonate without any systemic signs of illness. Wright-stained slide preparations of a scraping from the center of the lesion demonstrate numerous eosinophils. Cultures from these lesions will be negative. The differential diagnosis includes neonatal acne, transient neonatal pustular melanosis, newborn milia, miliaria, infantile acropustulosis, neonatal herpes simplex, bacterial folliculitis, candidiasis, and impetigo of the newborn.

Management and Disposition

No specific therapy is indicated in the setting of a well-appearing newborn with normal activity and appetite. Parents should be educated and reassured about the evanescence of the rash. In cases where impetigo, Candida, or herpes infections are suspected, a smear from the center of the lesion and bacterial and viral cultures may be necessary to make a final diagnosis.

Pearls

1. Erythema toxicum is the most common newborn rash.
2. The lesions may present anywhere on the body but tend to spare the palms and soles.
3. Laboratory evaluation is unnecessary.



FIGURE 14.1 ■ **Erythema Toxicum.** Newborn infant with diffuse macular rash of erythema toxicum. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 14.2 ■ **Erythema Toxicum.** Close-up of lower extremity of a neonate with erythema toxicum. (Photo contributor: Robert W. Hickey, MD.)

Clinical Summary

Nevus simplex (salmon patch) is the most common vascular lesion in infancy, present in about 40% to 60% of newborns. It appears as a blanching, slightly pink-red macule or patch most commonly on the nape of the neck, the glabella, mid-forehead, or upper eyelids. Lesions generally fade over the first 2 years of life but may become more prominent with crying or straining.

Management and Disposition

Parental education and reassurance can be helpful, but no immediate treatment is indicated. Pulsed dye laser may be considered for persistent lesions that are cosmetically undesirable.

Pearls

1. Salmon patches are composed of ectatic dermal capillaries.
2. Salmon patches appear symmetrically and cross the midline in contrast to the unilateral distribution of a port-wine stain.
3. When seen on the nape of the neck, this lesion is referred to as a stork bite or as an angel's kiss when appearing on the forehead.
4. About 5% of those appearing at the nape of the neck will remain permanently or recur.
5. Obtain imaging to evaluate for spinal dysraphism in patients with a lumbosacral nevus simplex and another lumbosacral abnormality (dermal sinus or pit, patch of hypertrichosis, or deviated gluteal cleft).



FIGURE 14.4 ■ **Salmon Patches.** Child with patch over lower back consistent with salmon patches. (Photo contributor: Anne W. Lucky, MD.)



FIGURE 14.3 ■ **Salmon Patches.** Newborn with characteristic salmon patches over his face. (Photo contributor: Anne W. Lucky, MD.)



FIGURE 14.5 ■ **Salmon Patches.** Characteristic lesion on the nape of the neck is commonly called a stork bite. (Photo contributors: Katharine Hanlon and Kara Shah, MD, PhD.)

NEONATAL JAUNDICE

Clinical Summary

Neonatal jaundice is the yellowing of the skin, sclerae, and/or mucous membranes caused by bilirubin deposition. It occurs when total serum bilirubin is in excess of 5 mg/dL and progresses in a head-to-toe fashion as levels increase. Most cases of physiologic (<12 mg/dL) jaundice are self-limited without sequelae, and appear on the second or third day of life peaking between the third and fifth day. Preterm infants may peak later.

The increased bilirubin production in newborns is a result of turnover of fetal red blood cells, a temporary decrease in conjugation and clearance by the immature newborn liver, and increased enterohepatic circulation. Risk factors for indirect hyperbilirubinemia include maternal diabetes, prematurity, drugs, polycythemia, traumatic delivery with cutaneous bruising or hematoma, breastfeeding, and ABO (O mother and A/B infant) or Rh(D) incompatibility (Rh(D) negative mother and Rh(D) positive infant). Most infants with jaundice have no "disease" per se, but a careful history and organized approach is necessary to identify potentially pathologic causes. Kernicterus manifests in a multitude of irreversible neurologic abnormalities and is the long-term result of bilirubin-induced neurologic dysfunction secondary to extreme unconjugated hyperbilirubinemia which leads to neuronal death and pigment deposition in the basal ganglia and cerebellum.

Management and Disposition

Initial laboratory workup should include blood type, Coombs test, complete blood count (CBC) with smear for red cell morphology, reticulocyte count, and indirect and direct bilirubin levels. Although transcutaneous bilirubin measurement devices are widespread, they can underestimate total bilirubin at levels >15 mg/dL; therefore, a serum measurement is recommended for severely jaundiced newborns. Initial management should ensure adequate hydration and treatment of the underlying condition. The level of serum bilirubin at which to start phototherapy can be obtained from a standardized nomogram and is dependent upon the infant's postnatal age in hours, gestational age, and an assessment of risk factors. In general, the goal of phototherapy is to maintain the bilirubin level below 20 mg/dL. Exchange transfusion is considered if the serum level remains elevated (22-25 mg/dL) despite appropriate phototherapy.

Pearls

1. Onset of clinical jaundice in the first 24 hours of life strongly suggests the presence of a pathologic process.
2. Direct serum bilirubin concentration exceeding 10% of total serum bilirubin or 2 mg/dL suggests hepatobiliary disease or a metabolic disorder.
3. Bilirubin levels at which to initiate phototherapy or exchange transfusion should be modified for prematurity, sepsis, low birth weight, and other risk factors.



FIGURE 14.6 ■ **Neonatal Jaundice.** Newborn with yellowish hue to skin consistent with jaundice. (Photo contributor: Kevin J. Knoop, MD, MS.)

NEONATAL MILK PRODUCTION (WITCH'S MILK)

Clinical Summary

Neonatal galactorrhea occurs in up to 6% of term newborns and is usually secondary to transplacental transfer of maternal estrogen. These hormonal effects (maternal estrogens and endogenous prolactin) lead to palpable breast buds in approximately one-third of all term newborns. Males and females are equally affected. In most cases, the breast enlargement and galactorrhea begin to subside after the second week of life in males and 2 to 6 months in females. Infants with neonatal breast hypertrophy may be predisposed to infections (mastitis or abscess) possibly incited by repetitive manipulation of the enlarged breast bud by a caregiver. The differential diagnosis includes early mastitis with purulent nipple discharge.

Management and Disposition

Treatment is not necessary unless infection is suspected. Parents can be reassured that this is a normal finding, and follow-up to resolution should occur at routine well child care visits.

Pearls

1. Classical presentation includes the presence of clear colostrum-like secretions in newborns with hypertrophied mammary tissue without erythema or tenderness. Persistence of enlarged breast buds beyond 6 months of age should prompt follow-up with a pediatric endocrinologist.
2. In an older child, galactorrhea may be the presenting sign of hypothyroidism or pathologically elevated prolactin levels.



FIGURE 14.7 ■ **Witch's Milk.** Milky fluid draining from the nipple in a newborn. (Photo contributor: Michael J. Nowicki, MD.)

NEONATAL MASTITIS

Clinical Summary

Neonatal mastitis is an infection of the breast tissue that occurs in full-term neonates with a peak incidence in the third week of life. Females are affected more often than males in a 2:1 distribution. Clinically, it manifests as swelling, induration, erythema, warmth, and tenderness of the affected breast. The ipsilateral axillary lymph nodes may be swollen. Approximately two-thirds have palpable fluctuance. In some cases, purulent discharge may be expressed from the nipple. Fever may be present in 25% of affected patients. Other systemic symptoms (irritability, decreased appetite, and vomiting) are less common but indicate a more severe infection if present. Bacteremia is rare. Staphylococcus aureus is the most common pathogen, causing 75% to 85% of cases. Rarely, gram-negative organisms or group B or D Streptococcus are the cause. If treatment is delayed, mastitis may progress rapidly with involvement of subcutaneous tissues and subsequent toxicity. In the initial stages, neonatal mastitis may mimic mammary tissue hypertrophy owing to maternal passive hormonal stimulation. Minor trauma, cutaneous infections, and duct blockage may precede this infection.

Management and Disposition

Immediate treatment is important to avoid cellulitic spread and breast tissue damage. In cases of mild cellulitis with no associated fluctuance in an otherwise well-appearing, afebrile neonate, culture of nipple discharge (if present) and oral antibiotics (antistaphylococcal penicillin or first-generation cephalosporin) with close outpatient follow-up are sufficient. Adjustment of coverage can be made once results of cultures or Gram stain are available, especially in the presence of gram-negative bacilli. In cases involving systemic signs of infection, rapid subcutaneous spread, or toxic appearance, a complete sepsis workup should be performed followed by hospitalization. If no organism is seen on Gram stain, a parenteral antistaphylococcal penicillin plus an aminoglycoside or cefotaxime alone should be used. In cases of palpable fluctuance, prompt surgical consultation should be obtained to assess the need for needle aspiration or incision and drainage. Conservative treatment with intravenous (IV) antibiotics often results in resolution of the fluctuance without surgical intervention. Recovery is usually within 5 to 7 days.

Pearls

1. Antibiotic choice should include coverage for S aureus.
2. Maintain a low threshold for initiating a sepsis workup and IV antibiotics.
3. Delays in the diagnosis and treatment may lead to distortion of the nipple, impairment of the secretory capacity of the breast, and reduction in the size of the adult breast.



FIGURE 14.8 ■ **Neonatal Mastitis.** Neonate with left-sided breast swelling, erythema, and purulent discharge. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 14.9 ■ **Neonatal Mastitis.** Neonate with marked swelling, erythema, and purulent discharge. (Photo contributor: Emergency Medicine Department, Naval Medical Center, Portsmouth, VA.)

Clinical Summary

An umbilical granuloma is granulation tissue with incomplete epithelialization that persists following cord separation. It is the most common cause of an umbilical mass in neonates. Parents will describe a persistent discharge from the umbilicus after the cord has dried and separated. It appears soft, pink, wet, and friable. Infants with an umbilical granuloma do not have localized swelling, redness, warmth, tenderness, or fever. An umbilical polyp is a rare anomaly resulting from the persistence of the omphalomesenteric duct or the urachus and may have a similar appearance. A polyp is usually firm with a mucoid secretion. The differential diagnosis also includes omphalitis, an infection of the umbilicus and surrounding structures, which should be considered in ill-appearing neonates.

Management and Disposition

Advise parents to keep the granuloma dry and exposed to the air as often as possible. Cleaning and drying of the umbilical cord base with alcohol is not necessary and may irritate the skin and delay healing. Cauterization of the granuloma by application of topical silver nitrate is the treatment of choice. It is important to protect the surrounding skin (apply petroleum jelly or antibiotic ointment) and remove excess silver nitrate to avoid chemical burns and skin staining. The cauterization may need to be repeated at 3-day intervals if drainage persists.

Pearls

1. An umbilical granuloma is the most common umbilical mass in neonates.
2. The only sign of granuloma formation may be the presence of nonpurulent discharge noted in the diaper area that is in contact with the umbilicus.
3. Omphalitis presents with redness of the periumbilical area typically tracking upward in the midline and often with a purulent discharge from the umbilicus. It can progress to abdominal wall cellulitis or peritonitis and requires a complete sepsis workup and hospital admission for treatment with broad-spectrum parenteral antibiotics.



FIGURE 14.10 ■ **Umbilical Granuloma.** Newborn infant with umbilical granuloma visible in umbilicus. (Photo contributor: Anne W. Lucky, MD.)



FIGURE 14.11 ■ **Omphalomesenteric Duct.** This red mass resembling a granuloma was found to be an omphalomesenteric duct. (Photo contributor: Kevin J. Knoop, MD, MS.)

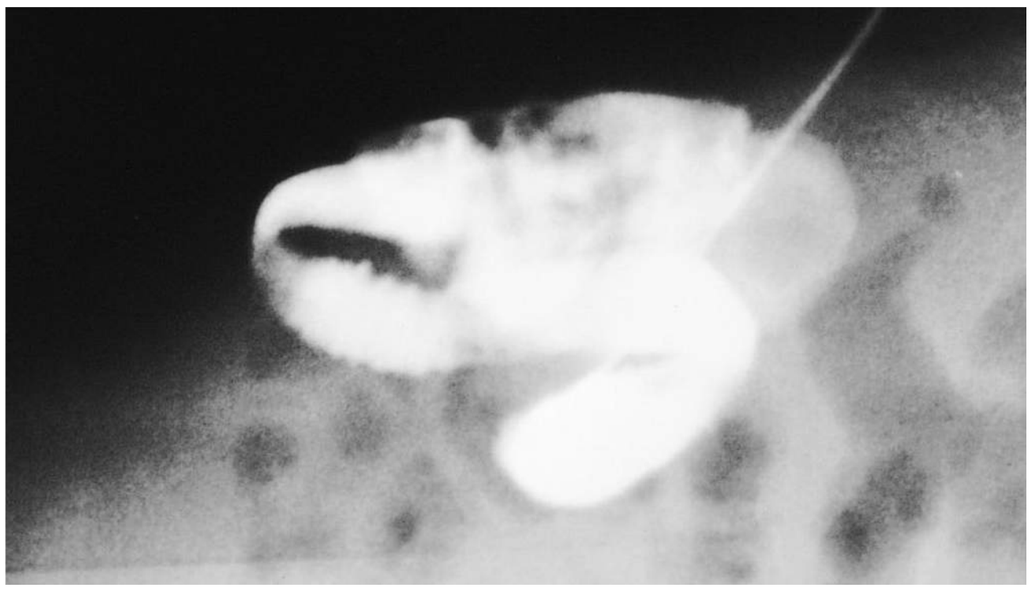


FIGURE 14.12 ■ **Omphalomesenteric Duct.** A fistulogram confirms continuity between the duct and intestine. (Photo contributor: Kevin J. Knoop, MD, MS.)

HYPERTROPHIC PYLORIC STENOSIS

Clinical Summary

Hypertrophic pyloric stenosis (HPS) is characterized by progressive postprandial, nonbilious vomiting that steadily increases in frequency and amount due to hypertrophy of the pyloric musculature and edema of the pyloric canal, producing gastric outlet obstruction. It is usually diagnosed in infants from birth to 5 months, most commonly at 2 to 8 weeks of life. The vomiting may become forceful and is then described as projectile (although this pattern is not always present). There is a familial tendency, and white males (especially firstborn) are more frequently affected. During the physical examination, peristaltic waves may be observed traveling from the left upper to right upper quadrants. The hypertrophy of the antral and pyloric musculature produces the "olive" to palpation (best palpated in the epigastrium or right upper quadrant after emesis or emptying the stomach with a nasogastric tube). As a result of persistent vomiting, hypochloremic, hypokalemic metabolic alkalosis with varying degrees of dehydration, and failure to thrive may occur when the diagnosis is not made early in the course.

The finding of a pyloric olive on palpation of the abdomen is pathognomonic. Ultrasound is a useful tool to confirm the diagnosis when the olive is not evident on examination or early in the course. The differential diagnosis includes intestinal obstruction or atresia, malrotation with volvulus, hiatal hernia, gastroenteritis, adrenogenital syndrome, increased intracranial pressure, esophagitis, sepsis, gastroesophageal reflux, and poor feeding technique.

Management and Disposition

Treatment includes correction of electrolyte imbalances and dehydration, as well as emergent surgical consultation for curative Ramstedt pyloromyotomy. Failure to correct metabolic alkalosis prior to surgery can increase the risk of postoperative apnea. Patients may benefit from a nasogastric tube on low intermittent suction.

Pearls

1. HPS is the most common cause of metabolic alkalosis in infancy.
2. Serial examinations and observation of the child after oral fluid challenges for persistent projectile vomiting may aid in making the diagnosis.
3. Clinical manifestations of pyloric stenosis begin at a mean age of 3 weeks after birth.
4. Pyloric ultrasonography is the diagnostic study of choice.
5. Less than 2% of infants with HPS have bilious vomiting.

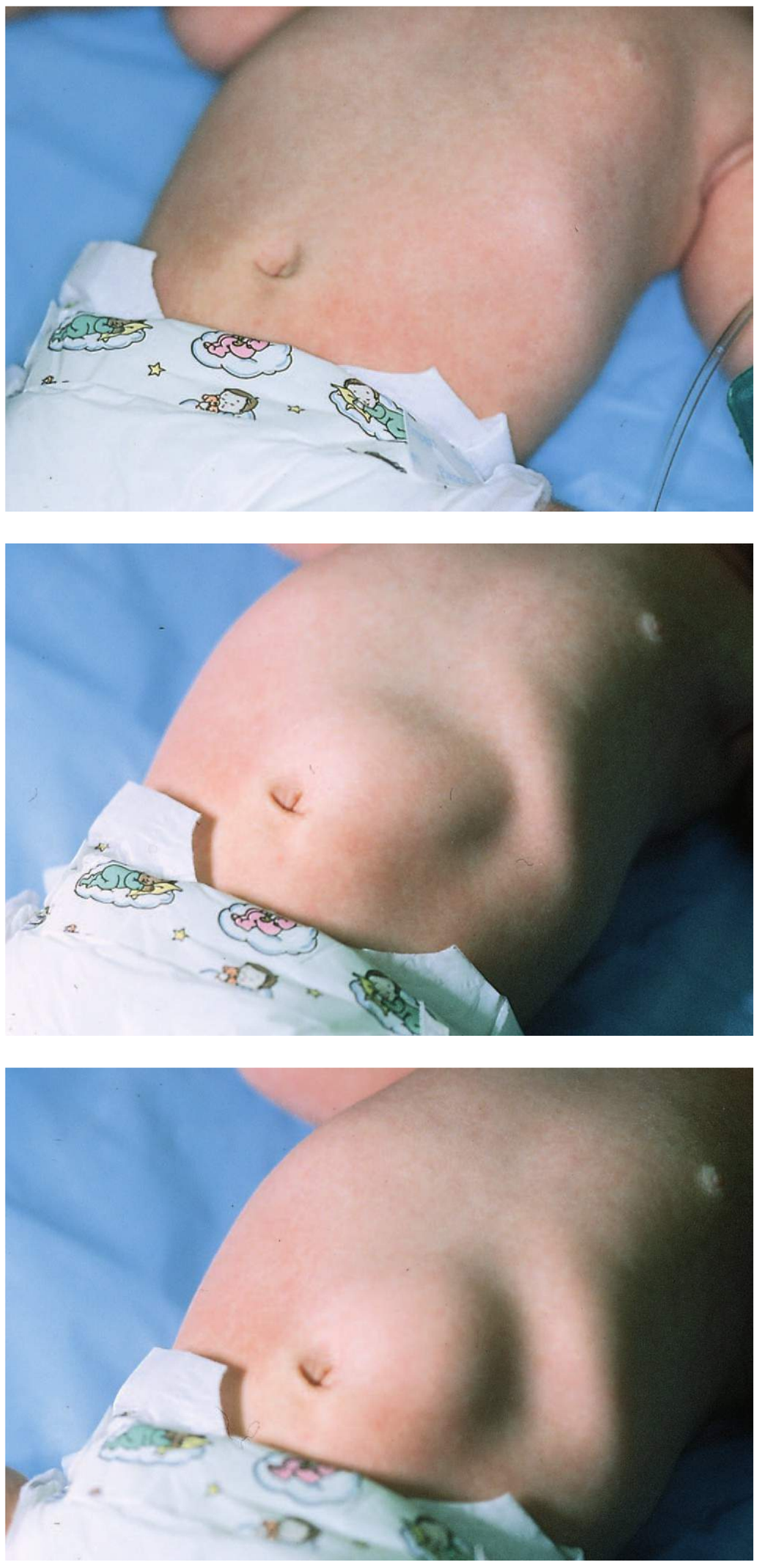


FIGURE 14.13 ■ **Gastric Wave of Hypertrophic Pyloric Stenosis.** A gastric wave can be seen traversing the abdomen in this series of photographs of a patient with HPS. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 14.14 ■ **Ultrasound Target Sign in HPS.** This transverse ultrasound shows redundant, infolded mucosa (arrowheads) between muscular components (arrows), referred to as the "target sign." (Photo contributor: Lawrence B. Stack, MD.)

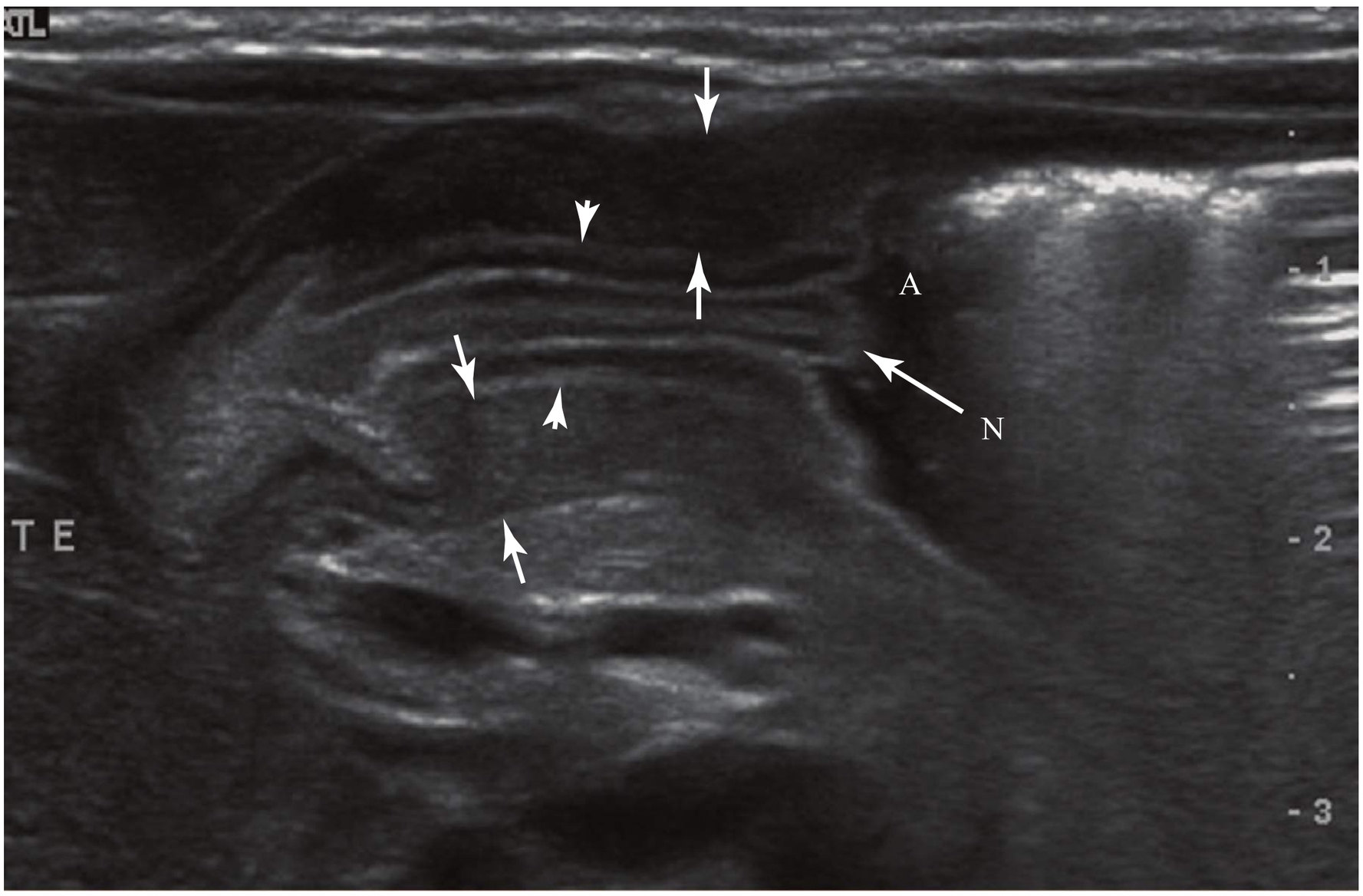


FIGURE 14.15 ■ **Antral Nipple Sign in HPS.** This longitudinal sonogram shows two-layered, thickened mucosa (arrowheads) surrounded by muscular components (arrows). Redundant pyloric mucosa protrudes into the fluid in the gastric antrum (A), forming the "antral nipple sign" (N) in HPS. (Photo contributor: Lawrence B. Stack, MD.)

INTESTINAL MALROTATION WITH VOLVULUS

Clinical Summary

Intestinal malrotation is primarily a condition of infancy but can be seen in older children and adults. During the fourth week of embryonic development, the primary intestinal loop bulges into the yolk sac, rotates 270 degrees counterclockwise, and returns to the enlarged abdominal cavity between the eight and tenth week where it is fixed via mesentery that extends from the ligament of Treitz to the ileocecal valve in the right lower quadrant. In malrotation, the duodenum, jejunum, and cecum are partially rotated with the bowel anchored by an abnormally thin band of mesentery. The bowel can twist on this thin mesentery, which contains the superior mesenteric artery, leading to acute intestinal obstruction and midgut vascular compromise known as volvulus. Also, the abnormally positioned cecum now rests in the upper abdomen fixed to the right lateral abdominal wall by bands of peritoneum (Ladd bands) that cross and can obstruct the duodenum.

Presentation is with vomiting in 50% of cases. The vomiting may not be bilious, but it is classically described as such. Patients are often irritable with significant abdominal tenderness. Third space fluid losses increase as gut ischemia progresses. Differential diagnosis includes pyloric stenosis, intestinal atresias, appendicitis, necrotizing enterocolitis, and intussusception.

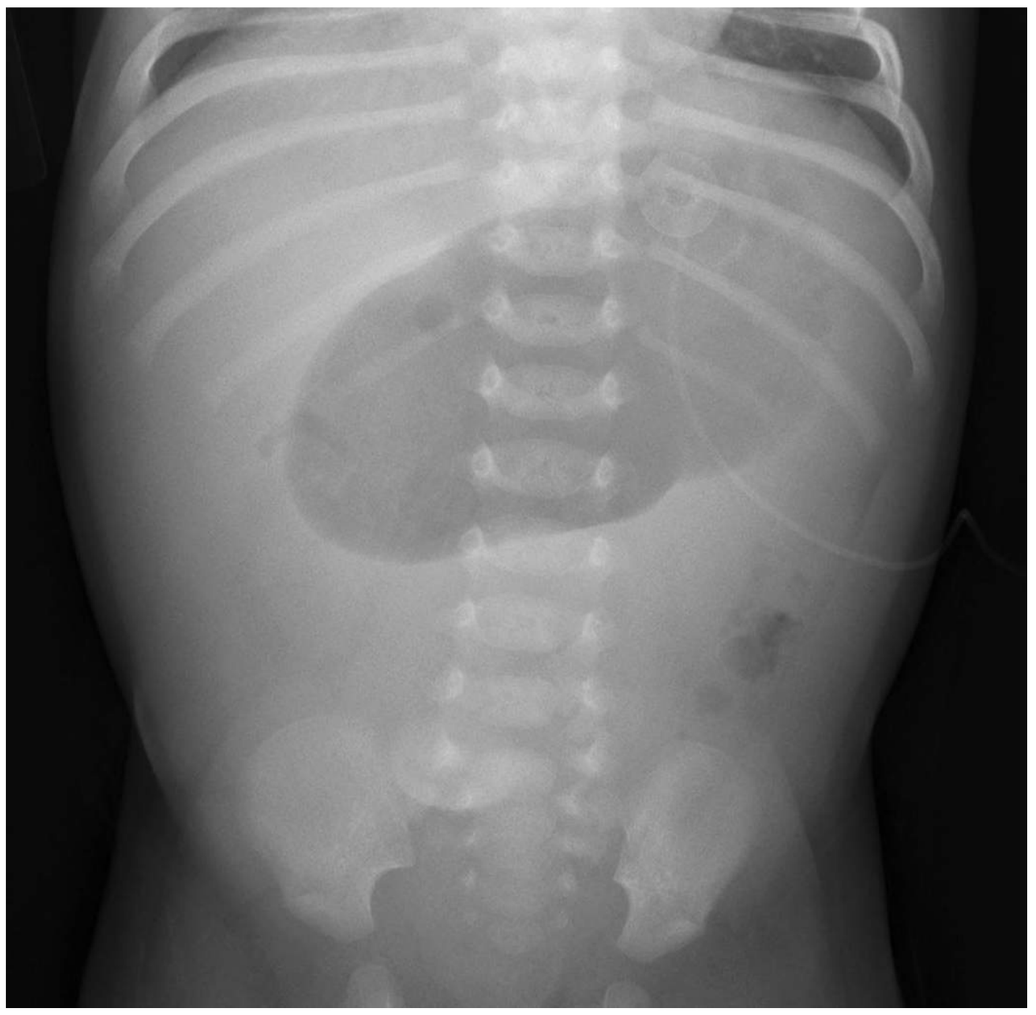


FIGURE 14.16 ■ **Intestinal Malrotation with Volvulus.** Antero-posterior x-ray of the abdomen in a newborn with bilious emesis. Dilated, air-filled stomach with a relative paucity of gas in the distal bowel. (Photo contributor: Alexander Towbin, MD.)

Management and Disposition

Emergent surgical consultation is indicated. Plain x-rays are rarely diagnostic, but may show signs of small bowel obstruction. An upper gastrointestinal (GI) series is the most helpful study, though one-quarter of cases may have an equivocal result. Preoperative management consists of resuscitation with IV fluids, placing a gastric tube for decompression, and administration of broad-spectrum antibiotics.

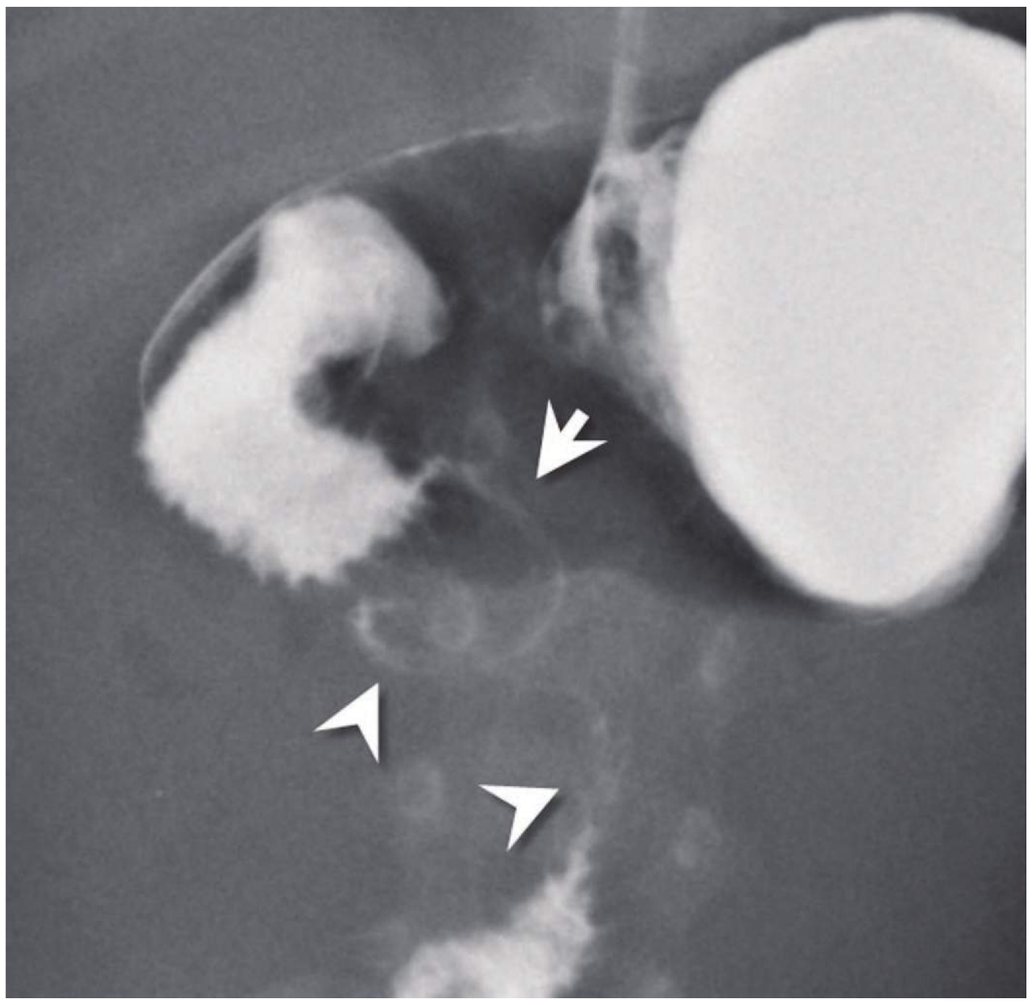


FIGURE 14.17 ■ **Intestinal Malrotation with Volvulus.** Antero-posterior radiograph from an upper GI series shows malrotation with midgut volvulus. The duodenum (arrowheads) does not cross midline and does not extend superiorly to the level of the pylorus. The spiraling downward appearance is consistent with volvulus. (Photo contributor: Alexander Towbin, MD.)

Pearls

1. Fifty percent of patients with malrotation present with volvulus in the first month of life.
2. Over 30% of cases of malrotation with volvulus present beyond early childhood usually with an insidious onset with abdominal pain as the most common symptom.
3. The imaging test of choice is the upper GI series, but computed tomography (CT) should be considered in ill-appearing patients in which administration of enteral contrast may be difficult.

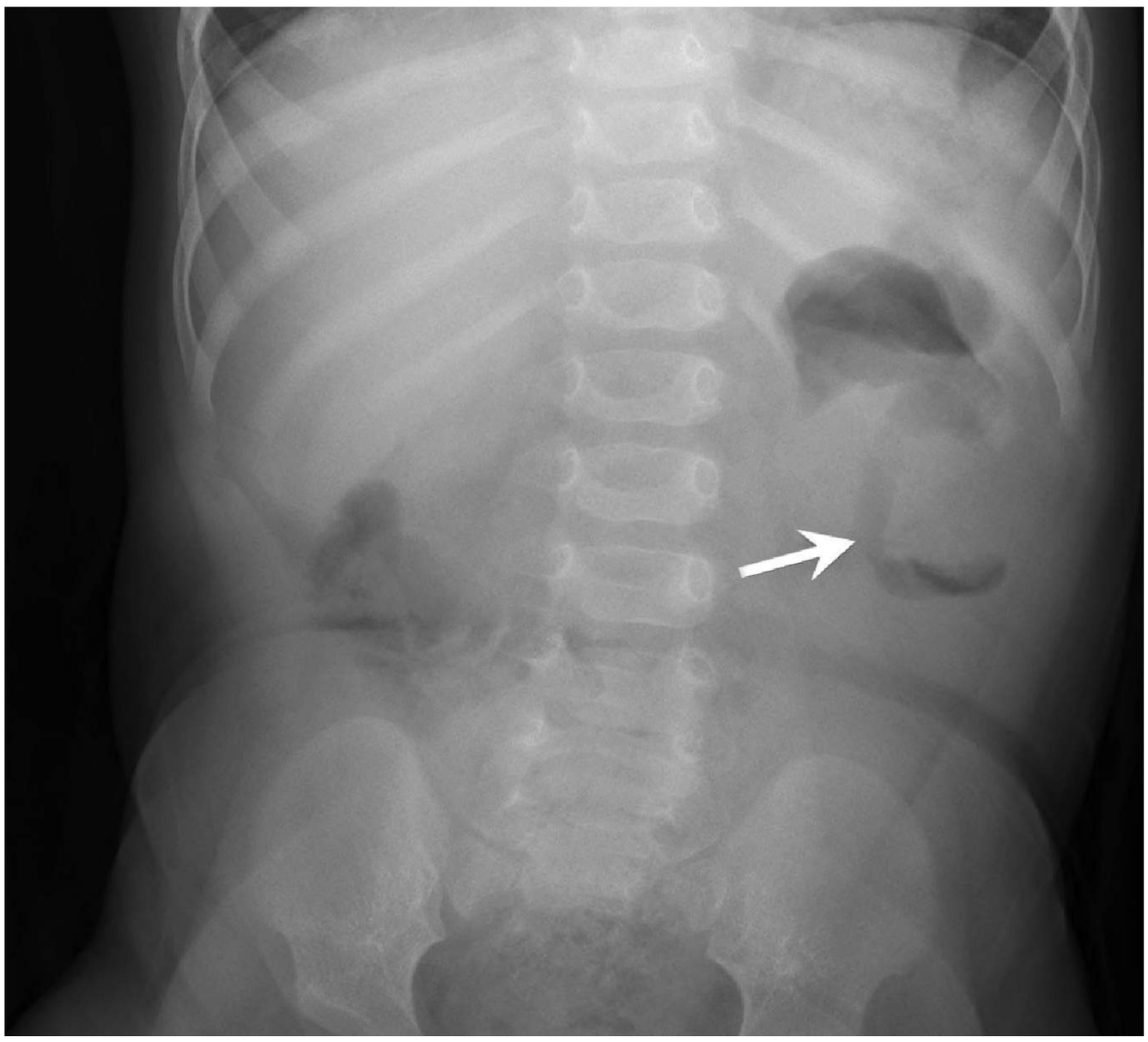


FIGURE 14.18 ■ **Intestinal Malrotation with Volvulus.** Soft tissue mass (arrow) surrounded by air within the left upper quadrant near the hepatic flexure. (Photo contributor: Alexander Towbin, MD.)

ERYTHEMA INFECTIOSUM (FIFTH DISEASE)

Clinical Summary

Erythema infectiosum is a viral infection caused by parvovirus B19, presenting most commonly between 4 and 10 years of age. It begins with nonspecific prodromal symptoms including malaise, coryza, headache, fever, nausea, and diarrhea. Two to 5 days into the illness, the classic rash appears characterized initially by the "slapped cheeks" appearance of a bright red malar macular rash that spares nasal ridge and perioral areas. A reticulated, lacy erythematous maculopapular eruption with central clearing then appears on the extensor surfaces of extremities. The differential diagnosis includes other morbilliform eruptions such as measles, rubella, roseola, and infectious mononucleosis. Bacterial infections (eg, scarlet fever), drug reactions, and other skin conditions such as guttate psoriasis, papular urticaria, atopic dermatitis, and erythema multiforme are also included in the differential.

Management and Disposition

Treatment is aimed at symptomatic relief. Parents can be reassured that this exanthem is benign and self-limited. Once the rash appears, the patient is no longer contagious. It is important to educate the patient and family about the possible risk of parvovirus B19 as a cause of hydrops fetalis or fetal deaths early in pregnancy. It can also cause an aplastic crisis in patients with hematologic conditions such as sickle cell disease, hereditary spherocytosis, and various hemolytic anemias, or in the immunocompromised.

Pearls

1. Recrudescence of the lacy, reticular rash may occur with exercise, overheating, emotional upset, or sun exposure as a result of cutaneous vasodilatation.
2. Parvovirus B19 is the most common cause of hydrops fetalis. Pregnant mothers of children diagnosed with erythema infectiosum should have their serologic status determined.
3. In young adults, parvovirus B19 can cause papular purpuric gloves and socks syndrome.



FIGURE 14.19 ■ **Fifth Disease.** Toddler with the classic slapped-cheek appearance of fifth disease caused by parvovirus B19. Note the lacy reticular macular rash on the shoulder and upper extremity. (Photo contributor: Anne W. Lucky, MD.)

ROSEOLA INFANTUM (EXTHANTHEM SUBITUM)

Clinical Summary

The typical presentation of roseola infantum involves a prodrome characterized by a 3- to 5-day history of high fever often exceeding 40°C (104°F) in a child 6 months to 3 years of age. The child may be fussy, but is often otherwise well-appearing. After the child's fever abates, the typical exanthem appears characterized by blanching erythematous macules and papules on the trunk, neck, proximal extremities, and occasionally the face. The rash fades within 2 to 4 days, but may only last several hours. The causative agent in 90% of cases is human herpesvirus 6 (HHV-6). The differential diagnosis includes viruses such as measles, rubella, parvovirus B19, or infectious mononucleosis. Bacterial infections (eg, scarlet fever), drug reactions, and other skin conditions such as guttate psoriasis, papular urticaria, and erythema multiforme are also included in the differential.

Management and Disposition

As with most viral infections, only supportive therapy is necessary. Special attention should be paid in maintaining fluid intake, fever control for the patient's comfort, and parental education about the benign, self-limited nature of this illness.

Pearls

1. With defervescence and appearance of the rash, the patient is no longer contagious.
2. The most frequent complication of roseola is febrile seizures.



FIGURE 14.20 ■ **Roseola Infantum (Exanthem Subitum).** Toddler with maculopapular eruption of roseola. (Photo contributor: Raymond C. Baker, MD.)

IMPETIGO

Clinical Summary

Impetigo is a contagious bacterial infection of the superficial skin that is caused by Streptococcus pyogenes (group A β-hemolytic Streptococcus) and S aureus. It most commonly affects children 2 to 5 years of age and usually involves the face and extremities. It begins as small vesicles or pustules with very thin roofs that rupture easily with the release of a cloudy fluid and subsequent formation of honey-colored crusts. Variants include bullous impetigo and the ulcerative form. The lesions may spread rapidly by autoinoculation secondary to scratching and coalesce to form larger areas of infection. The differential diagnosis includes second-degree burns, varicella, herpes simplex infections, nummular dermatitis, superinfected eczema, and scabies.

Management and Disposition

These lesions are highly contagious and spread by direct contact. Hand washing and personal hygiene should be emphasized to the patient and family. Application of topical antibacterials, such as mupirocin ointment, has proven to be as effective as oral antibiotics if there are a limited number of lesions and no bullae. Over-the-counter triple antibiotic ointments (bacitracin-neomycin-polymyxin B) may not be as effective. If lesions are extensive and/or bullous, oral antibiotic coverage is indicated.



FIGURE 14.22 ■ **Impetigo.** Chest wall and axillary erosions oozing discharge forming honey-colored crusts. (Photo contributor: Lawrence B. Stack, MD.)

Effective oral agents include first-generation cephalosporins, dicloxacillin, amoxicillin with clavulanic acid, or clindamycin.

Pearls

1. Inflicted cigarette burns may resemble the lesions of impetigo.
2. Poststreptococcal glomerulonephritis and rheumatic fever can be complications of impetigo caused by group A β-hemolytic Streptococcus.



FIGURE 14.21 ■ **Impetigo.** Infant with perioral erosions oozing discharge forming honey-colored crusts. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 14.23 ■ **Bullous Impetigo.** A child with impetiginous lesions on the face. Note the formation of bullae. (Photo contributor Anne W. Lucky, MD.)



FIGURE 14.24 ■ **Bullous Impetigo.** Bullae with honey-colored exudate in a neonate with fever. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Measles presents as an acute febrile illness with a 3- to 4-day prodromal period characterized initially by fever, malaise, and anorexia, followed closely by conjunctivitis, coryza, and cough. Koplik spots, the pathognomonic enanthem of measles, appear as 1- to 3-mm red papules with gray-white centers on the buccal mucosa. They usually present approximately 48 hours before the development of the characteristic erythematous blanching maculopapular rash. This rash appears during the third or fourth day of the illness as dark red to purple macules and papules on the forehead, around the hairline, behind the earlobes, subsequently spreading in a cephalocaudad progression. Individual lesions often become confluent. The lesions tend to fade in the same order that they appear.

Most cases recover without complications; others may develop otitis media, croup, pneumonia, encephalitis, myocarditis/pericarditis, keratitis, and rarely subacute sclerosing panencephalitis (SSPE), a very late complication. The differential diagnosis of the characteristic rash is vast and includes exanthem subitum, rubella, infections caused by echovirus, coxsackie and adenoviruses, toxoplasmosis, infectious mononucleosis, scarlet fever, Kawasaki disease, drug reactions, Rocky Mountain spotted fever, and meningococcemia.

Management and Disposition

Supportive therapy includes bed rest, antipyretics, and adequate fluid intake. Complications should be treated accordingly. Currently available antivirals are not effective. Postexposure prophylaxis includes administration of the measles-mumps-rubella (MMR) vaccine within 72 hours of exposure to a patient with active measles. Passive immunization with intravenous immune globulin (IVIG) is effective for prevention and attenuation of measles if given within 6 days of the initial exposure especially in pregnant women and the immunocompromised. During outbreaks, MMR vaccine can be given to infants younger than 12 months. However, such infants require an additional two doses of MMR at the recommended ages after their first birthday.

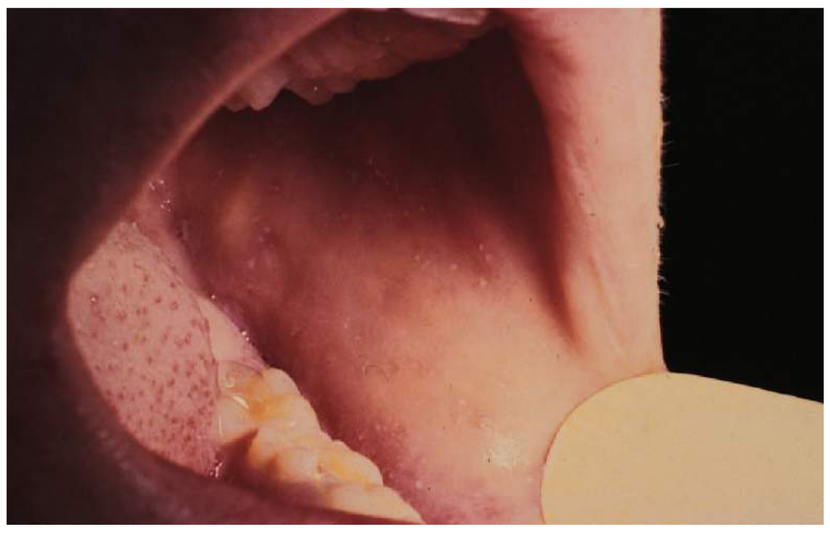


FIGURE 14.25 ■ **Koplik Spots.** Tiny white dots (Koplik spots) are seen on the buccal mucosa. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. Measles is now rare in the United States but remains endemic in other parts of the world.
2. MMR vaccine is preferable to IVIG as postexposure prophylaxis.
3. Morbilliform means measles-like.

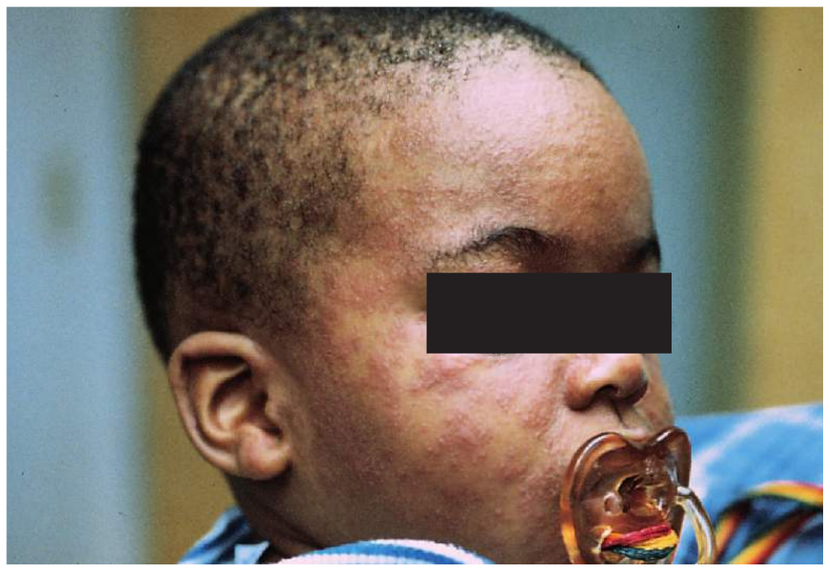


FIGURE 14.26 ■ **Measles.** School-aged child with a morbilliform rash on his face consistent with measles. (Photo contributor: Javier A. Gonzalez del Rey, MD.)



FIGURE 14.27 ■ **Measles.** School-aged child with a morbilliform rash on his face consistent with measles. (Photo contributor: David Effron, MD.)

VARICELLA (CHICKENPOX)

Clinical Summary

Chickenpox results from primary infection with varicella zoster virus (VZV) and is characterized by a generalized pruritic vesicular rash, fever, and mild systemic symptoms. Fifteen days after exposure and following a prodrome of fever, malaise, pharyngitis, and/or loss of appetite, the characteristic generalized pruritic vesicular rash develops. The lesions usually develop 24 hours after the onset of illness, appear in crops, start on the trunk and spread peripherally, and evolve from erythematous, pruritic macules to papules and vesicles (rarely bullae) that finally crust over within 48 hours. The classic lesions are teardrop vesicles surrounded by an erythematous ring ("dewdrop on a rose petal"). The most common complication of varicella is secondary bacterial skin infection, usually with S pyogenes or S aureus. Other complications from varicella include encephalitis, glomerulonephritis, hepatitis, pneumonia, arthritis, and meningitis. Cerebellitis (manifested clinically as ataxia) may develop and is usually self-limited. Other viral infections that may manifest with vesicular rashes include herpes simplex, zoster, coxsackie, influenza, echovirus, and vaccinia. On occasion, varicella can be confused with papular urticaria.

Management and Disposition

Suspected varicella infection should lead to strict isolation early in the emergency department or office encounter. Most children have a self-limited illness and do not develop any complications. Treatment should be supportive and directed to pruritus and fever control while avoiding salicylates because of their association with Reye syndrome. Oral acyclovir initiated within 24 hours of the onset of the rash may result in a modest decrease in the duration of symptoms and in the number and duration of skin lesions. Acyclovir is not recommended routinely for treatment of uncomplicated varicella in an otherwise healthy child less than 12 years of age. In the immunocompromised host, varicella zoster immunoglobulin (VZIG), IV acyclovir, and hospital admission are indicated.

Pearls

1. Skin lesions in varicella present in successive crops so that macules, papules, vesicles, and crusted lesions may all be present at the same time.
2. Healthy children are no longer contagious when all lesions have crusted over (usually 4-5 days from the development of the initial lesions).
3. Consider oral acyclovir for those at risk for more severe infection, including anyone older than 12 years, with chronic diseases, and taking chronic aspirin or corticosteroid therapy. Postexposure prophylaxis includes VZIG within 96 hours of exposure in high-risk patients (immunocompromised patients or pregnant women) or immunization with varicella vaccine within 72 hours of exposure in non–high-risk patients.



FIGURE 14.28 ■ **Varicella (Chickenpox).** Multiple cloudy vesicles of varicella. (Photo contributors: Katharine Hanlon and Kara Shah, MD.)



FIGURE 14.29 ■ **Varicella (Chickenpox).** Vesicles in different stages of maturation. Note vesicles surrounded by an erythematous ring ("dewdrop on a rose petal"). (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Zoster (shingles) represents a reactivation of latent VZV and has been noted as early as the first week of life in infants born to mothers who contracted varicella during pregnancy. The lesions present as clustered vesicles or bullae in a dermatomal distribution. The pain of acute neuritis occurs in 75% of patients with herpes zoster. Prodromal pain can be constant or intermittent, burning or stabbing and can precede the lesions by days to weeks. Sometimes pain can persist beyond 1 month after the lesions have disappeared (known as postherpetic neuralgia).

The diagnosis is usually made clinically; however, tissue cultures, direct fluorescent antibodies, and Tzanck smears can be done from vesicle scrapings for confirmation. Impetigo and cutaneous burns may mimic the appearance of herpetic vesicles. Varicella (chickenpox) is more diffusely spread, although a small crop of lesions may mimic zoster. Zoster also may be confused with herpes simplex virus (HSV) infection, although a close examination should reveal a dermatomal distribution in zoster.

Management and Disposition

Currently, acyclovir (800 mg orally five times per day for 7-10 days initiated within 48 hours of the onset of the rash in children ≥12 years) is the treatment of choice for zoster infections in immunocompetent children. Pain relief and prevention of secondary infection are also important. IV antiviral therapy is recommended for immunocompromised patients. There is no pediatric formulation of famciclovir or valacyclovir, and there are insufficient data on their use in children.

Pearls

1. Zoster can occur in children of all ages.
2. The most common sites for the development of zoster lesions are those supplied by the trigeminal nerve and the thoracic ganglia.
3. A patient with zoster can transmit chickenpox (varicella) to a nonimmune child or adult.
4. Herpes zoster ophthalmicus is a sight-threatening condition that presents with hyperesthesia in the effected eye. Vesicular lesions on the nose (Hutchinson sign) are associated with an increased risk of eye involvement.



FIGURE 14.30 ■ **Herpes Zoster.** Vesicles in a classic thoracic dermatomal distribution are seen in this child. (Photo contributor: Frank Birinyi, MD.)



FIGURE 14.31 **Herpes Zoster.** Vesicles in a trigeminal nerve distribution. (Photo contributor: Anne W. Lucky, MD.)

HAND, FOOT, AND MOUTH SYNDROME

Clinical Summary

Hand, foot, and mouth syndrome is a seasonal (summer-fall) viral infection caused by coxsackievirus A16. Toddlers and school-aged children are affected most commonly. Adults may also be affected. It is characterized by a prodrome of fever, malaise, sore throat, and anorexia over 1 to 2 days, followed by the appearance of the characteristic enanthem in the posterior oropharynx and tonsillar pillars consisting of small, red macules evolving into small vesicles 1 to 3 mm in diameter that rapidly ulcerate. Oral manifestations are followed by a vesicular eruption characterized by 3- to 7-mm erythematous macules with a central gray vesicle on the hands and feet involving the palmar and plantar surfaces and interdigital surfaces. Nonvesicular rash may also be present on the buttocks, face, and legs.

Management and Disposition

Supportive therapy (hydration maintenance with fever and pain control) is the mainstay of treatment. Discuss the duration and characteristics of the illness with the parents. In most cases, the course is self-limited resolving in 2 to 3 days after the appearance of rash without further complication. Rare secondary complications such as myocarditis, pneumonia, pulmonary hemorrhage, and meningoencephalitis may occur.

Pearls

1. The child is contagious until all vesicles have resolved.
2. The oral lesions tend to involve the posterior oropharynx as contrasted with those of herpetic gingivostomatitis which typically involve the anterior structures of the mouth.
3. Varicella vesicles are located more centrally, are more extensive, and usually spare the palms and soles.



FIGURE 14.32 ■ **Hand, Foot, and Mouth Disease.** Erythematous vesicular rash scattered on the palms consistent with coxsackievirus. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 14.33 ■ **Hand, Foot, and Mouth Disease.** Vesicular rash of the feet consistent with coxsackievirus. (Photo contributor: Larry B. Mellick, MD.)



FIGURE 14.34 ■ **Hand, Foot, and Mouth Disease.** Discrete vesicular erosions on the posterior oropharynx and soft palate secondary to coxsackievirus. (Photo contributor: Larry B. Mellick, MD.)



FIGURE 14.35 ■ **Hand, Foot, and Mouth Disease.** Nonvesicular rash on the buttocks, a common site. (Photo contributor: Binita R. Shah, MD. Used with permission from Shah B, Lucchesi M, Amodio J, Silverberg M. Atlas of Pediatric Emergency Medicine. 2nd ed. New York, NY: McGraw-Hill; 2013: Fig. 3.66D, p. 111.)

COLD PANNICULITIS (POPSICLE PANNICULITIS)

Clinical Summary

Cold panniculitis represents acute cold injury resulting in inflammation of the subcutaneous fat. It manifests as erythematous, indurated nodules, and plaques on exposed skin, especially the perioral areas and cheeks. Lesions appear 24 to 72 hours after exposure to cold and gradually soften and return to normal over 1 to 2 weeks usually without permanent sequelae. This phenomenon is caused by subcutaneous fat solidification and necrosis following exposure to cold temperatures. It is much more common in infants. The differential diagnosis includes facial cellulitis, frostbite, trauma, pressure erythema, giant urticaria, and contact dermatitis.

Management and Disposition

Treatment is supportive. Parental education and reassurance is important.

Pearls

1. Because these lesions may also be painful, the differentiation of cold panniculitis from cellulitis may be difficult. The absence of systemic symptoms, especially fever, and the history of cold exposure are more suggestive of cold panniculitis.
2. The lesions may resolve with resulting hyperpigmentation of the affected area.

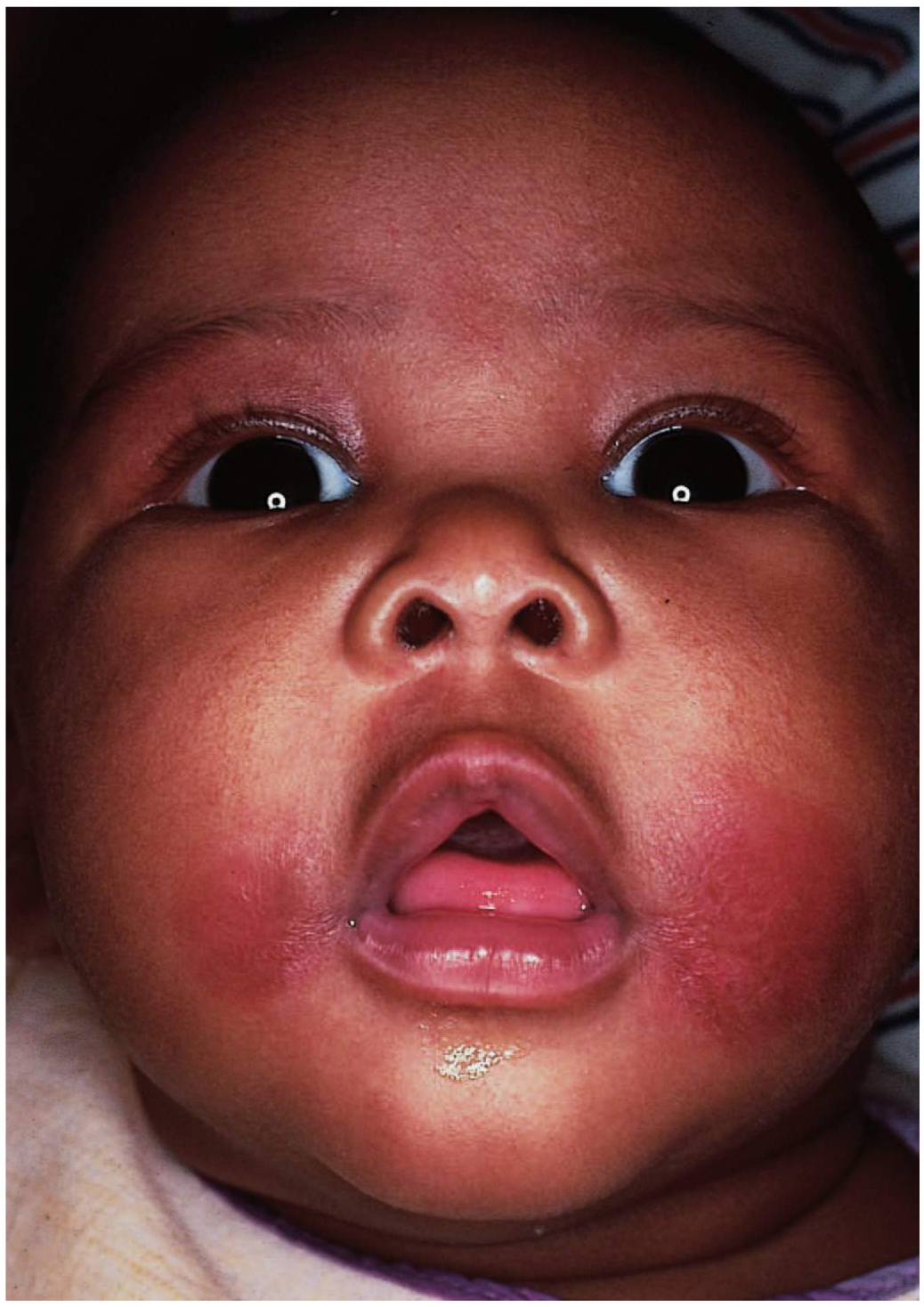


FIGURE 14.36 ■ **Cold Panniculitis.** Infant with cheek erythema, swelling, and discoloration consistent with popsicle panniculitis or cold injury. (Photo contributor: Anne W. Lucky, MD.)

HERPETIC GINGIVOSTOMATITIS

Clinical Summary

Herpetic gingivostomatitis is primary infection caused by HSV seen in up to 30% of children between 6 months and 5 years of age. Patients usually present with approximately 4 days of fever, malaise, decreased oral intake, cervical adenopathy, and pain in the mouth and throat. Following the prodrome, vesicular and ulcerative lesions appear throughout the oral cavity. The gingiva becomes very friable and inflamed, especially around the alveolar rim. Increased salivation, foul breath, and cervical lymphadenitis may be present. Although fever resolves in 3 to 5 days, children may have difficulty eating for 7 to 14 days. Sometimes, autoinoculation produces vesicular lesions on the fingers (herpetic whitlow).

Management and Disposition

Treatment includes pain control, hydration, and consideration of oral acyclovir therapy. The pain may be significant and often requires oral narcotic pain medications. Control of the pain will allow the patient to consume fluids and remain well hydrated. Acyclovir has been shown to reduce the duration of pain, gingival swelling, oral lesions, fever, and viral shedding if initiated within the first 72 to 96 hours of illness. Avoidance of citrus juices or spicy food is recommended. Cold clear fluids, popsicles, and ice cream may be useful in small children. Not infrequently, admission for IV hydration is necessary. Topical pain control may be achieved by using mixtures of antihistamine (diphenhydramine elixir) and antacid (1:1) applied to lesions with a cotton swab. Local application of viscous lidocaine should be avoided in children, since patients may develop toxic serum levels due to altered absorption from inflamed oral mucosa leading to seizures.

Pearls

1. Most lesions are in the anterior two-thirds of the oral cavity. Posterior lesions sparing the gingiva are most commonly seen in coxsackievirus infections.
2. Primary HSV infection in childhood is usually asymptomatic.
3. After primary oral infection, HSV remains latent in the trigeminal ganglion until reactivation as herpes labialis.



FIGURE 14.37 ■ **Herpetic Gingivostomatitis.** Multiple oral vesicular lesions and tongue ulcerations consistent with herpes gingivostomatitis. Vesicular lesions from autoinoculation are present on the finger (herpetic whitlow). (Photo contributor: Michael J. Nowicki, MD.)



FIGURE 14.38 ■ **Herpetic Stomatitis.** Multiple perioral vesicular lesions consistent with herpes gingivostomatitis. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Meningococcemia is an acute febrile illness caused by Neisseria meningitidis bacteremia characterized by its generally rapid onset, significant toxicity, and petechial rash involving the skin and mucous membranes. The petechiae progress to become palpable purpura and may coalesce to become purpura fulminans. It progresses rapidly to decompensated septic shock with hypotension and multisystem organ failure. In cases of fulminant disease, progressive shock is accompanied by disseminated intravascular coagulation and massive mucosal hemorrhages. Prodromal symptoms may include cough, coryza, headache, neck pain, and malaise. Children less than 5 years of age and college students not receiving the meningococcal vaccine during high school are at greatest risk.

Management and Disposition

In stable patients in which meningococcemia is in the differential diagnosis, obtain cultures of blood and spinal fluid and from the nasopharynx, along with a CBC and coagulation studies. Consider bedside screening studies (eg, blood gas to assess acid-base status), liver function tests, and other studies as clinically indicated. These patients should be admitted for intensive monitoring to institutions capable of delivering critical care services. Broad-spectrum parenteral antibiotics should be administered initially until the organism is identified and sensitivities are available as with any patient with suspected sepsis. In the unstable septic patient, support end organ perfusion and oxygenation via early goal-directed therapy. Hemodynamic monitoring and blood pressure support (fluids and vasoactive drugs) are of paramount importance. Peripheral and central venous catheters and urinary and arterial catheters are usually necessary for optimal management of these patients.

Pearls

1. Skin scrapings of the purpuric lesion can be cultured and microscopically examined for the presence of gram-negative diplococci.
2. A child with a fever and an evolving petechial rash is presumed to have meningococcemia until proven otherwise.
3. A quadrivalent meningococcal conjugate vaccine is now available and recommended for all adolescents between ages 11 and 12 and then again at age 15 years or high school entry (whichever comes first).
4. Prophylaxis for all close contacts is recommended with rifampin for 48 hours or a single dose of ceftriaxone or ciprofloxacin.

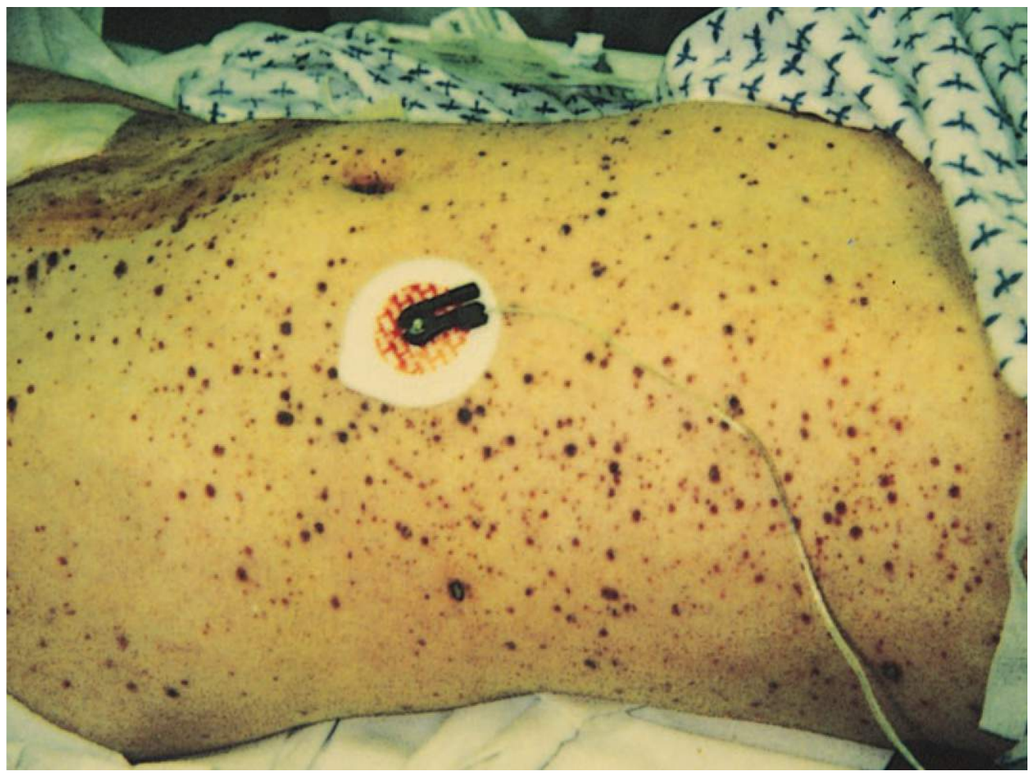


FIGURE 14.39 ■ **Meningococcemia.** Diffuse petechiae in a patient with meningococcemia. (Photo contributor: Richard Strait, MD.)

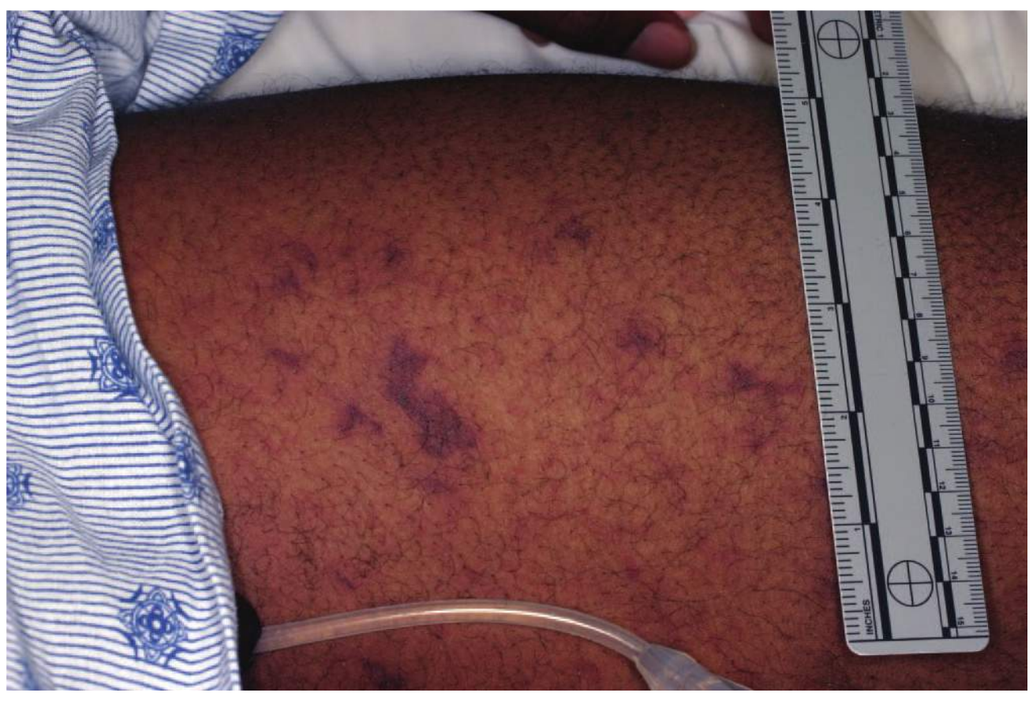


FIGURE 14.40 ■ **Meningococcemia.** Petechiae and purpura in an adolescent patient with meningococcemia. (Photo contributor: Kevin J. Knoop, MD, MS.)

Clinical Summary

Acute urticaria (defined as <6 weeks' duration) is a common condition in children caused by inflammatory mediator (histamine and others) release from mast cells and basophils in the superficial dermis. These lesions are often associated with an infection, an insect sting or bite, or ingestion of certain foods or medications, though in many cases the precise etiology cannot be identified. It is characterized by the sudden onset of intensely pruritic lesions that are raised, erythematous, well-circumscribed, and blanching. They may occur anywhere on the body and can vary in size from pinpoint to several centimeters in diameter. They can have central clearing or associated edema. Individual lesions usually resolve in 1 to 3 hours, but some may last for up to 24 to 48 hours. Rarely, they may take weeks to resolve completely.

Anaphylaxis is a systemic allergic reaction defined by multiorgan system involvement that can have an urticarial component along with wheezing, stridor, angioedema, vomiting, and hypotension. The differential diagnosis for urticaria includes erythema multiforme, Henoch-Schönlein purpura (HSP), arthropod bites, contact dermatitis, reactive erythemas, allergic vasculitis, juvenile rheumatoid arthritis, mastocytosis, and pityriasis rosea.

Management and Disposition

Treatment is symptomatic. If the offending agent can be identified, avoidance is recommended. Oral antihistamines are useful in the control of pruritus and often lead to improvement or complete resolution of the rash within an hour of administration. As the effect of the antihistamine wanes, the urticaria will often recur. If the patient has systemic signs suggesting anaphylaxis, administer intramuscular (IM) epinephrine as soon as possible. Antihistamines (H1 and H2 blockers), steroids, and IV fluids may also be helpful. Unless there is evidence of acute angioedema or anaphylaxis, most cases can be discharged home on oral antihistamines to follow up with a primary care physician. Specific allergen testing is not recommended in the emergency department.

Pearls

1. Ninety percent of patients with anaphylaxis have cutaneous reactions, which are often urticarial. IM epinephrine is the only medication that decreases the risk of mortality in anaphylaxis.
2. By definition, a lesion of urticaria must change or resolve within 24 hours of its appearance.
3. The most common causes of urticaria in children are viral illnesses and foods. Egg, milk, soy, peanuts, and wheat are the most common allergens in young children. In older children, fish, seafood, nuts, and peanuts are more common causes.
4. The addition of an H2 antagonist (ranitidine or famotidine) may be helpful when H1 agents alone are ineffective.



FIGURE 14.41 ■ **Urticaria.** Well-circumscribed, raised, pruritic extremity lesions consistent with urticaria. (Photo contributor: Anita P. Sheth, MD.)



FIGURE 14.42 ■ **Urticaria.** Preschool child with annular, raised pruritic lesions with the central clearing and tense edema of polycyclic urticaria. The lesions had completely disappeared after about 5 minutes. (Photo contributor: Kevin J. Knoop, MD, MS.)

Clinical Summary

Summer penile syndrome is a benign hypersensitivity reaction involving the skin of the penile shaft. As the name suggests, it is seen during the warm weather months. It is also known as seasonal acute hypersensitivity reaction and lion's mane penis. The history may include recent play outside in the grass or a wooded area within the last 24 hours. It is most commonly associated with insect bites, usually chiggers, but may also be caused by exposure to plants (poison ivy, sumac, and oak). The diagnosis is made clinically and is suspected when the skin of the shaft of the penis, most often just proximal to the glans, is markedly edematous. There is minimal erythema and no fluctuance. Four out of five patients have pruritus and few have urinary symptoms. Symptoms (both swelling and pruritus) can last up to 2 to 3 weeks, but most patients have resolution in 4 to 5 days. The differential diagnosis includes trauma, nephrotic syndrome, HSP, balanitis, phimosis, paraphimosis, and priapism.



FIGURE 14.43 ■ **Summer Penile Syndrome.** Edema of the distal penile shaft and left scrotum. An insect bite is seen at the base of the left mons pubis. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Treatment consists of an oral antihistamine if the patient has pruritus and cool compresses. Systemic corticosteroids should only be considered if the child is in significant pain or is having trouble with the voiding stream. Patients can be discharged home after education and reassurance. Urology involvement and referral is usually not necessary.

Pearls

1. Summer penile syndrome is usually painless.
2. Pruritus is the most common symptom.
3. Testing is unnecessary, but the diagnosis must be distinguished from paraphimosis.



FIGURE 14.44 ■ **Summer Penile Syndrome.** Also known as "lion's mane penis" due to the marked circumferential edema just proximal to the glans. (Photo contributor: Larry B. Mellick, MD.)

STAPHYLOCOCCAL SCALDED SKIN SYNDROME

Clinical Summary

Staphylococcal scalded skin syndrome (Ritter disease) most commonly affects infants and children less than 5 years of age and is caused by an exfoliative exotoxin-producing strain of S aureus. Initial presentation includes fever, malaise, and irritability following an upper respiratory infection with pharyngitis or conjunctivitis. Patients first develop a diffuse faint blanching erythematous rash that is tender to the touch. Crusting around the mouth, eyes, and neck may be seen. Within 2 to 3 days, flaccid blisters and bullae develop especially in flexor creases, the buttocks, hands, and feet. These bullae are sterile. In some patients, widespread desquamation occurs. The differential diagnosis includes toxic epidermal necrolysis, exfoliative erythroderma, bullous erythema multiforme, bullous pemphigoid, bullous impetigo, sunburn, acute mercury poisoning, toxic shock syndrome, and epidermolysis bullosa.

Management and Disposition

If staphylococcal scalded skin syndrome is suspected, obtain cultures from the blood, urine, nasopharynx, abnormal area of skin, or other suspected foci of infection. Treatment is directed at the eradication of S aureus, thus terminating the production of toxin. Semisynthetic penicillinase-resistant penicillins or clindamycin should be used intravenously. Consider the addition of vancomycin in areas with a high prevalence of methicillin-resistant S aureus (MRSA). Admission is usually necessary, especially in young infants. This age group requires careful attention to fluid and electrolyte losses and the prevention of secondary infection of the denuded skin. Management of pain associated with the rash may require narcotics.

Pearls

1. The wrinkling or peeling of the upper layer of the epidermis (pressure applied with a Q-tip or gloved finger) that occurs within 2 or 3 days of the onset of this illness is known as Nikolsky sign.
2. The fluid in the bullae of staphylococcal scalded skin syndrome is sterile. The toxin is produced at a remote site and delivered to the skin via the bloodstream.
3. Infants with involvement of large areas of the body surface are at increased risk for hypothermia and fluid/electrolyte losses.
4. Corticosteroids are contraindicated in the treatment of staphylococcal scalded skin syndrome.
5. These lesions do not typically scar as the cleavage plane is intraepidermal.



FIGURE 14.45 ■ **Staphylococcal Scalded Skin Syndrome.** Toddler with diffuse macular peeling eruption consistent with scalded skin syndrome from S aureus. (Photo contributor: Judith C. Bausher, MD.)

Clinical Summary

Scarlet fever manifests as diffuse blanching "sandpaper-like" erythematous macules and papules caused by erythrogenic toxin production from group A β-hemolytic Streptococcus pharyngitis. Occasionally, the site of infection is skin (impetigo) or perianal. The disease usually occurs in children 2 to 10 years of age. The typical presentation of scarlet fever includes fever, headache, sore throat, nausea, vomiting, and malaise followed by the characteristic scarlatiniform rash. The rash initially occurs centrally on the face (often with perioral sparing), neck, and upper trunk but quickly becomes generalized. Desquamation occurs after 5 to 7 days. On the tongue, a thick, white coat and swollen papillae may be seen ("strawberry tongue"). Palatal petechiae and tender anterior cervical lymphadenopathy may be present. The differential diagnosis includes enteroviral infections, staphylococcal scalded skin syndrome, viral hepatitis, infectious mononucleosis, toxic shock syndrome, drug eruptions, rubella, mercury poisoning, and Kawasaki disease.

Management and Disposition

Penicillin, either a single IM dose of benzathine penicillin G or oral amoxicillin for 10 days, is the treatment of choice. Alternatives include erythromycin or clindamycin in penicillin-allergic patients.



FIGURE 14.46 ■ **Scarlatina.** Erythematous scarlatiniform rash of scarlet fever. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. In scarlet fever, petechiae in a linear pattern along the major skin folds in the axillae and antecubital fossae are known as "Pastia lines."
2. In dark-skinned individuals, the rash may be difficult to differentiate and may consist only of punctate papular elevations called "goose flesh."
3. There has never been an isolate of group A Streptococcus that is resistant to penicillin.



FIGURE 14.47 ■ **Goose Flesh.** This sandpaper rash started 2 days after sore throat and fever began in this 6-year-old. The grouping of the fine papules gives the skin a "goose flesh" texture in darker skin colors. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 14.48 ■ **Palatal Petechiae.** Petechiae present on the posterior soft palate of a child with group A streptococcal infection. (Photo contributor: Hannah F. Smitherman, MD.)

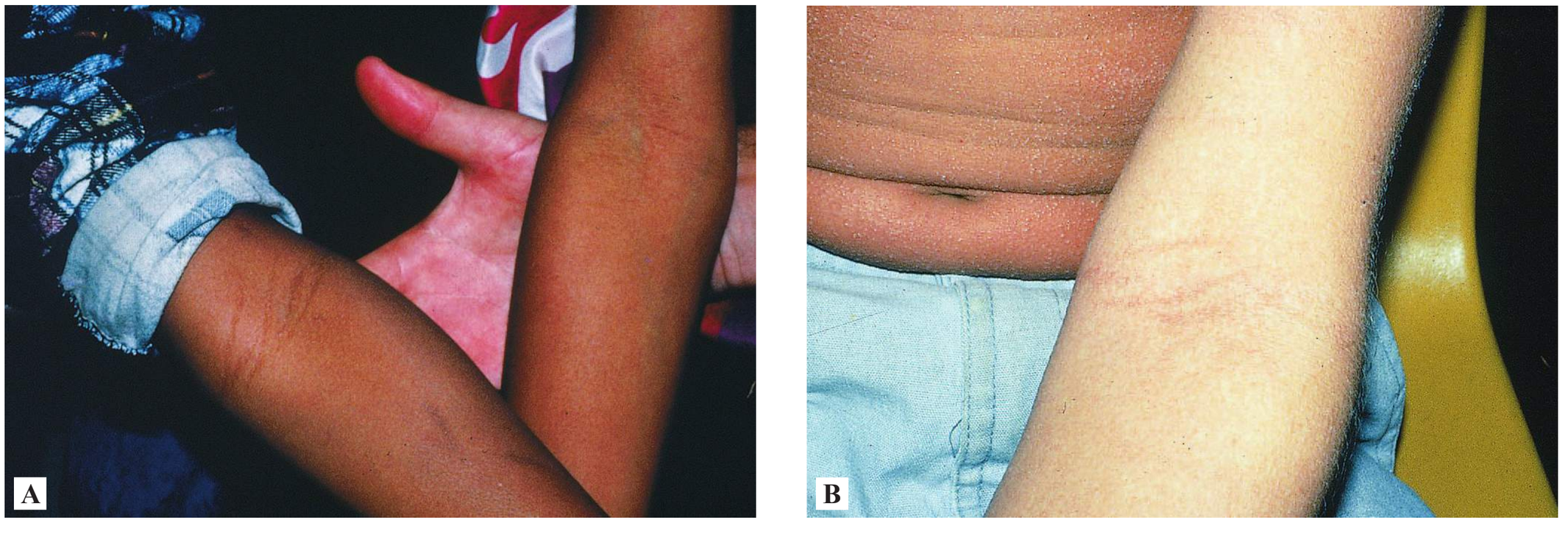


FIGURE 14.49 ■ **Pastia Lines.** Confluent petechiae in a linear pattern in the antecubital fossa consistent with Pastia lines are seen in these patients with scarlet fever. (**A**) The forearm on the right belongs to the patient's sister and does not show Pastia lines. (**B**) Pastia lines in a Caucasian patient. A classic sandpaper rash is also evident on the arm and trunk. (Photo contributor: Stephen W. Corbett, MD.)



FIGURE 14.50 ■ **GABHS Toxic Shock Syndrome.** Nine-year-old girl with strawberry tongue, nausea, vomiting, fever, and sandpaper rash over trunk, arms, and legs for 4 days suggestive of toxic shock syndrome. Vital signs on presentation: HR: 152, BP: 84/55. (Photo contributor: Eric M. Sergienko, MD.)



FIGURE 14.51 ■ **Desquamating Rash.** Desquamation of the fingertips occurred as the scarlatiniform rash began to fade. Desquamation also occurred in the groin and toes. (Photo contributor: Clay. B. Smith, MD.)



FIGURE 14.52 ■ **Perianal Strep Desquamation.** Desquamating rash in the perineum of a child with GABHS infection. (Photo contributor: Kevin J. Knoop, MD.)

BLISTERING DISTAL DACTYLITIS

Clinical Summary

Blistering distal dactylitis is a cellulitis of the fingertip caused by group A β-hemolytic Streptococcus or S aureus infection in children from infancy to teenage years. The typical lesion is a fluid-filled, painful, tense blister with surrounding erythema located over the volar fat pad on the distal portion of a finger or toe. Polymorphonuclear leukocytes and gram-positive cocci can be found in the Gram stain of the purulent exudate from the lesion. The differential diagnosis includes bullous impetigo, burns, friction blisters, felon, and herpetic whitlow.

Management and Disposition

There is usually a rapid response to incision and drainage of the blister and a 10-day course of antibiotic therapy. Consider use of agents active against MRSA (clindamycin or trimethoprim/sulfamethoxazole) if there is a high community prevalence.

Pearl

1. Nonpurulent vesicular lesions that become confluent multilocular bullae are characteristic of herpetic whitlow and help distinguish it from blistering distal dactylitis.



FIGURE 14.53 ■ **Blistering Distal Dactylitis.** Blistering rash of the distal fingers with surrounding erythema typically caused by Streptococcus. Note the location of the rash over the volar finger pads. (Photo contributor: Anne W. Lucky, MD.)

HENOCH-SCHÖNLEIN PURPURA

Clinical Summary

Henoch-Schönlein purpura (HSP) is the most common systemic vasculitis in children, with a peak incidence between ages 3 and 15 years. It is characterized by four main clinical manifestations. The classic **exanthem** of HSP begins with erythematous macules or urticaria that eventually coalesce and evolve into ecchymotic lesions and palpable purpura. The lesions are more often located on the buttocks and gravity dependent areas (lower extremities) in ambulatory children. In nonambulatory children, the lesions can be seen on the face, trunk, and upper extremities as well. Mucosal involvement is rare; however, edema of the scalp, hands, scrotum, and periorbital tissue occurs.

Migratory oligoarticular (1 to 4 joints) **arthritis** and **arthralgia** are eventually seen in 84% of patients. **Gastrointestinal symptoms** (colicky abdominal pain, occult or gross blood in the stool, and small bowel-small bowel intussusception) usually develop within a week of the rash, but may precede it. **Renal involvement** is the most frequent serious complication and usually occurs during the first month. It commonly manifests as microscopic hematuria and may progress to glomerulonephritis. One percent progress to end-stage renal disease. Hypertension is uncommon.

Diagnosis is made by history and clinical examination. Laboratory tests are usually normal (platelets, complement level, coagulation studies, and antinuclear antibodies) except for the urinalysis, which may be positive for blood or protein in 50%. The overall prognosis is excellent, with full recovery in most. The course is marked by relapses and remissions in 50% (the rash tends to recur within the first 6 weeks). Prognosis is linked to renal involvement. The rash may be confused with drug reactions, erythema multiforme, urticaria, and even physical abuse. Consider other causes of purpura, such as bleeding disorders or infection (meningococcemia).

Management and Disposition

Treatment is supportive. Patients with HSP limited to the skin and joints with minimal urinary findings can be managed as outpatients. Hospitalization is warranted when patients cannot maintain oral intake, have severe abdominal pain, GI hemorrhage, and intussusception, are unable to ambulate or care for themselves because of significant joint pain, or have evidence of renal injury. Ibuprofen or naproxen can be effective for symptomatic pain relief. Corticosteroids may shorten the duration of abdominal pain in patients with severe symptoms that are unable to maintain oral intake. They are not recommended routinely for rash or joint pains. In cases where intussusception is suspected, prompt surgical consultation and diagnostic ultrasonography or air-contrast enema are indicated. In contrast to ileocolic intussusception, small bowel-small bowel intussusception generally resolves spontaneously and rarely requires surgical intervention.

Pearls

1. Obtain a stool occult blood test and urinalysis in patients with abdominal pain and suspected HSP. The fecal occult blood test is positive in more than half.
2. Intussusception associated with HSP is seen in 2% of patients, most commonly in boys, particularly those about 6 years of age. The intussusception is often ileo-ileal.
3. The typical rash occurs in nearly 100% and is the presenting feature in at least 50%. Joint symptoms may precede the rash as the presenting complaint in 25% of patients. Ankles and knees are the most commonly affected joints.
4. Scrotal swelling and pain may be the initial presenting sign in boys and can mimic testicular torsion.



FIGURE 14.54 ■ **Henoch-Schönlein Purpura.** These erythematous, hemorrhagic papules and petechiae in a symmetric acral distribution are classic findings in HSP. This child presented with no other symptoms. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

It is characterized by rash, fever, and polyarthralgias with onset 1 to 2 weeks following exposure to an offending agent, and resolves within 1 to 2 weeks after exposure is discontinued. It is felt to occur via an immune complex–mediated mechanism and can be precipitated by exposure to a number of drugs, with cefaclor, trimethoprim-sulfamethoxazole, and penicillins being the most common. Almost all patients develop a polymorphous pruritic rash that starts in the trunk, groin, and axillae, eventually spreading to the limbs. The urticarial lesions are longer lasting than typical hives. Some patients can exhibit palpable purpura, maculopapular lesions, or erythema multiforme–type exanthema. In all patients, the mucous membranes are spared. Almost all patients develop remittent fever without temporal spikes. Two-thirds will have arthralgias, most often in the peripheral extremities. Frank arthritis is more rare, but may lead to a child being unable/unwilling to walk. The differential diagnosis includes viral exanthems, hypersensitivity vasculitis, scarlet fever, acute rheumatic fever, meningococcemia, disseminated gonococcemia, reactive arthritis, Lyme disease, Still disease, and Stevens-Johnson syndrome.

Management and Disposition

The diagnosis is clinical and based on history of exposure to a potential offending agent. Treatment consists of discontinuing the causative agent and supportive care. Glucocorticoids may benefit patients with severe symptoms. Diphenhydramine can be used in cases where pruritus is a prominent symptom. Patients who are ill-appearing or do not have a history of a potential offending agent should have a CBC with differential, erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP), urinalysis, blood urea nitrogen (BUN), creatinine, serum electrolytes, urinalysis, and blood culture obtained to exclude other inflammatory or infectious causes.

Pearls

1. Serum sickness, as originally described, is typically in response to administration of a nonhuman species protein antigen. Present-day examples include sheep-derived Fab snake antivenom, heterologous immunomodulators containing murine components (rituximab and infliximab), streptokinase, and the human diploid cell rabies vaccine.
2. Patients with a history of serum sickness–like reaction will have a more rapid onset of symptoms if reexposed to the causative agent.
3. Mucous membrane and eye involvement does not occur in serum sickness–like reactions. These findings should prompt consideration for Stevens-Johnson syndrome, a potentially fatal hypersensitivity reaction.



FIGURE 14.55 ■ **Serum Sickness–Like Reaction.** Fever, joint pain, and erythematous plaques to face (**A**) and legs (**B**) 14 days after receiving amoxicillin in a 7-year-old girl. These clinical findings suggest serum sickness. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 14.56 ■ **Serum Sickness–Like Reaction.** Fever, joint pain, ankle swelling, and erythematous plaques seen here in an infant 11 days after taking ceclor, suggesting serum sickness. (Photo contributor: Lawrence B. Stack, MD.)

STRAWBERRY HEMANGIOMA

Clinical Summary

Hemangiomas are benign vascular tumors characterized by a rapid proliferative phase followed by a spontaneous involutional phase. They are the most common soft tissue tumors of infancy. The appearance of hemangiomas is defined by the lesion's depth, location, and stage of evolution. A strawberry hemangioma lies in the upper dermis and often originates as an erythematous macular patch, a pale macule, or a localized telangiectasia with a pale halo. The lesion grows and becomes vascularized during the first 2 months of life. The classic presentation is a bright red, slightly raised, noncompressible plaque. It commonly regresses by 2 to 3 years of age but may persist throughout life. Hemangiomas can also affect the airway, eyes, and liver or cause high-output cardiac failure if sufficiently large. The most important local complication is ulceration which can be exquisitely painful. The differential diagnosis includes vascular malformations, malignant vascular neoplasms, pyogenic granulomas, and giant melanocytic birthmarks.



FIGURE 14.57 ■ **Strawberry Hemangioma.** Raised umbilicated vascular lesion on the left upper chest consistent with strawberry hemangioma. (Photo contributors: Kara Shah, MD, PhD, and Katharine Hanlon.)

Management and Disposition

Most cases require no therapy because strawberry hemangiomas usually regress without residual problems. Treatment of hemangiomas in general is indicated when there is an obstruction of a vital orifice (ie, airway, mouth, or nares) or vision (eyelids) or if hematologic or cardiovascular complications are present. Education and parental reassurance are important because there is great pressure to treat for cosmetic reasons. Oral corticosteroids, intralesional corticosteroids, pulsed dye laser, and recombinant interferon-α are some of the therapeutic modalities available. In complex cases, consultation with a vascular malformation specialist or dermatologist is recommended.

Pearls

1. Kasabach-Merritt phenomena (hemolytic anemia, thrombocytopenia, and coagulopathy) is associated with Kaposiform hemangioendothelioma or tufted angiomas and not with common hemangiomas as was previously thought.
2. As many as 20% of affected infants have multiple lesions.
3. In contrast to hemangiomas, vascular malformations, such as port-wine stains, do not proliferate or involute. They persist throughout life and grow in proportion with the child.
4. Patients with hemangiomas of the face, and especially in the "beard" distribution, have a higher risk of also having an airway hemangioma. Hemangiomas over the midline lumbar spine should prompt an evaluation for spinal dysraphism.



FIGURE 14.58 ■ **Strawberry Hemangioma.** Vascular lesion on the nipple. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 14.59 ■ **Strawberry Hemangioma.** Vascular lesion on the upper lip consistent with strawberry hemangioma. (Photo contributors: Kara Shah, MD, PhD, and Katharine Hanlon.)

ORBITAL AND PRESEPTAL (PERIORBITAL) CELLULITIS

Clinical Summary

Orbital cellulitis is a serious bacterial infection characterized by fever, painful purple-red eyelid swelling, ophthalmoplegia, pain with extraocular movements, proptosis, and variable decreased visual acuity. It may begin with eye pain and fever. In general, it is caused by Streptococcus pneumoniae, Haemophilus influenzae, Moraxella catarrhalis, and S aureus. It usually arises as a complication of ethmoid or maxillary sinusitis. If not treated promptly, it can lead to blindness, cavernous venous sinus thrombosis, meningitis, subdural empyema, or brain abscess.

Preseptal (periorbital) cellulitis is much more common and usually presents with edema and circumferential erythema of the eyelids and periorbital skin, fever, and minimal pain. Proptosis and ophthalmoplegia are not characteristic as it does not involve the orbit or other ocular structures. Preseptal cellulitis usually results from sinusitis or contiguous infection due to local skin trauma, insect bite, or hordeolum. Common organisms are S aureus and group A Streptococcus.

Management and Disposition

A broad-spectrum parenteral antibiotic with staphylococcal coverage, ophthalmologic consultation, and admission are indicated in cases of orbital cellulitis. Orbital CT with contrast is needed to assess for abscess requiring surgical drainage. In febrile or ill-appearing patients with preseptal cellulitis, admission with broad-spectrum IV antibiotic therapy is also indicated. Adults and children greater than 1 year with mild cases (especially those with a history of trauma such as abrasion or insect bite or sting) can be treated with outpatient oral antibiotic therapy with close follow-up. Consider the use of antimicrobials with MRSA coverage.

Pearls

1. Obtain orbital and sinus CT if the diagnosis is uncertain and in patients who have pain with eye movement, vision changes, or if unable to assess vision (usually age less than 1 year).



FIGURE 14.60 ■ **Orbital Cellulitis.** CT confirmed orbital cellulitis in this 2-month-old. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 14.61 ■ **Orbital Cellulitis.** Eye redness, swelling, purulent drainage, and mild entrapment are seen in this patient with painful orbital cellulitis. (Photo contributor: Kevin J. Knoop, MD, MS.)

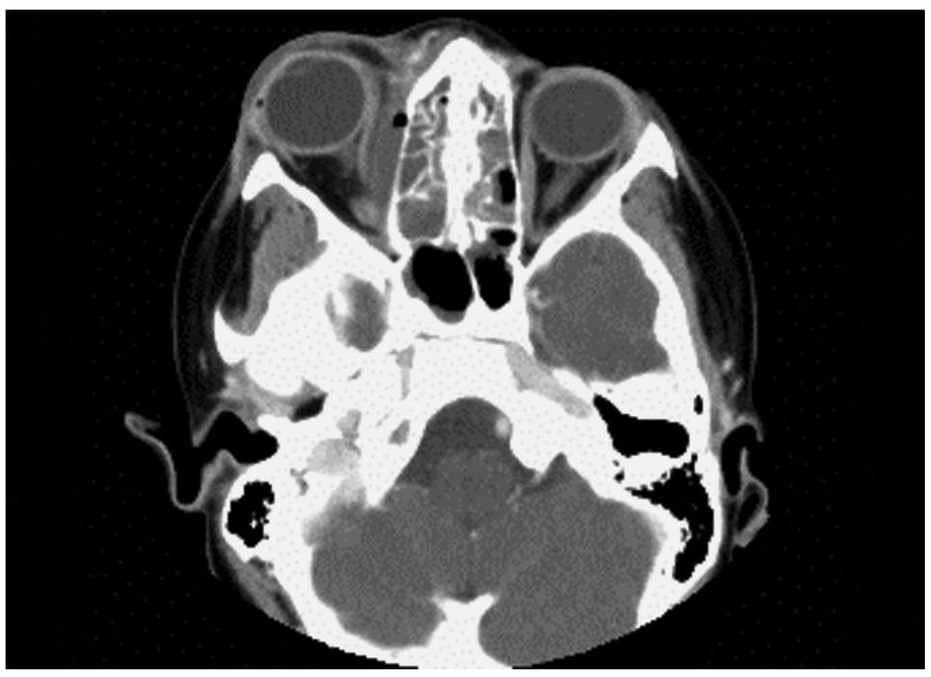


FIGURE 14.62 ■ **Orbital Cellulitis.** Right ethmoid sinusitis with subperiosteal abscess and extension into the orbital space in above patient. (Photo contributor: Kevin J. Knoop, MD, MS.)

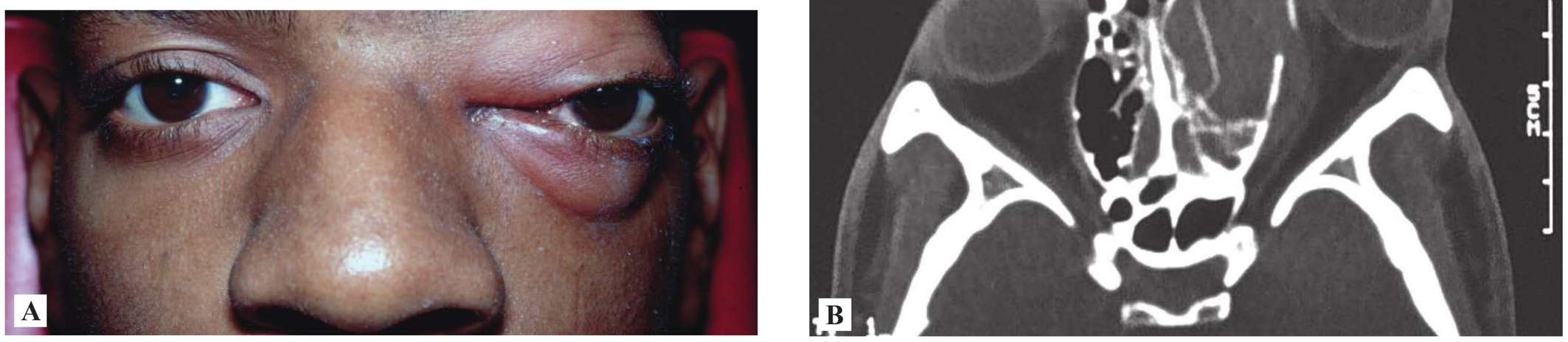


FIGURE 14.63 ■ **Orbital Cellulitis.** Left ethmoid sinusitis with extension into the orbital space, periosteal abscess formation, and proptosis is seen in this patient (**A**) and on CT (**B**). (Photo contributor: Lawrence B. Stack, MD.)

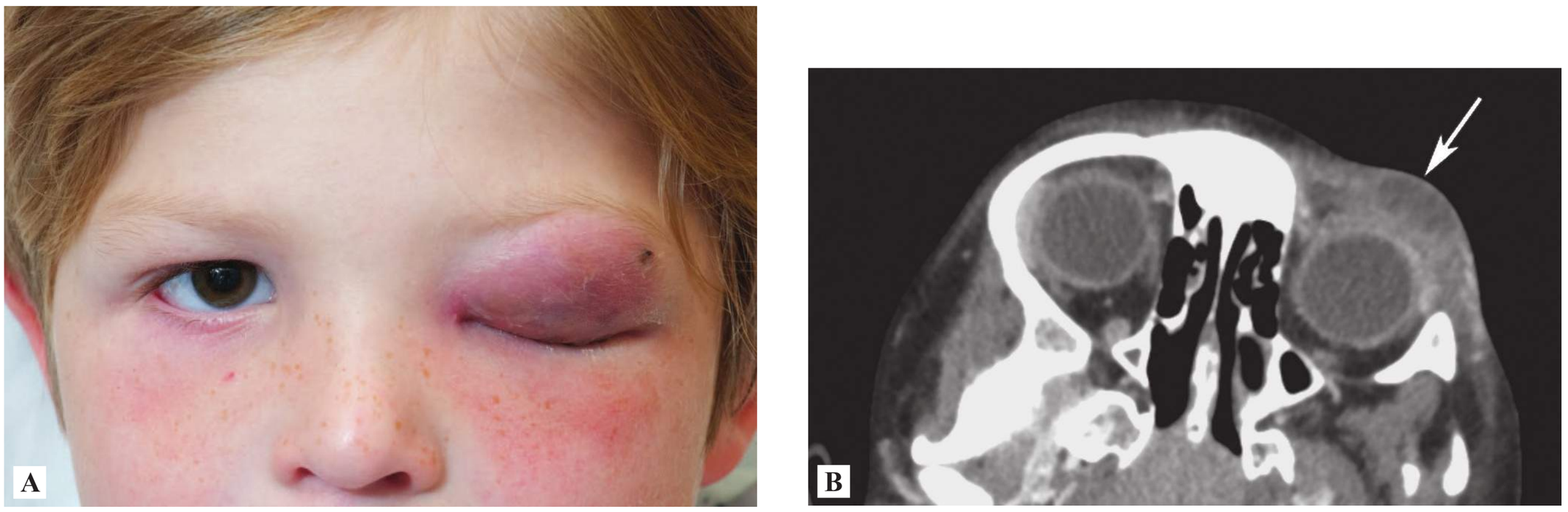


FIGURE 14.64 ■ **Preseptal Cellulitis.** Periorbital cellulitis with abscess formation seen in this patient (**A**) and on CT (**B**). (Photo contributor: Eftitan Akam.)



FIGURE 14.65 ■ **Preseptal Cellulitis.** Cellulitis originating from local skin trauma in the left eyebrow. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 14.66 ■ **Preseptal Cellulitis.** Preseptal cellulitis extending from a hordeolum. (Photo contributor: David Effron, MD.)



FIGURE 14.67 ■ **Preseptal Cellulitis.** Left periorbital cellulitis with edema and erythema of the eyelids in a nontoxic cooperative toddler with a normal ocular exam. (Photo contributor: Kevin J. Knoop, MD, MS.)

Clinical Summary

A branchial cleft cyst arises from the incomplete obliteration of one of the four branchial clefts during embryogenesis. As the obliteration of the clefts occurs, a portion may remain, forming a cystic space. The anatomic location of a branchial cleft cyst depends upon the specific arch/cleft involved. Involvement of the first branchial cleft may result in a cyst in the region of the parotid gland, the preauricular or postauricular area, or inferior to the angle of the mandible. A second cleft anomaly represents 95% of these malformations, and may be found along the anterior border of the sternocleidomastoid muscle or deeper, in the vicinity of the carotid arteries. Third and fourth arch/cleft anomalies are very rare. Cysts usually present clinically when they become infected and enlarge acutely usually in association with an upper respiratory infection.

Management and Disposition

The diagnosis is suggested by history and physical examination. Treatment includes antibiotics to decrease the inflammation if there is an associated infection. Ensure that there is no evidence of airway compromise. Complete excision by a specialist is necessary in order to prevent recurrence. CT or ultrasound may better define the extent of the lesion.

Pearls

1. Branchial cleft anomalies are second only to thyroglossal duct cysts in frequency of congenital head and neck lesions in children.
2. These lesions may be intimately involved with major vessels and nerves. Excision by a specialist familiar with the underlying anatomy is recommended.



FIGURE 14.69 ■ **Second Branchial Cleft Cyst.** Right neck mass seen in second branchial cleft cyst. (Photo contributor: Scott Manning, MD.)



FIGURE 14.68 ■ **First Branchial Cleft Cyst.** Periauricular mass from a first brachial cleft cyst. (Photo contributor: Lawrence B. Stack, MD.)

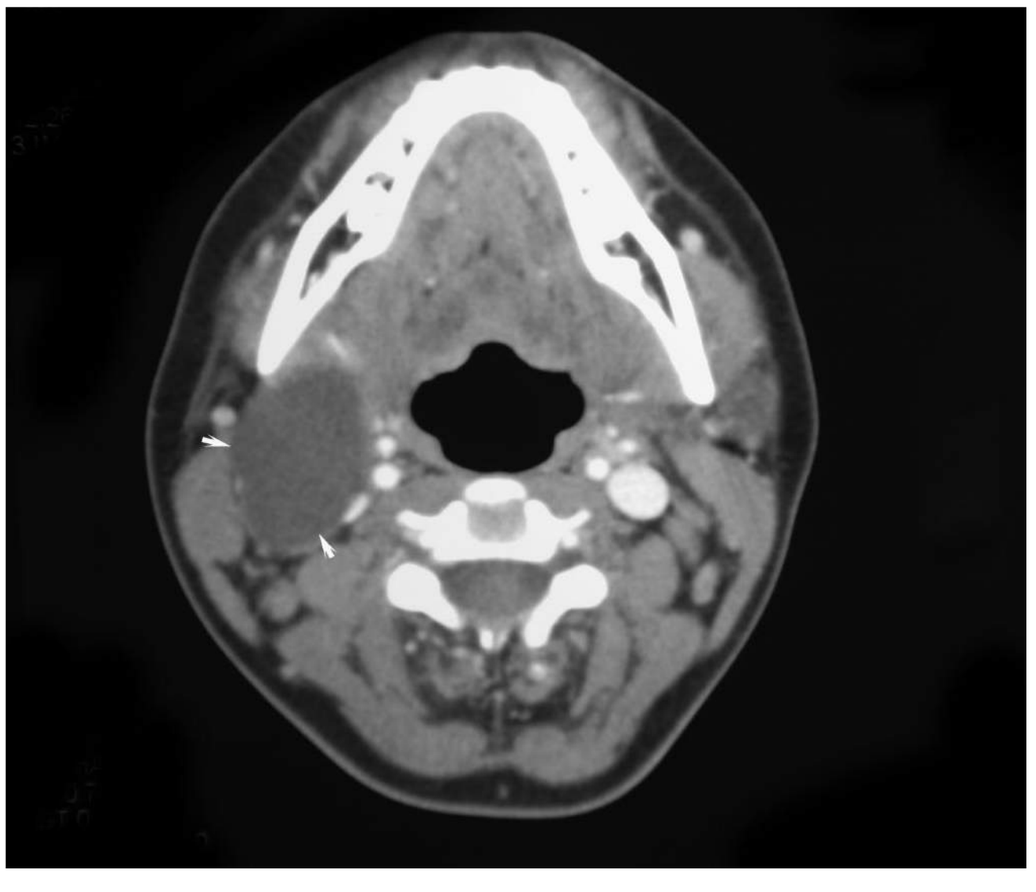


FIGURE 14.70 ■ **Second Branchial Cleft Cyst.** Neck CT showing second branchial cleft cyst. (Photo contributor: Scott Manning, MD.)

THYROGLOSSAL DUCT CYST

Clinical Summary

A thyroglossal duct cyst arises from the incomplete obliteration of the thyroglossal duct during fetal development. It usually presents as a painless, midline, anterior neck mass that moves with swallowing and protrusion of the tongue. It may occur anywhere from the base of the tongue to the sternal notch but is usually located at or below the hyoid bone adjacent to the thyrohyoid membrane. These cysts may rapidly enlarge if infected, which often occurs in association with upper respiratory symptoms.

Management and Disposition

The diagnosis of this lesion is suggested by history and physical examination. Antibiotics are indicated if the lesion has rapidly enlarged due to infection. Common pathogens include H influenzae, S aureus, and Staphylococcus epidermidis. Treatment involves excision of the cyst and midportion of the hyoid bone if involved (Sistrunk procedure) following resolution of any associated infection. Referral to an otolaryngologist is appropriate.

Pearls

1. A thyroglossal duct cyst is the most frequent congenital head and neck lesion in children.
2. In approximately 1% of patients with this lesion, the only functional thyroid tissue is located within the cyst. Therefore, patients should be screened by history for symptoms of hypothyroidism. If symptoms are present, a serum thyroid-stimulating hormone (TSH) should be sent, and ultrasound of the midline neck should be considered prior to surgical removal.
3. The cyst typically moves up with protrusion of the tongue or with swallowing due to its relationship with the hyoid bone and larynx.

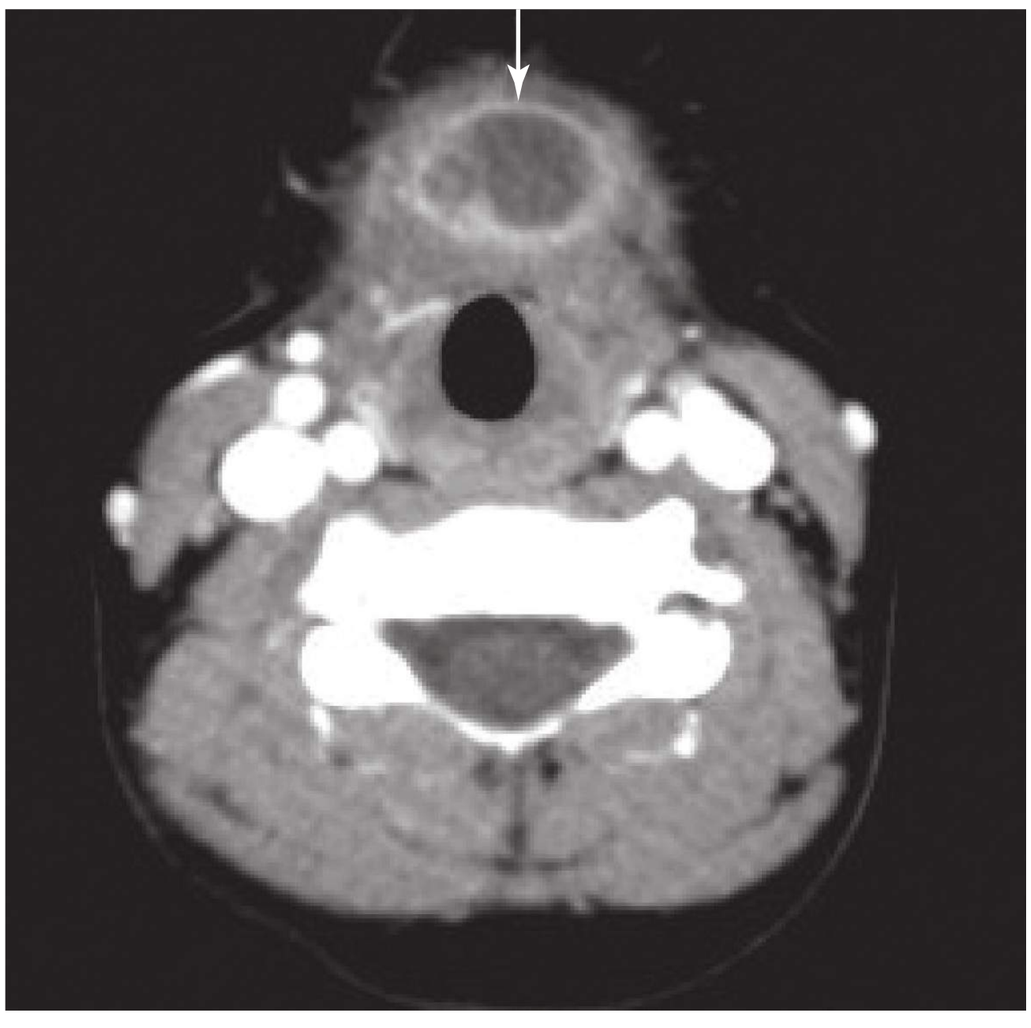


FIGURE 14.72 ■ **Thyroglossal Duct Cyst.** CT of the neck showing anterior thyroglossal duct cyst. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 14.71 ■ **Thyroglossal Duct Cyst.** A midline mass is seen in thyroglossal duct cyst. (Photo contributor: Lawrence B. Stack, MD.)

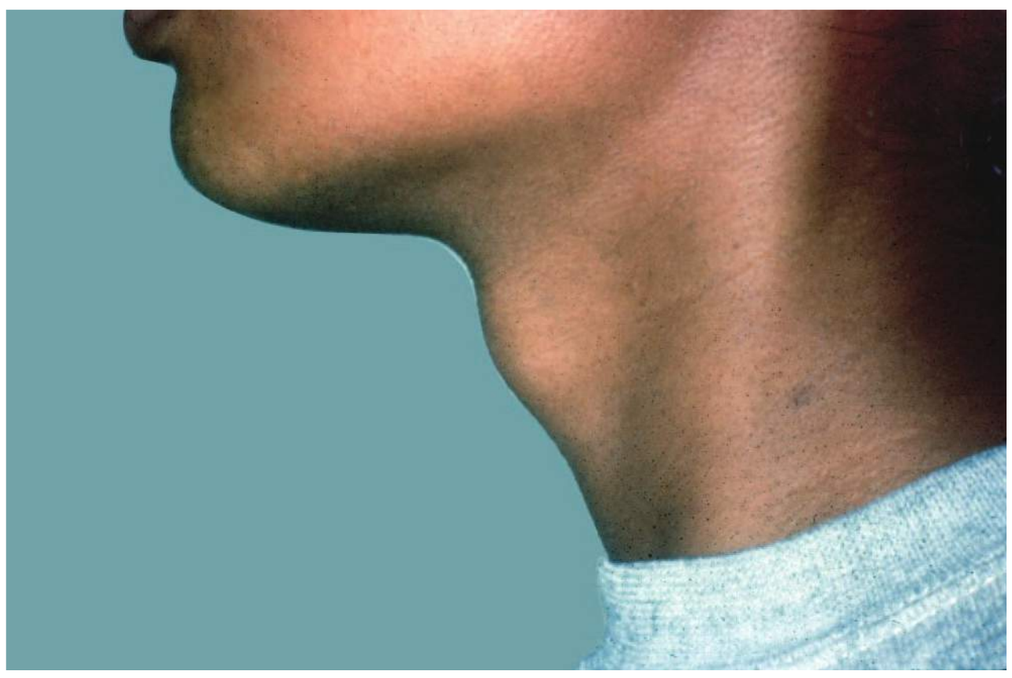


FIGURE 14.73 ■ **Thyroglossal Duct Cyst.** Lateral view of thyroglossal duct cyst. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Cystic hygromas are congenital lymphatic malformations found most commonly in the neck in infants and children less than 2 years of age. They present as nontender, compressible, unilocular, or multilocular masses with thin, transparent walls filled with straw-colored fluid. Unlike hemangiomas, these lesions rarely undergo spontaneous involution. The vast majority tend to grow and infiltrate adjacent structures. In cases where the tongue is involved, they may lead to upper airway obstruction. The differential diagnosis includes branchial arch remnants, thyroglossal duct cysts, cystic teratomas, cervical lymphadenopathy, and other primary neoplastic diseases.

Elective surgical removal is the treatment of choice in the vast majority of cases, since these lesions do not regress and may affect local tissues. Extent of the lesion should be evaluated prior to its removal (CT). The earlier these lesions can be removed, the better the cosmetic result. Aspiration of the lesion, with or without injection of a sclerosing agent, is another therapeutic option.

Pearls

1. Rapid enlargement of a cystic hygroma is most likely due to bleeding or infection. This can result in airway compromise.
2. Chromosomal abnormalities are found in a significant number of infants with cystic hygromas. These lesions are frequently associated with Noonan, Turner, and Down syndromes.

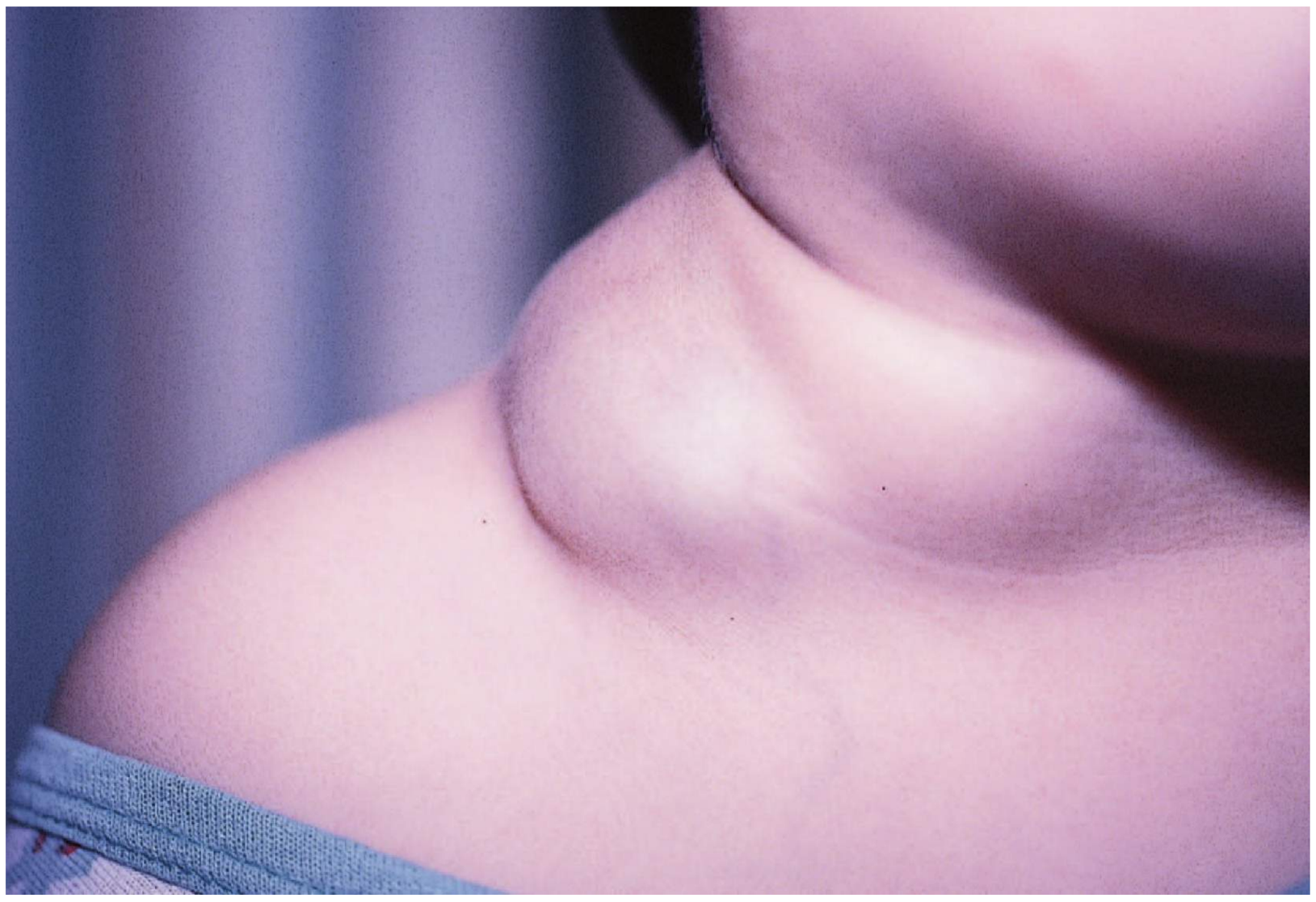


FIGURE 14.74 ■ **Cystic Hygroma.** A bright, supraclavicular, soft, boggy, compressible mass consistent with cystic hygroma. (Photo contributor: Richard M. Ruddy, MD.)

CAT-SCRATCH DISEASE

Clinical Summary

Cat-scratch disease is a benign, self-limited condition caused primarily by Bartonella henselae that usually manifests with regional lymphadenopathy, though visceral organ, neurologic, or ocular involvement can occur. A history of contact with or scratch from a cat (especially kittens with fleas) is usually present. Cat-scratch disease typically starts with an inoculation lesion, which sequentially will appear vesicular, erythematous, and then papular. Lymphadenopathy usually appears within 2 weeks of skin inoculation and may persist for months. In rare cases, patients may develop complications such as encephalitis, osteolytic lesions, hepatosplenic lesions, weight loss, prolonged fever, and fatigue. The differential diagnosis includes lymphogranuloma venereum, bacterial adenitis, sarcoidosis, infectious mononucleosis, tumors (benign or malignant), tuberculosis, tularemia, brucellosis, and histoplasmosis.

Management and Disposition

The disease is usually self-limited and management is primarily symptomatic. Parents and patients should be reassured that the nodes are benign and frequently resolve within 2 to 4 months. Though the evidence is inconclusive, there appears to be a benefit to treating immunocompetent patients with 5 days of azithromycin. Other options include clarithromycin, trimethoprim-sulfamethoxazole, rifampin, or ciprofloxacin. Patients with hepatosplenic, neurologic, or neuroretinal disease should be treated with parenteral antibiotics (rifampin plus gentamicin or azithromycin). If the diagnosis is in doubt, serologic assays for Bartonella species can be sent. Surgical excision of the affected nodes is generally unnecessary.

Pearls

1. Cat-scratch disease is the most common cause of regional adenopathy and should be considered in all children or adolescents with persistent lymphadenopathy.
2. Parinaud oculoglandular syndrome is characterized by a unilateral conjunctivitis and preauricular lymphadenopathy caused by B henselae.
3. Even in the presence of severe and multiple hepatic lesions, liver transaminase levels are normal and hepatomegaly is rare.
4. B henselae is a rare cause of unilateral neuroretinitis and vision loss.
5. In cases treated with antibiotics, a Jarisch-Herxheimer reaction may occur which manifests as fever, tachycardia, hyperventilation, hypotension, peripheral vasodilation, diffuse myalgias, and exacerbation of skin lesions.



FIGURE 14.75 ■ **Cat-scratch Disease.** An erythematous, tender, suppurative node is seen in a young febrile patient with a history of cat scratch on the extremity. The node required drainage 2 days later. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 14.76 ■ **Cat-scratch Disease.** The precipitating wound that caused the suppurative node in Fig. 14.75. (Photo contributor: Kevin J. Knoop, MD, MS.)

Clinical Summary

Epiglottitis (AKA supraglottitis) is a life-threatening condition characterized by sudden onset of fever, irritability, sore throat, moderate to severe respiratory distress with stridor, and variable degrees of drooling. It results from a cellulitis of the epiglottis, aryepiglottic folds, and adjacent tissues. The patient generally appears toxic and prefers a sitting position, leaning forward with the neck extended in a sniffing position with an open mouth. With the addition of the H influenzae type B vaccine to the routine immunization schedule, there has been a dramatic decrease in the incidence of epiglottitis as well as a shift in the bacterial etiology. Though H influenzae type B is still the most common cause, many cases are now caused by nontypeable H influenzae, streptococci, staphylococci (especially MRSA), and Candida albicans. Adults typically have a more indolent course characterized by severe sore throat and odynophagia. Direct thermal injury has been reported as a noninfectious cause. On soft tissue lateral neck x-ray, the epiglottis is seen as rounded and blurred (thumbprint sign). Epiglottitis may progress to complete upper airway obstruction if not treated. Differential diagnosis includes acute infectious laryngitis, acute laryngotracheobronchitis (croup), acute spasmodic laryngitis, membranous (bacterial) tracheitis, anaphylactic reaction, foreign-body aspiration, retropharyngeal abscess (RPA), and extrinsic or intrinsic compression of the airway (tumors, trauma, cysts).

Management and Disposition

Immediate intervention is required. If epiglottitis is suspected, the child should be allowed to remain in a position of comfort if they are maintaining an adequate airway. If impending respiratory failure is present, an airway must be established. If possible, this should be done in the operating room or designated area where advanced airway management with sedation is available. An experienced anesthesiologist and surgeon should be readily available in case a surgical airway is necessary. Once the airway has been controlled, the patient should be sedated to avoid unplanned extubation. Parenteral antibiotic therapy should be initiated with ceftriaxone or cefotaxime. Antistaphylococcal coverage against MRSA with clindamycin or vancomycin is also indicated, and therapy can be adjusted once culture results are available.

Pearls

1. Definitive diagnosis of epiglottitis requires direct visualization of a red, swollen epiglottis, preferably in an operating room with advanced airway measures readily available.
2. Allow the child to remain undisturbed in a position of comfort while preparing for airway management. An agitated child is at increased risk for sudden, complete upper airway obstruction.
3. H influenzae type B can still cause epiglottis even in immunized children.



FIGURE 14.77 ■ **Epiglottitis.** Lateral soft-tissue x-ray of the neck demonstrating thickening of aryepiglottic folds and thumbprint sign of epiglottis. (Photo contributor: Richard M. Ruddy, MD.)

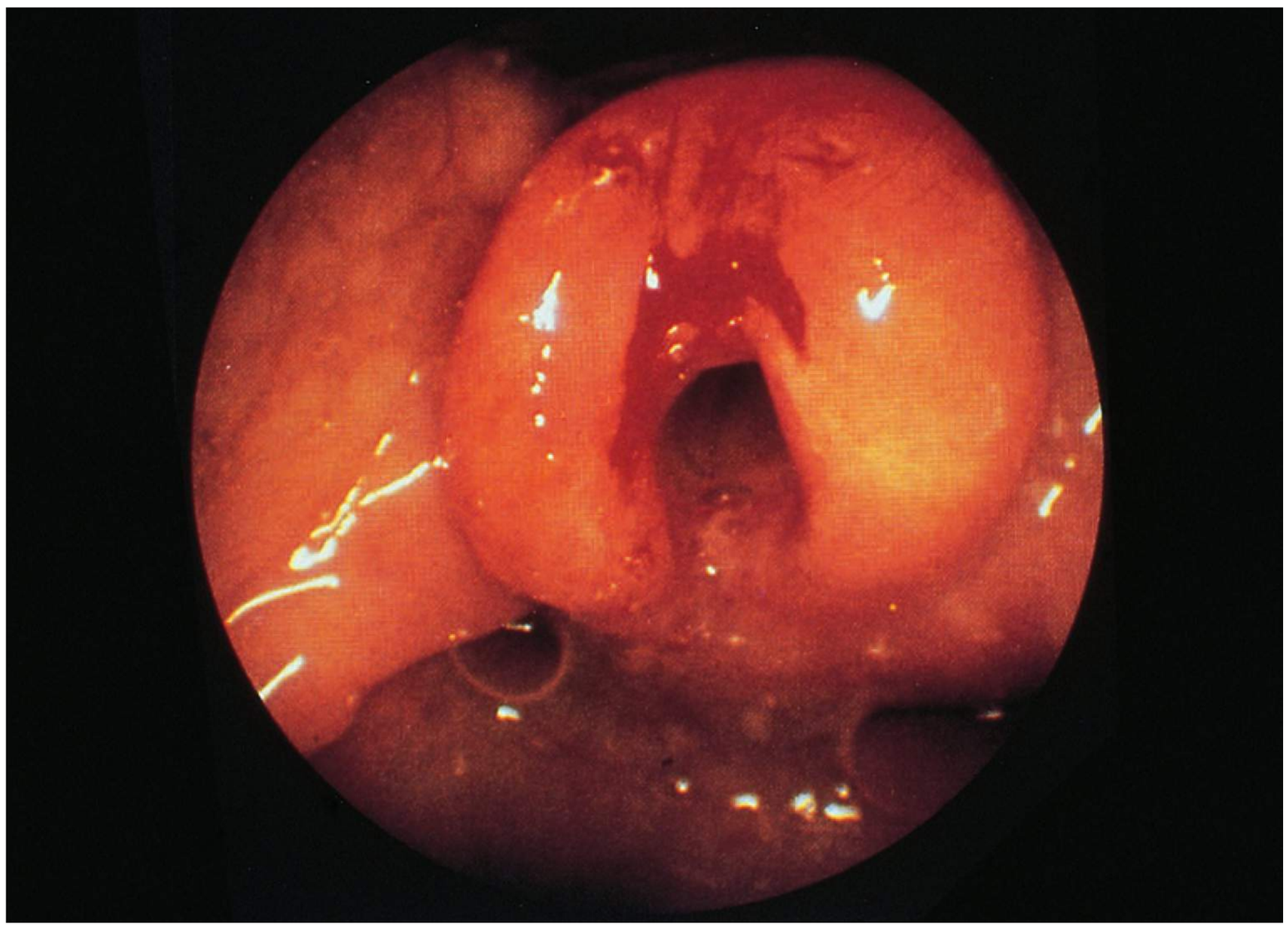


FIGURE 14.78 ■ **Epiglottitis.** The same patient immediately after extubation. Although erythema and some edema persist, the airway is widely patent. (Photo contributor: Department of Otolaryngology, Cincinnati Children's Hospital Medical Center, Cincinnati, OH.)

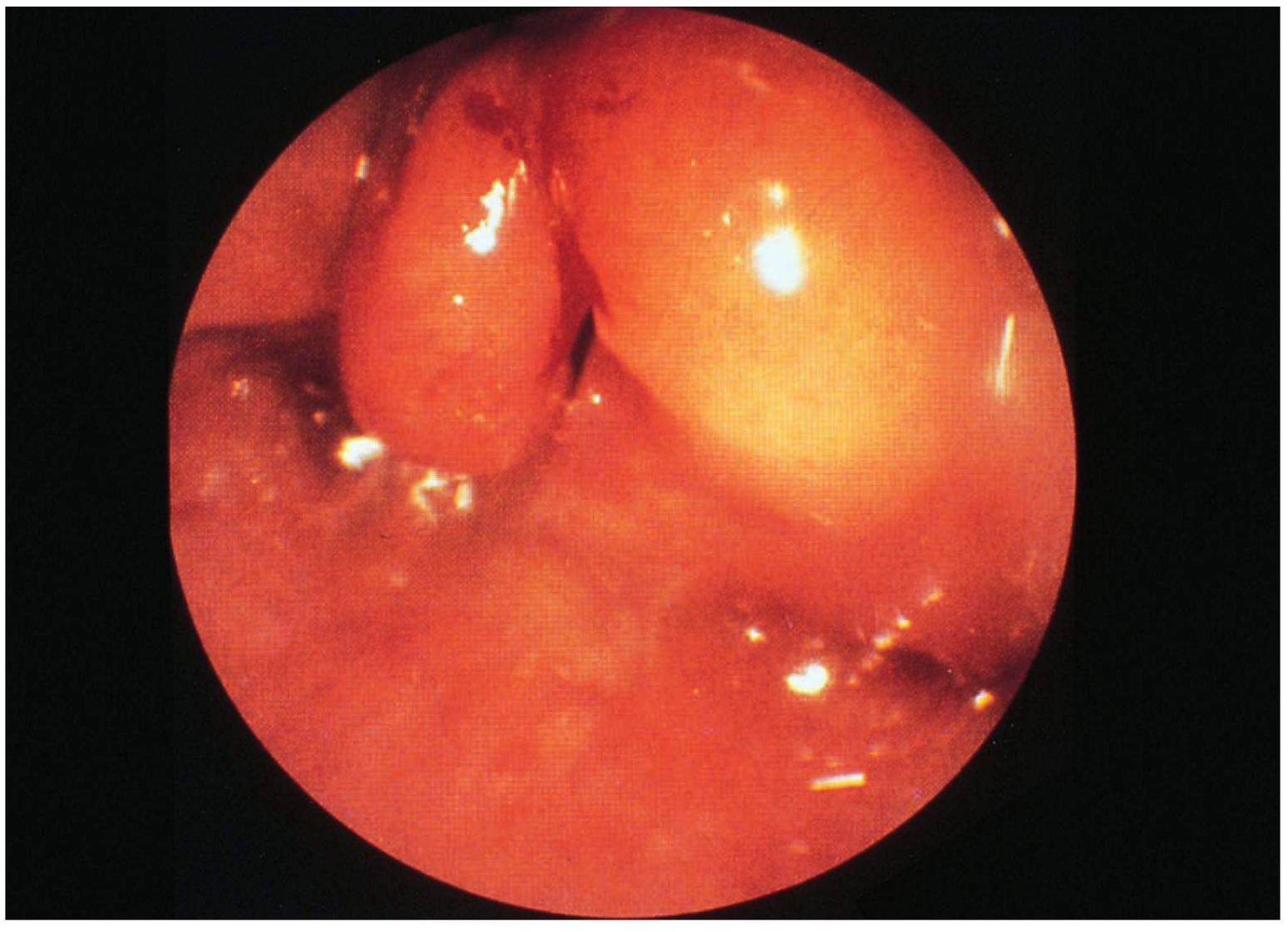


FIGURE 14.79 ■ **Epiglottitis.** Endoscopic view of almost complete airway obstruction secondary to epiglottitis. Note the slit-like opening of the airway. (Photo contributor: Department of Otolaryngology, Children's Hospital Medical Center, Cincinnati, OH.)

Clinical Summary

Retropharyngeal abscess (RPA) usually presents with fever, difficulty swallowing, excessive drooling, sore throat, changes in voice, or neck stiffness. Limitation of neck movement on examination, especially with hyperextension, or torticollis will often be seen. The stiff neck may mimic meningitis. The characteristic retropharyngeal edema is a result of cellulitis and suppurative adenitis of the lymph nodes located in the prevertebral fascia. It is seen on a soft tissue lateral x-ray of the neck as prevertebral soft tissue thickening. The RPA may be preceded by an upper respiratory infection, pharyngitis, otitis media, or a wound infection following a penetrating injury to the posterior pharynx. The differential diagnosis includes pharyngitis, acute laryngotracheobronchitis, epiglottitis, membranous (bacterial) tracheitis, cervical adenitis, infectious mononucleosis, peritonsillar abscess, foreign-body aspiration, and diphtheria.

Management and Disposition

RPA requires immediate assessment of the airway with establishment of a definitive airway if physical exam indicates progressive upper airway obstruction. Antibiotic coverage should be initiated immediately (clindamycin or a β-lactamase-resistant penicillin in areas where S aureus remains susceptible to methicillin). Analgesia should be administered as needed. Radiologic evaluation includes soft tissue lateral neck x-ray and neck CT with contrast to define the extent of infection. In the absence of airway obstruction, medical



FIGURE 14.80 ■ **Retropharyngeal Abscess.** This ill-appearing 6-year-old child presented with a several-day history of fever, neck pain, sore throat, cough, and headache. Soft tissue lateral radiography of the neck showed thickened prevertebral tissues opposite C2 to C4. CT showed the airway narrowed to a width of 5 mm within the oropharynx. (Photo contributor: Mark Ralston, MD.)

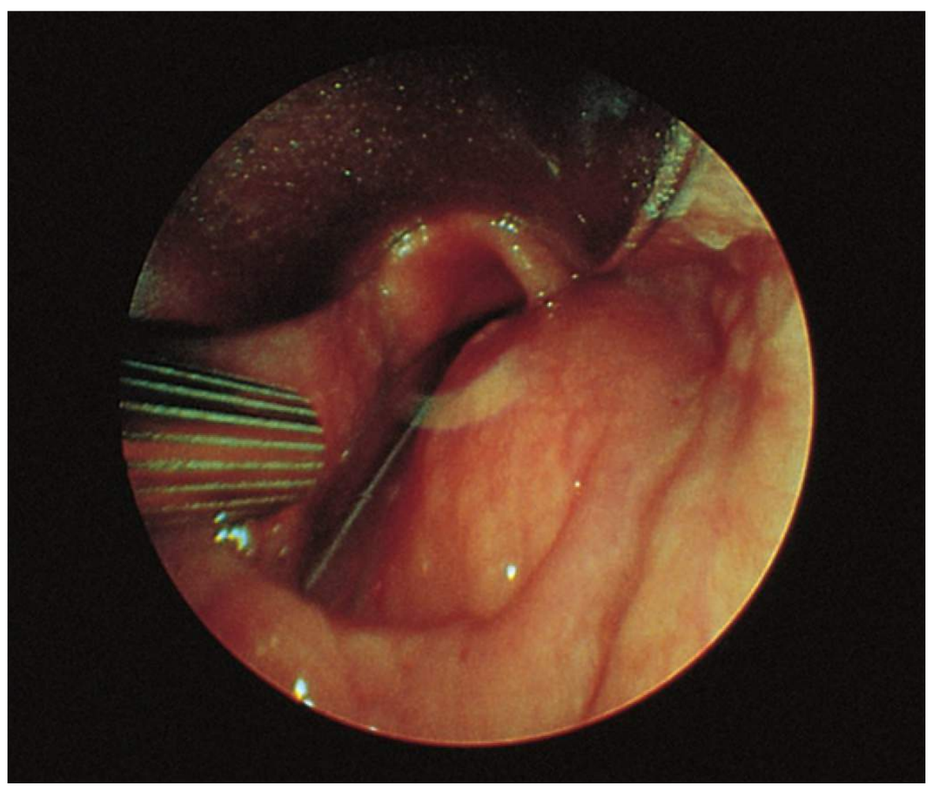


FIGURE 14.81 ■ **Retropharyngeal Abscess.** Endoscopic view of a retropharyngeal abscess. Note the massive swelling posteriorly. (Photo contributor: Department of Otolaryngology, Cincinnati Children's Hospital Medical Center, Cincinnati, OH.)

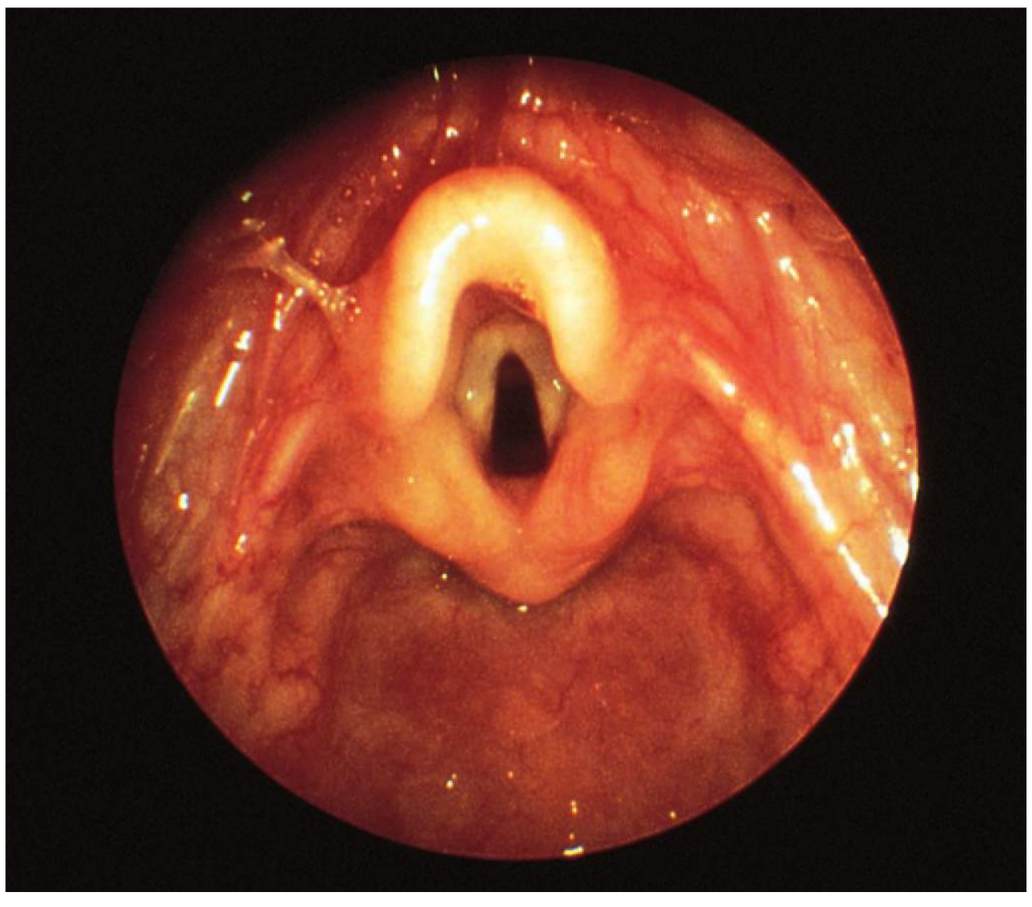
treatment with IV antibiotics for 24 to 48 hours is first-line therapy. If impending obstruction is present or the infection is unresponsive to IV antibiotic therapy, needle aspiration or incision and drainage should be performed in the operating room. These patients require hospitalization and immediate otolaryngologic or surgical consultation.

Pearls

1. On lateral soft tissue x-ray of the neck, the prevertebral soft tissue can measure up to 7 mm in width at the level of C2. At C6, it can measure up to 14 mm in width. This represents approximately one-half the width of the corresponding vertebral body.
2. The prevertebral soft tissue may appear falsely enlarged during neck flexion or crying.
3. The peak incidence occurs in 3- to 5-year-olds. It is rare beyond 6 years of age as the retropharyngeal lymph nodes involute.
4. RPAs in older patients most commonly arise as a complication of trauma or an immunocompromised state.



FIGURE 14.82 ■ **Retropharyngeal Abscess.** Lateral soft tissue neck x-ray demonstrating prevertebral soft tissue density consistent with retropharyngeal abscess. (Photo contributor: Richard M. Ruddy, MD.)



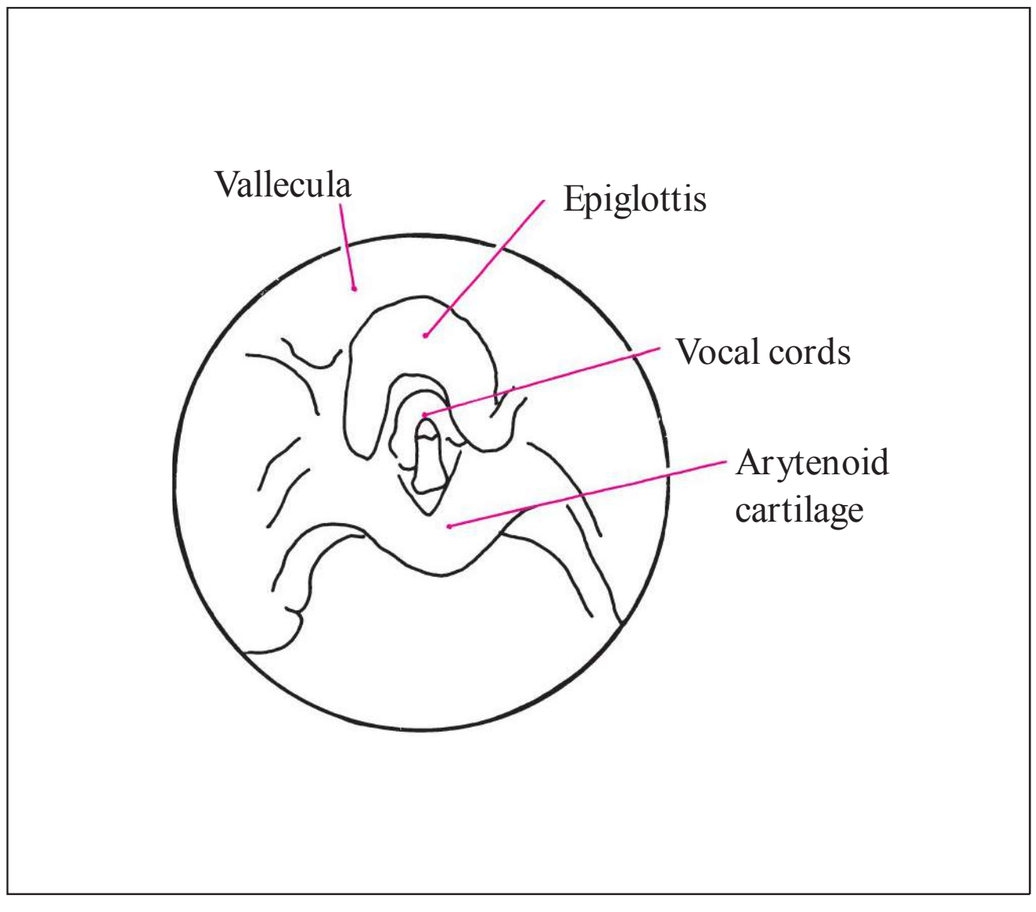


FIGURE 14.83 ■ **Normal Laryngeal Structures.** Endoscopic view of a normal epiglottis and surrounding structures. (Photo contributor: Department of Otolaryngology, Cincinnati Children's Hospital Medical Center, Cincinnati, OH.)

Clinical Summary

The majority of button battery ingestions occur in children less than 6 years of age, peaking between age 1 and 2 years. The most important factors in determining symptoms at presentation, as well as management, are location within the GI tract and duration of contact with the mucosal surface. Batteries that are lodged in the esophagus may be asymptomatic initially or can present with pain, drooling, dysphagia, poor oral intake, cough, vomiting, or fever. Mechanisms of injury associated with button battery ingestion include electrical-current-induced soft tissue injury, liquefactive necrosis resulting from alkali exposure due to battery leakage or the de novo synthesis of alkali at the surface of the battery, and pressure tissue necrosis.

Management and Disposition

Anteroposterior and lateral plain radiographs should be obtained to locate the battery within the GI tract. Button batteries can be distinguished from coins on plain x-ray by demonstration of a double contour. Batteries that are lodged in the esophagus can lead to potentially fatal complications and require immediate removal in consultation with a gastroenterologist or surgeon. This is best accomplished with direct visualization via endoscopy and with a definitive airway in place to prevent aspiration of the battery upon removal from the esophagus. Batteries that have passed into the stomach are likely to traverse the GI tract without complication. However, patients with symptoms of GI injury (pain, obstruction, hematemesis) or those that coingested a magnet are candidates for endoscopic removal. X-rays can be repeated at weekly intervals until passage is documented. Instructions detailing concerning symptoms (abdominal pain, abdominal distention, hematemesis, or blood in the stools) should be provided to the parents at the time of discharge.

Pearls

1. Button batteries with a diameter of 20 mm or greater that remain in the stomach beyond 48 hours should be considered for removal.
2. Button batteries lodged in the nasal cavity or external auditory canal also require emergent removal to prevent complications such as perforation or stenosis.
3. Heavy metal poisoning due to absorption of button battery components though the GI tract is extremely rare.

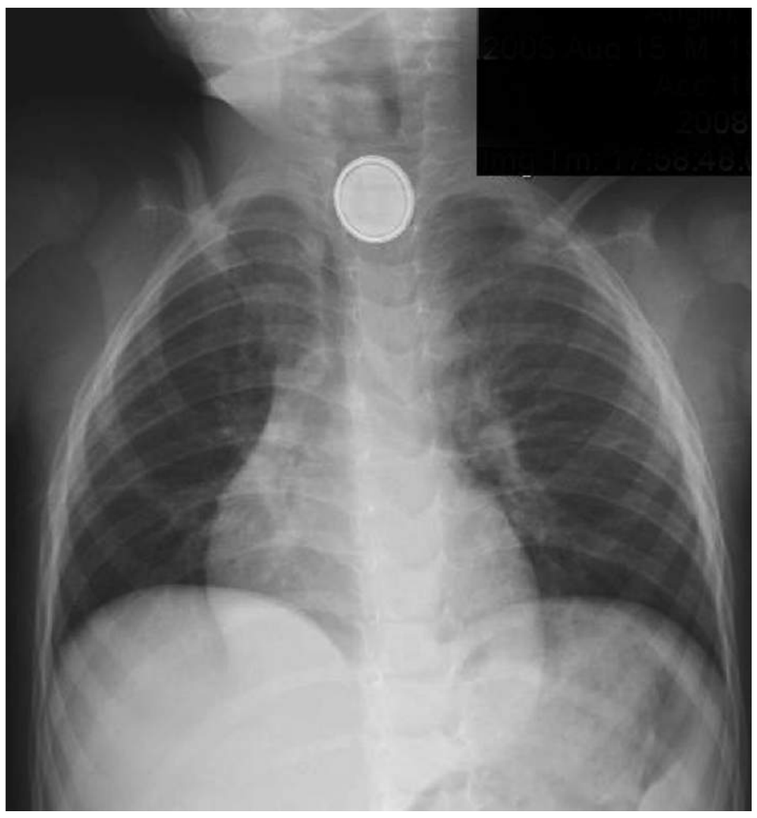


FIGURE 14.84 ■ **Disk Battery Ingestion.** Chest x-ray showing circular "coin-like" appearance with a second concentric ring from the plastic insulating grommet. This identifies the foreign body as a disk battery. (Photo contributor: Scott Manning, MD.)



FIGURE 14.85 ■ **Disk Battery.** This battery was retrieved after ingestion. (Photo contributor: Lawrence B. Stack, MD.)

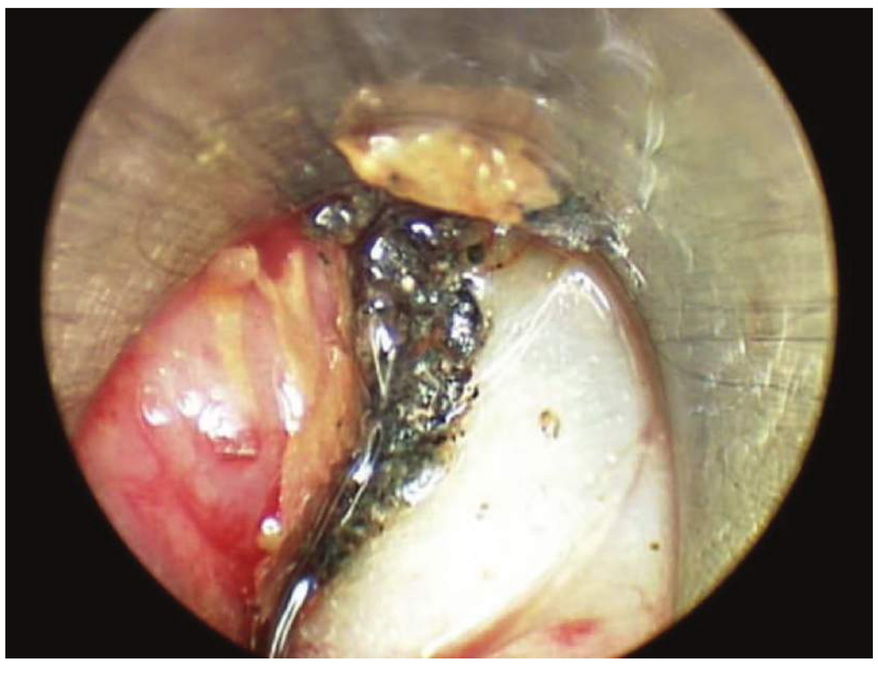


FIGURE 14.86 ■ **Disk Battery Ingestion.** This endoscopic view shows esophageal necrosis after disk battery lodged in the esophagus. (Photo contributor: Scott Manning, MD.)

MEMBRANOUS (BACTERIAL) TRACHEITIS

Clinical Summary

Membranous tracheitis is an acute exudative bacterial infection (S aureus, H influenzae, M catarrhalis, streptococci, and pneumococci) of the upper airway capable of causing life-threatening airway obstruction. It may present as a primary infection or occur as a secondary bacterial complication of a viral infection of the upper respiratory tract. This locally invasive infection of the tracheal mucosa below the vocal cords produces copious purulent secretions. The exudate can form a thick plug that may ultimately lead to an acute tracheal obstruction. Patients appear toxic, with high fever and a croup-like syndrome that can progress rapidly. The characteristic "membranes" may be seen on x-rays of the airway as edema with an irregular border of the subglottic tracheal mucosa. On direct laryngoscopy, profuse purulent secretions can be found in the presence of a normal epiglottis. The differential diagnosis includes acute laryngotracheobronchitis, RPA, epiglottitis, peritonsillar abscess, foreign-body aspiration, and acute diphtheric laryngitis.

Management and Disposition

Otolaryngologic consultation should be obtained as soon as the diagnosis is considered. Direct visualization of the trachea is more important than pursuing a radiologic diagnosis. Aggressive airway management, including endotracheal intubation, may be needed to protect the airway and allow for repeated suctioning to prevent acute airway obstruction. The patient should be admitted to the intensive care unit for close monitoring. Parenteral antibiotic coverage against suspected organisms should be instituted immediately.

Pearls

1. Bacterial tracheitis often presents with acute, severe airway obstruction after a short prodrome. It should be suspected in all patients with an atypical croup-like presentation: unusual age group, toxic appearance, not improving with routine croup therapy, and unusual appearance of the tracheal lumen on plain radiographs.
2. In bacterial tracheitis, up to 50% of soft tissue films may delineate a subglottic membrane.



FIGURE 14.87 ■ **Membranous Tracheitis.** Lateral soft tissue x-ray of the neck in a 13-year-old girl with the acute onset of stridor after 3 days of sore throat. Membranes (arrows) are visible in the subglottic region. (Photo contributor: Matthew R. Mittiga, MD.)

Clinical Summary

The nose is the most common site of aerodigestive tract foreign bodies in children. Most children present with a history of witnessed or suspected foreign-body insertion and are symptom free. However, one quarter are discovered incidentally, with no preceding history by the caregiver. Common symptoms include mucopurulent discharge, foul odor, epistaxis, pain, and nasal obstruction. Objects most often include inorganic material (beads or small toys) and food. Unilateral foul-smelling discharge suggests organic or porous material (paper, sponge, foam rubber) or the long-standing presence of a foreign body leading to a localized inflammatory reaction.

On examination, foreign bodies are typically lodged anteriorly on the floor of the nasal passage under the inferior turbinate or superiorly in front of the middle turbinate. Diagnosis is through direct visualization, though high foreign bodies may preclude visual diagnosis even with a head lamp or otoscope. Most are radiolucent, and unless you suspect a concomitant airway or GI foreign body, plain films are unnecessary. Button batteries and paired magnets can be especially dangerous due to the risk of local tissue compromise and should be removed promptly. Otherwise, removal can be an elective procedure. Techniques for removal include the use of positive pressure through the mouth with simultaneous occlusion of the unaffected nares ("mother's kiss maneuver"), removal with alligator forceps, a day hook (right-angle hook), or via a balloon catheter device threaded past the foreign body with subsequent balloon inflation and device retraction (Katz extractor). Children should be adequately prepared and properly immobilized. The value of an adept holder cannot be underestimated. The differential diagnosis includes allergic rhinitis, upper respiratory infection, acute sinusitis, cerebrospinal fluid leak, and nasopharyngeal polyp or mass.

Management and Disposition

Most foreign bodies can be successfully removed in the emergency department. After removal, always inspect for trauma and the presence of additional foreign bodies. Otolaryngology consultation or referral is warranted for poorly visualized posterior foreign bodies, chronic or impacted objects with significant inflammation, button batteries, penetrating or hooked foreign bodies, or any foreign body that cannot be removed due to poor cooperation or limited resources. After successful removal, patients can be safely discharged with instructions to return with any persistent, unilateral nasal discharge, recalcitrant epistaxis, or new fevers. Patients with minor bleeding after removal can be treated with a vasoconstricting nasal spray like oxymetazoline for no longer than 3 days.

Pearls

1. Organic materials that may expand with exposure to moisture (such as dried beans) require urgent removal.
2. A button battery can lead to septal perforation in as little as 4 hours!
3. There is little concern for migration of inert foreign bodies into the airway in children with intact protective reflexes.

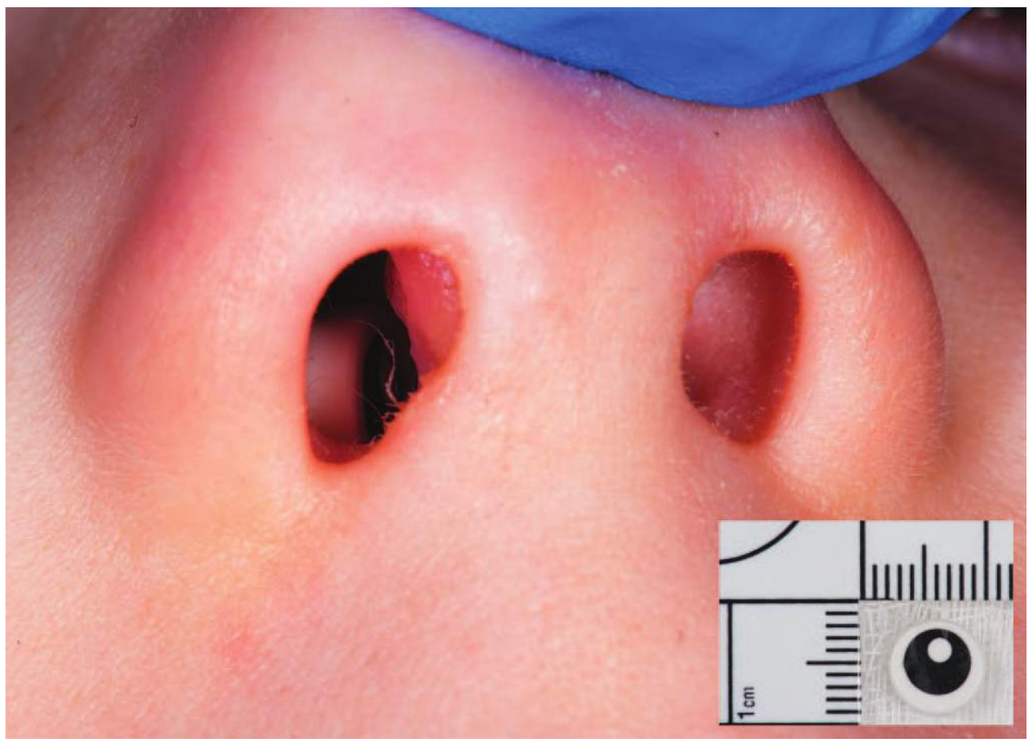


FIGURE 14.88 ■ **Nasal Foreign Body.** A round circular bead (insert) is embedded in the right nostril. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 14.89 ■ **Nasal Foreign Body.** Purulent drainage from the left nostril due to a candy wrapper which had been in place for 2 weeks. (Photo contributor: Larry B. Mellick, MD, MS, FAAP, FACEP.)

PHYSEAL FRACTURES: SALTER-HARRIS CLASSIFICATION

Clinical Summary

There are several fracture patterns that are unique to children. Physeal fractures, torus (buckle) fractures, greenstick fractures, and bowing fractures or deformities all occur secondary to the physiologic differences between immature and mature bone. Physis (growth plate) fractures are common because of the relative weakness of the growth plate compared to the cortical lamellar bone. This weakness is related to the relatively large ratio of cells to matrix in the physis. The Salter-Harris (SH) classification was designed to describe each type of physeal fracture, its prognosis, and its treatment.

SH type I: Fracture that extends only through the growth plate. It is a difficult radiologic diagnosis since the fracture line is not visible unless the fracture is displaced. It is usually a clinical diagnosis marked by pain at the location of the physis, but occasionally a physeal widening is observed on the x-ray.

SH type II: Fracture along the growth plate with an oblique extension through a piece of the metaphysis. This is the most common growth plate fracture.

SH type III: Fracture through the growth plate that extends into the epiphysis and articular cartilage into the joint space.

SH type IV: Fracture through the growth plate that extends into both the metaphysis and the epiphysis and into the joint space.

SH type V: Crushing injury resulting in compression of the growth plate.

Management and Disposition

Immobilization by splinting or casting is the treatment of choice in types I and II (minimum of 3 weeks). In these cases, reduction is easy to achieve and maintain. Growth is generally unimpaired. Types III and IV may require open reduction to avoid later traumatic arthritis and, in some cases, growth arrest. Type V fractures are rare and require very close orthopedic follow-up because of arrest of growth caused by the death of the germinal cells. Types III, IV, and V require immediate orthopedic consultation.

Pearls

1. After initial assessment and evaluation, always suspect SH type I if there is evidence of tenderness and swelling around the growth plate area despite negative x-rays.
2. Comparison views of the unaffected side may be useful in diagnosing SH types I and V.
3. Types I and II generally possess a substantial potential for remodeling and have a very good prognosis. Types III and IV threaten growth potential and articular integrity and require anatomic (often surgical) reduction. Type V is very high risk for growth arrest.

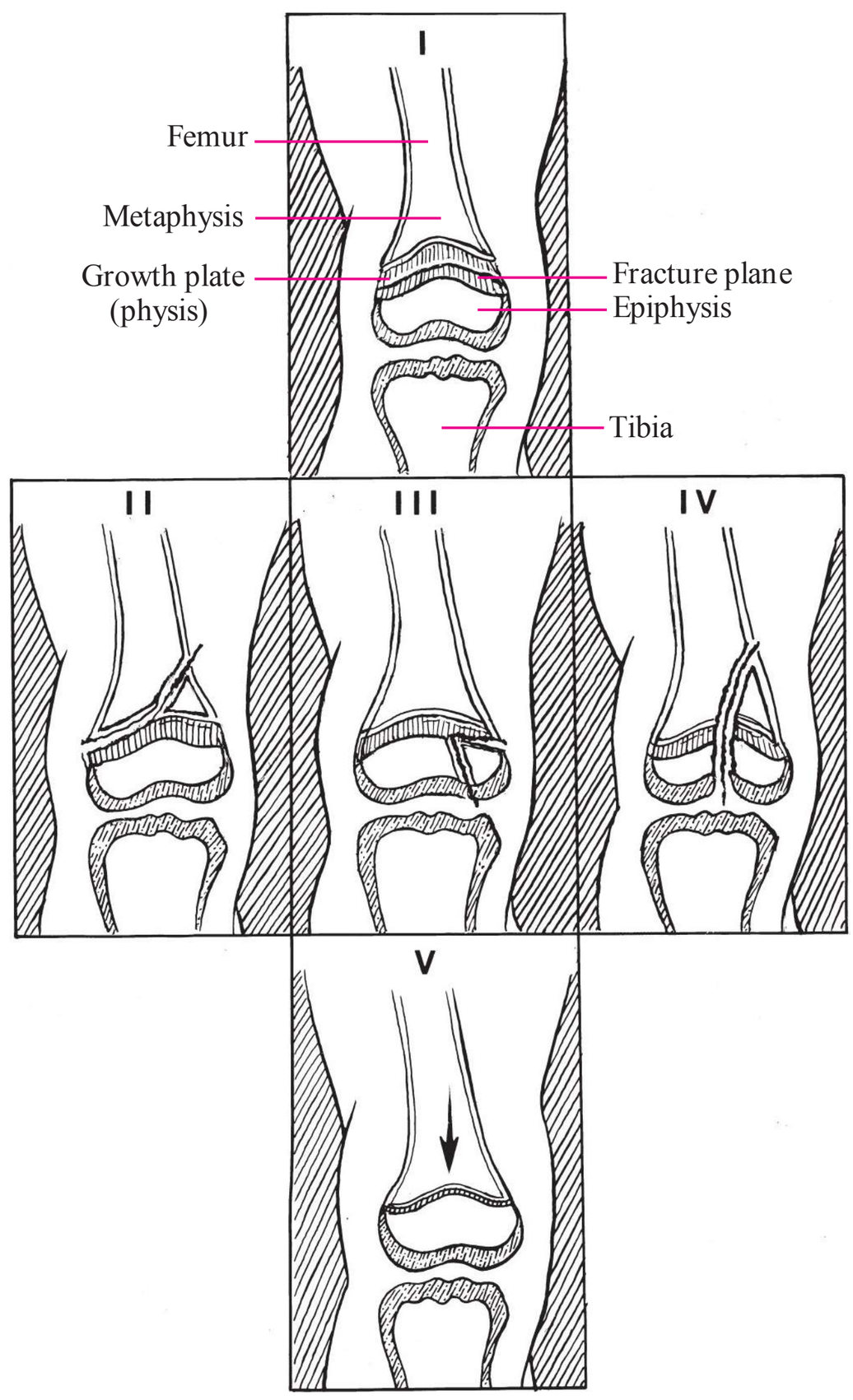


FIGURE 14.90 ■ **Salter-Harris Fracture Classification.** Salter-Harris classification for physeal (growth plate) fractures.

Clinical Summary

The toddler's fracture is a nondisplaced oblique or "spiral" fracture of the mid to distal tibial in a child who has just begun to bear weight. The typical age range is 9 months to 3 years. The clinical history is often nondescript and may include a fall or injury with a twisting mechanism of the lower extremity. Many children will limp or outright refuse to bear weight on the affected leg. Point tenderness over the distal third of the tibia can often be elicited with a careful exam.

Management and Disposition

Obtain anteroposterior and lateral films of the tibia and fibula. The child with a toddler's fracture should be immobilized in a long leg posterior splint or cast. If clinical suspicion remains high for toddler's fracture based on history (refusal to bear weight) or exam (point tenderness over the distal third of the tibia), a splint should be applied even if the initial radiographs are negative. X-rays obtained 10 to 14 days after the initial evaluation may show periosteal reaction or sclerosis. Most toddler's fractures heal without secondary sequelae. The differential diagnosis includes other lower extremity fractures including those associated with nonaccidental trauma, toxic synovitis, septic hip, bony tumors, and osteomyelitis.

Pearls

1. A child who outright refuses to bear weight despite a "negative" radiograph should be treated presumptively for a fracture. A faint hairline fracture may not be apparent on initial x-rays.
2. A toddler's fracture would not be expected if the child cannot yet support their own weight in a standing posture. Spiral fractures of any lower extremity long bone in a nonambulatory child should raise concern for child abuse.
3. Children often walk or crawl on splints leading to an increased risk of pressure sores. Be sure to pad the splint liberally and impress the importance of non–weight bearing on the splint and early orthopedic follow-up for casting.



FIGURE 14.91 ■ **Toddler's Fracture.** Anteroposterior radiograph of the tibia and fibula shows an oblique (spiral) fracture lucency in the distal tibial diaphysis. (Photo contributor: Alexander Towbin, MD.)

DACTYLITIS (HAND-FOOT SYNDROME)

Clinical Summary

This painful vaso-occlusive condition is commonly the first clinical manifestation of sickle cell disease in infants. It usually presents in children younger than 5 years of age. Patients are acutely ill with fever, leukocytosis, and swollen hands and/or feet that are exquisitely painful due to bone infarction despite initially normal x-rays. It is not until 1 to 2 weeks later that subperiosteal new bone, cortical thickening, and even complete bone destruction can be seen. The differential diagnosis includes osteomyelitis, trauma, cold injuries, acute rheumatic fever, juvenile rheumatoid arthritis, and leukemia.

Management and Disposition

Treatment of vaso-occlusive crises in sickle cell disease centers around providing adequate fluid balance, oxygenation, and analgesia. Therapy should be individualized. Codeine, hydromorphone, morphine, and ketorolac are analgesic agents commonly used in the treatment of children with painful sickle crises. If fever is present, bacterial infection should be assumed until proven otherwise. CBC, reticulocyte count, and blood cultures should be obtained from all febrile sickle cell patients. If the patient is afebrile, you can omit the blood culture. Empiric broad-spectrum antibiotic coverage should be instituted immediately (third-generation cephalosporin). In cases of dactylitis, very close follow-up is necessary not only for the management of sickle cell disease but also to reevaluate the radiologic changes in the small bones of the hands and feet. In most instances, the previously described changes disappear; however, rarely, shortening of the fingers and toes may occur due to significant infarct.

Pearls

1. Most clinical manifestations of sickle cell disease occur after the first 5 to 6 months of life. Hemolytic anemia gradually develops over the first 2 to 4 months (changes that follow the replacement of fetal hemoglobin by hemoglobin S) and leads to the clinical syndromes associated with an increased percentage of abnormal SS hemoglobin.
2. Sickle cell patients are at high risk for infection from encapsulated organisms due to their functional asplenia. The most common organisms causing osteomyelitis in sickle cell patients are Salmonella sp, S aureus, and S pneumoniae.
3. Dactylitis can often be differentiated from osteomyelitis based on the symmetrical involvement of multiple bones and a negative blood culture.
4. Dactylitis, severe anemia, and leukocytosis within the first 2 years of life increase the risk for adverse sickle cell–related outcomes by age 10 years (death, stroke, frequent pain crises, or acute chest syndrome).



FIGURE 14.92 ■ **Acute Sickle Dactylitis.** Bilateral cylindrical swelling of soft tissue of the hands in sickle cell disease consistent with vaso-occlusive crisis or dactylitis. (Photo contributor: Donald L. Rucknagel, MD, PhD.)

Clinical Summary

A single strand of hair or thread may encircle a finger, toe, or the penis, leading to circumferential strangulation of the appendage. Children in their first year of life are particularly at risk from inadvertent encirclement by a parent's hair or loose thread especially within confining clothing. The affected digit appears edematous, erythematous, and painful. If not corrected, vascular compromise or infection can ensue. The differential diagnosis includes insect bites, trauma, cellulitis of the digit, osteomyelitis, or ainhum (dactylosis spontanea), a painful constriction in the base of the fifth toe of unknown etiology followed by spontaneous autoamputation months to years later.

Management and Disposition

Visualization of the constricting material may be difficult. Edema, erythema, and periarticular skin folds may hide the hair or thread. It is imperative to carefully retract the skin around the proximal aspect of the edematous appendage. A magnifying lens may be helpful in identifying the band. Since the removal can be painful, consider a digital or penile block prior to removal. A topical anesthetic may also be applied to assist in reducing local pain. Using a small hemostat, grasp a portion of the material, cut it with a surgical blade, and unwind it. Occasionally, chemical hair removers have been used to dissolve the hair. This is not advisable on deeply cut skin and will be ineffective for synthetic fibers. Elevation of the involved digit after removal of the constricting agent provides resolution of the edema and erythema within 2 to 3 days. In some cases, the digit's blood supply may have been irreversibly compromised. Subspecialty consultation should be considered whenever neurovascular integrity is in question or if the constricting band cannot be visualized and removed.

Pearls

1. In the vast majority of cases, a clear line of demarcation can be identified between the normal tissue and the affected area.
2. Fussiness or irritability may be the only presenting symptom. Examination for hair tourniquets should be included in the evaluation of any inconsolable infant.
3. There may be an insidious onset with reepithelialization over the hair tourniquet, leaving it difficult to visualize.



FIGURE 14.93 ■ **Hair Tourniquet.** A strand of hair has encircled the middle toe in two places, causing erythema and swelling. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 14.94 ■ **Hair Tourniquet.** Erythema distal to the hair tourniquet of the affected toes due to strangulation by the tourniquet. (Photo contributor: Robert W. Hickey, MD.)



FIGURE 14.95 ■ **Hair Tourniquet.** Erythema and edema from ischemia due to a hair tourniquet. (Photo contributor: David Effron, MD.)

FAILURE TO THRIVE

Clinical Summary

Failure to thrive (FTT) is the inability to maintain a normal growth pattern in weight, stature, and occasionally in head growth. Definitions are varied and include a fall in weight below the second percentile relative to age or growth deceleration that crosses two major percentiles on a standardized growth chart. It is most common in infancy, and the condition is nonorganic (50%), organic (25%), or mixed (25%) in etiology. The diagnosis is made after complete history and physical examination with comparison of the measurements of length (supine in children <3 years of age), weight, and head circumference (maximal occipital-frontal circumference) to standard measurements. In cases of deficient caloric intake or malabsorption, the patient's head circumference is normal and the weight is reduced out of proportion to length/height. The differential diagnosis of FTT is lengthy. Nonorganic disorders include poor feeding technique, disturbed maternal-child interaction, emotional deprivation, inadequate caloric intake, and child neglect. Organic causes are numerous.



FIGURE 14.96 ■ **Failure to Thrive.** This infant has not been able to maintain a normal growth pattern. Note skin folds in upper extremities due to loss of subcutaneous fat. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Depending on history, physical findings, the social situation, and ability to ensure close monitoring by a primary care physician, most cases can be managed as outpatients. The primary care provider can assist in determining whether outpatient management is indicated. If the diagnosis of FTT is made in the emergency department, admission is suggested to complete the evaluation. That the child presents to the emergency department could be the only indicator of a poor social environment or inadequate access to medical care. Simple testing including CBC, urinalysis, electrolytes, BUN, creatinine, and lead testing is appropriate. More specific testing should be directed by findings in the history and physical examination. Early involvement of social services may facilitate the evaluation and follow-up. Treatment will vary according to the underlying disorder and often involves a team approach, employing the assistance of nutritionists, social services, and home health workers.

Pearls

1. FTT in neglected children is accompanied by signs of developmental delays, emotional deprivation, apathy, poor hygiene, withdrawn behavior, and poor eye contact.
2. The major contributor to FTT is caloric inadequacy. Dietary history should include details of formula preparation, volume consumed, and, in toddlers, the volume of juice consumed.
3. In severely malnourished children, consider assessing for rickets by measuring calcium, phosphorous, albumin, and alkaline phosphatase.



FIGURE 14.97 ■ **Failure to Thrive.** Accentuation of the gluteal folds secondary to loss of subcutaneous fat in an infant with FTT. (Photo contributor: Andrew H. Urbach, MD.)

Clinical Summary

The etiology of dental caries is multifactorial with an interplay between microflora (plaque colonized with Streptococcus mutans), substrate (fermentable carbohydrates from breast milk, formula, or juice), and host (saliva and teeth). Nursing or milk bottle caries result from prolonged and frequent night time breastfeeding or sleeping with a bottle containing milk or sugar-containing juices. The sugars are fermented by the bacteria in plaque, lowering the pH in the mouth and resulting in demineralization of the tooth enamel. The condition generally occurs before 18 months of age and is more prevalent in medically underserved children. Upper central incisors are most commonly involved. Outpatient dental referral is indicated.

Management and Disposition

Parental education and timely referral to a dentist is necessary to prevent complications. If untreated, the caries may destroy the teeth and spread to contiguous tissues. These patients have a high risk for microbial invasion of the pulp and alveolar bone with the subsequent development of a dental abscess and facial cellulitis. In these cases, aggressive treatment with antibiotics (amoxicillin) and pain control, with prompt dental referral for definitive care, is necessary.

Pearls

1. The role of the emergency department physician is to recognize this pattern of dental decay (upper incisors most commonly) and initiate dental referral and parental education.
2. Nursing or milk bottle caries tends to spare the lower front teeth because of the shielding of the lip and tongue and the increased exposure to saliva from the sublingual glands that washes away cariogenic substrates.
3. The newborn mouth is generally devoid of microorganisms. Newborns and infants are colonized with S mutans from parents and family members. Education on avoidance of sharing utensils and cups may help delay colonization of infants.



FIGURE 14.98 ■ **Nursing Bottle Caries.** Extensive tooth decay from sleeping with bottle containing milk or sugar-containing juices. (Photo contributor: Lawrence B. Stack, MD.)

NURSEMAID'S ELBOW (RADIAL HEAD SUBLUXATION)

Clinical Summary

Nursemaid's elbow is a condition that occurs commonly in children younger than 6 years of age who are usually picked up or pulled by the extended, pronated arm. The peak incidence is 2 to 3 years of age. These children present unwilling to supinate or pronate the hand on the affected side. Generally they hold the affected arm close to their side in a passive pronation with partial flexion at the elbow. Radiographic studies should be considered only in patients with an unusual mechanism of injury, significant bony tenderness, or those who do not become rapidly asymptomatic after the reduction maneuver. The differential diagnosis includes radial head fracture or complete dislocation, posterior elbow dislocation, condylar and supracondylar fractures of the distal humerus, or buckle fracture of the radius or ulna.

Management and Disposition

Carefully palpate all points of the affected arm for tenderness. There should be minimal to no pain with palpation. Orthopedic consultation is generally not indicated unless an underlying fracture is diagnosed. Reduction is usually achieved by one of two maneuvers:

1. Flexion/supination. Beginning with the elbow flexed at 90 degrees, the wrist of the affected arm is grasped with one hand, while the other stabilizes the elbow. The forearm is then gently supinated while the arm is flexed until the palm approaches the anterior shoulder.
2. Hyperpronation. While holding the elbow in extension, hyperpronation of the forearm is maintained until reduction is achieved.

In either maneuver, a palpable click over the radial head is evident upon successful reduction. Studies have indicated that hyperpronation is more likely to be successful when used as the initial reduction maneuver. The patient usually begins using the arm normally within minutes. When the injury has been present for several hours, reduction may be difficult, and it may take several hours to recover full function of the elbow.

Pearls

1. Radiographs of radial head subluxation typically appear normal.
2. Immobilization after reduction is not necessary.
3. If the patient remains symptomatic after reduction attempts, obtain x-rays to assess for fractures.
4. Up to one-third of children will have recurrence, but rarely beyond age 4 to 5 years.

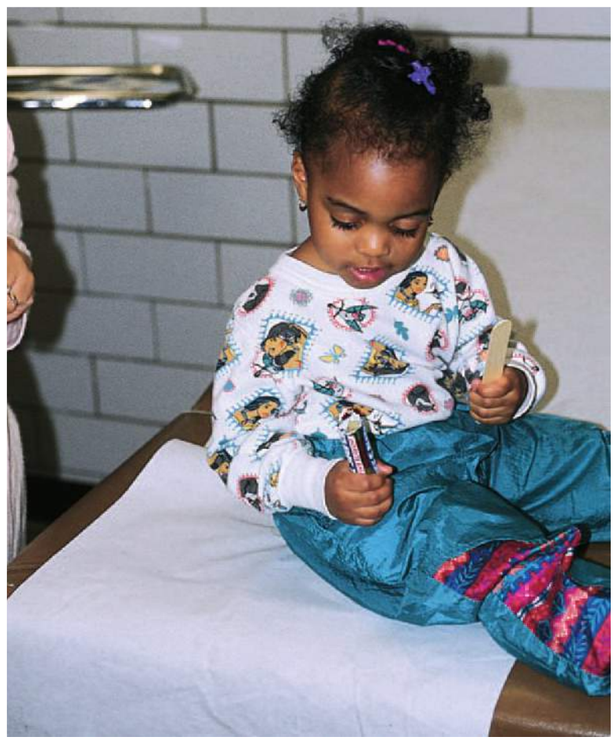


FIGURE 14.99 ■ **Nursemaid's Elbow Reduction.** Simultaneous supination with elbow flexion technique for reduction of nursemaid's elbow. (Photo contributor: R. Jason Thurman, MD.)





FIGURE 14.100 ■ **Nursemaid's Elbow.** This child presents with pseudoparalysis of the right arm after a pulling injury. Note how she avoids use of the affected arm and preferentially uses the other arm. (Photo contributor: Kevin J. Knoop, MD, MS.)



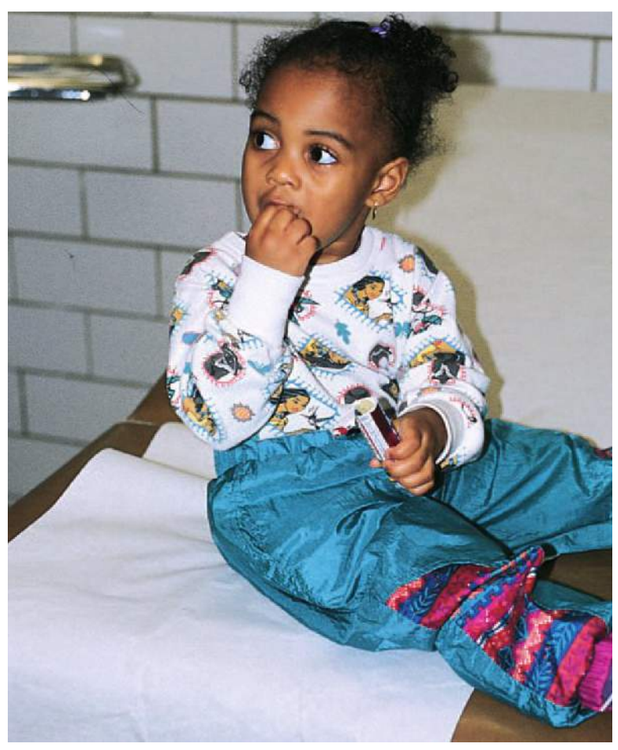


FIGURE 14.101 ■ **Nursemaid's Elbow—Reduced.** After reduction, there is initial reluctance to use the injured arm. With distraction and encouragement, the patient demonstrates successful use of the extremity. (Photo contributor: Kevin J. Knoop, MD, MS.)

ILEOCOLIC INTUSSUSCEPTION

Clinical Summary

Intussusception occurs when one segment of bowel invaginates into itself, most commonly at the ileocecal junction. It is the most common abdominal emergency and cause of intestinal obstruction in children less than 2 years of age. The precise cause is unknown in 75% of cases, though the incidence increases during seasonal viral gastroenteritis outbreaks. Adenovirus, bacterial enteric infections, Meckel diverticula, tumors, hematomas, and vascular malformations are thought to be potential lead points.

Patients typically present with sudden onset of intermittent severe episodes of cramping abdominal pain with inconsolable crying and drawing the legs up to the abdomen. Episodes usually occur at 20-minute intervals, but become more frequent as the illness progresses. Vomiting may follow painful episodes and can eventually become bilious. Initially, children appear normal between episodes, but progressive lethargy eventually develops. Bloody stools (occult or gross), a sign of intestinal ischemia, are seen in 70% of patients. A sausage-shaped mass may be palpated in the right upper quadrant. The differential diagnosis includes viral gastroenteritis, constipation, HPS, intestinal malrotation with volvulus, appendicitis, and meningoencephalitis.



FIGURE 14.102 ■ **Ileocolic Intussusception.** Bloody "currant jelly" stools in a hypersomnolent 3-month-old female infant with transverse colon intussusception reduced by air enema. (Photo contributor: Donald H. Arnold, MD, MPH.)

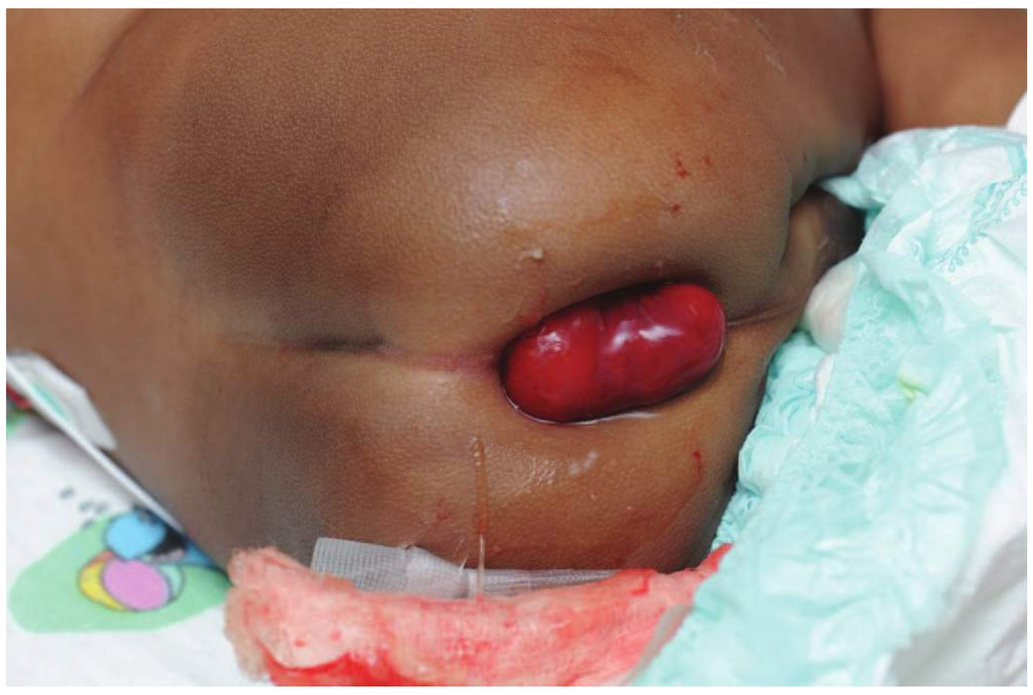


FIGURE 14.104 ■ **Rectal Prolapse.** Protrusion of the sigmoid colon is seen in this infant. (Photo contributor: Lawrence B. Stack, MD.)

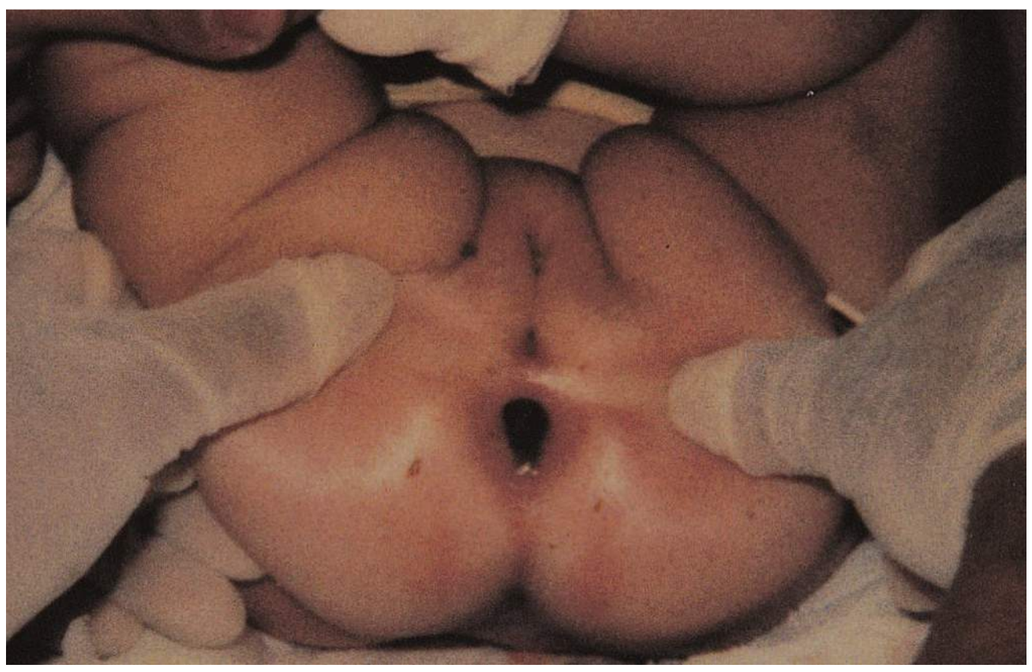


FIGURE 14.103 ■ **Intussusception.** The apex of the intussusception may extend through the anus mimicking rectal prolapse. It is distinguished from rectal prolapse by the separation between the protruding intestine and the rectal wall. (Photo contributor: Binita R. Shah, MD. Used with permission from Shah B, Lucchesi M, Amodio J, Silverberg M. Atlas of Pediatric Emergency Medicine. 2nd ed. New York, NY: McGraw-Hill; 2013: Fig. 10-3, p. 379.)

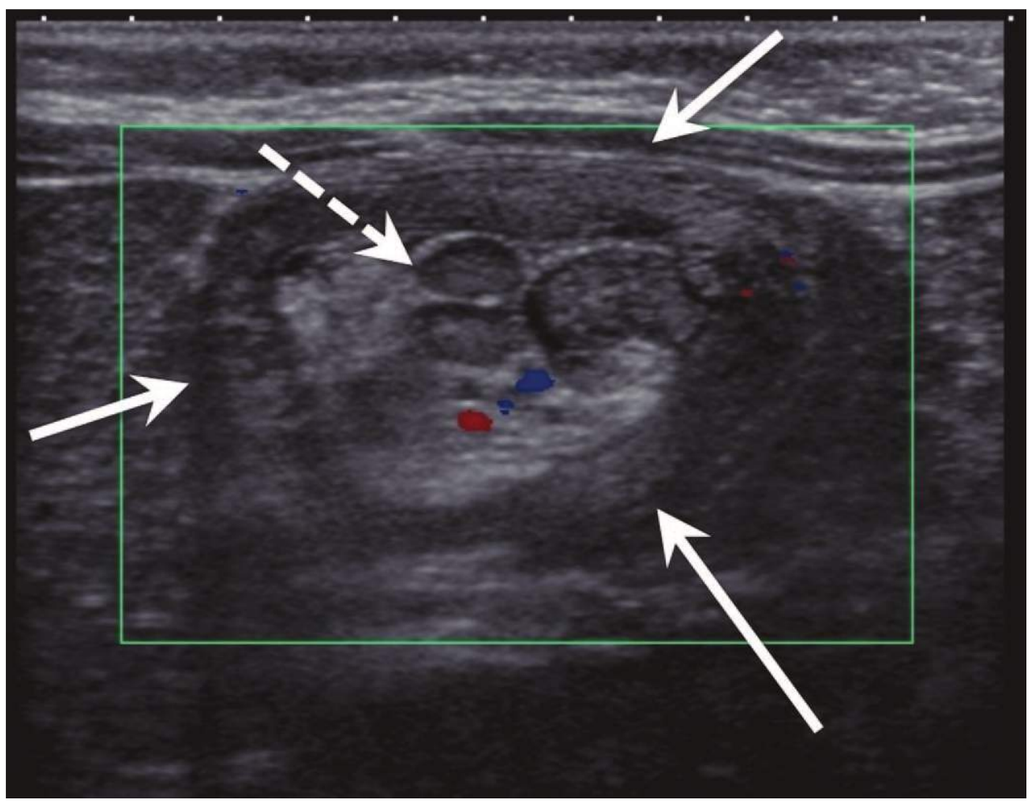


FIGURE 14.105 ■ **Ileocolic Intussusception.** Ultrasound showing intussuscepiens (arrows) containing echogenic fat and multiple lymph nodes (dashed arrow). (Photo contributor: Alexander Towbin, MD.)

Management and Disposition

If the diagnosis is uncertain, ultrasound is the imaging test of choice often demonstrating the classic "coiled spring" or "bull's eye" lesion. Plain x-rays obtained early in undifferentiated cases of abdominal pain may show a target or crescent sign, along with a paucity of cecal gas. Treatment begins with nonoperative reduction, using air enema under fluoroscopic or sonographic guidance utilizing pneumatic pressure to reduce the intussusception. The success rate is 80% to 95%. The risk of perforation is <1%. Observe patients for 12 to 24 hours following reduction. Seek surgical consultation, as laparoscopy is indicated when nonoperative reduction is unsuccessful or incomplete.

Pearls

1. Currant jelly stools are seen in only 20% of patients and are a combination of blood and mucous.
2. The classic triad of pain, palpable mass, and bloody stools is seen in less than 15%.
3. Infants can present with profound lethargy alone, with one-third having no apparent abdominal pain. Consider intussusception in any infant with altered mental status.

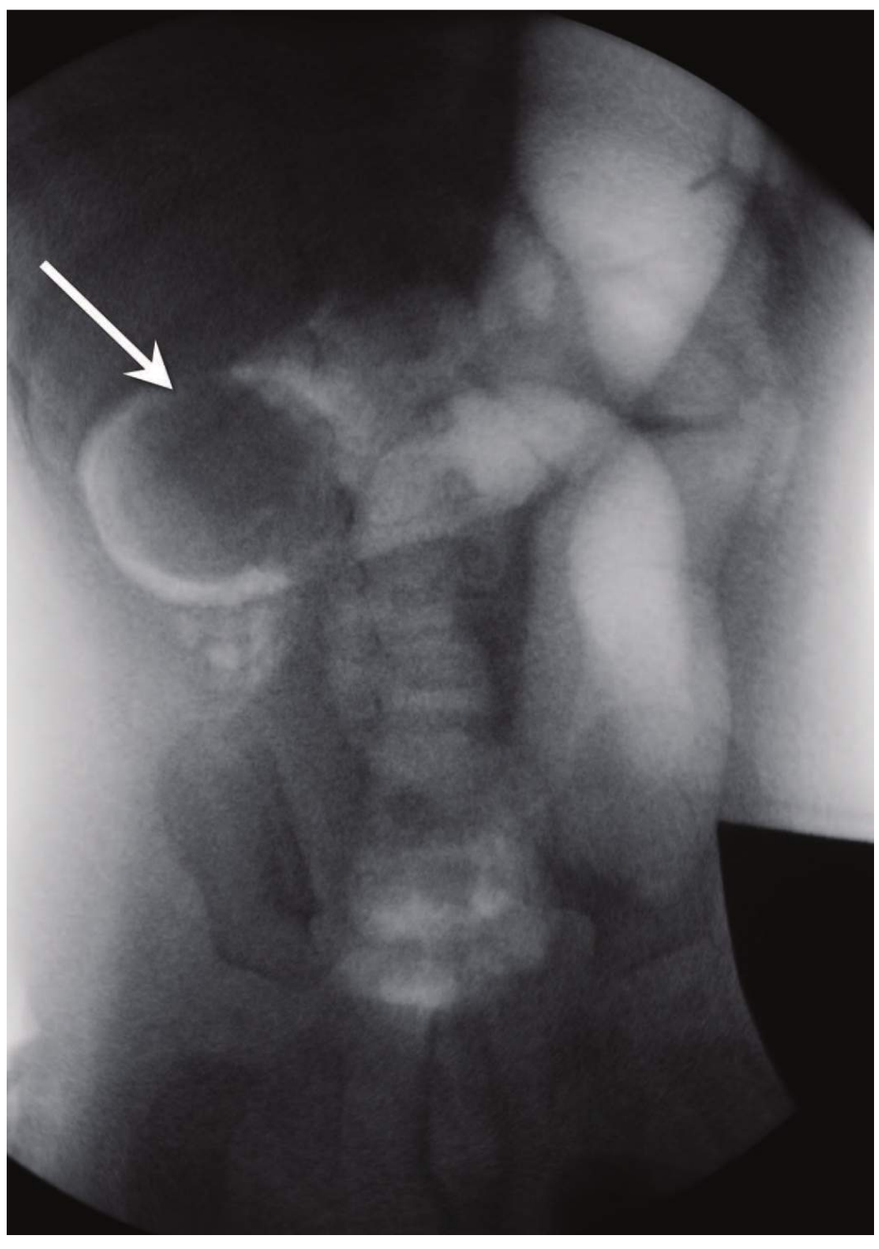


FIGURE 14.106 ■ **Intussusception reduction via air contrast enema part 1.** Air distending the rectum and colon just before reduction. The intussusception mass (arrow) is in the right upper quadrant near the hepatic flexure. (Photo contributor: Alexander Towbin, MD.)

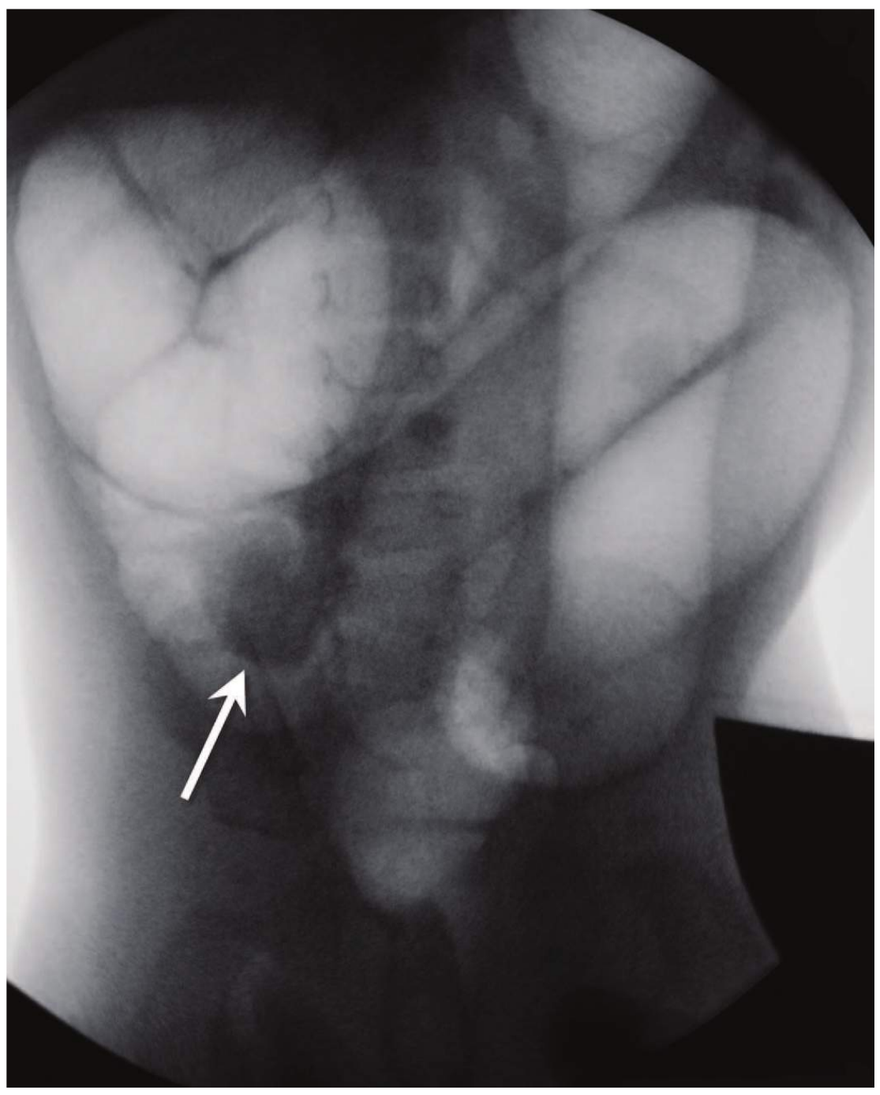


FIGURE 14.107 ■ **Intussusception reduction via air contrast enema part 2.** Reduction of the intussusception (arrow) to the level of the ileocecal valve. (Photo contributor: Alexander Towbin, MD.)



FIGURE 14.108 ■ **Intussusception reduction via air contrast enema part 3.** The intussusception has been reduced. There is free reflux of air into the small bowel. (Photo contributor: Alexander Towbin, MD.)

Clinical Summary

Inguinal hernias are common in childhood with an incidence as high as 5%. Premature infants have an even higher incidence, and boys are approximately 10 times more likely than girls to develop an inguinal hernia. There are two types of inguinal hernias: indirect (common) and direct (rare). Indirect inguinal hernias result from failure of the processus vaginalis to obliterate toward the end of fetal development. With a patent processus vaginalis, the intra-abdominal viscera can protrude through the internal inguinal ring. Indirect inguinal hernias are more common on the right and present as a bulge in the groin by parental history or on physical examination. Maneuvers that increase intra-abdominal pressure, such as crying in an infant or blowing bubbles in an older child, may make the hernia easier to visualize. Associated symptoms such as vomiting, abdominal distention, constipation, blood in the stool, lethargy, or irritability suggest incarceration or strangulation of the hernia. Incarceration is most common in the first year of life. The differential diagnosis includes hydrocele, inguinal lymphadenopathy, testicular torsion, torsion of the appendix testis/epididymis, epididymitis/orchitis, and a retractile testicle.

Management and Disposition

With the history of scrotal swelling but a normal physical examination, refer to a pediatric surgeon for timely evaluation and repair. If a hernia is palpable, ensure that it can be easily reduced manually. Reduction of an incarcerated inguinal

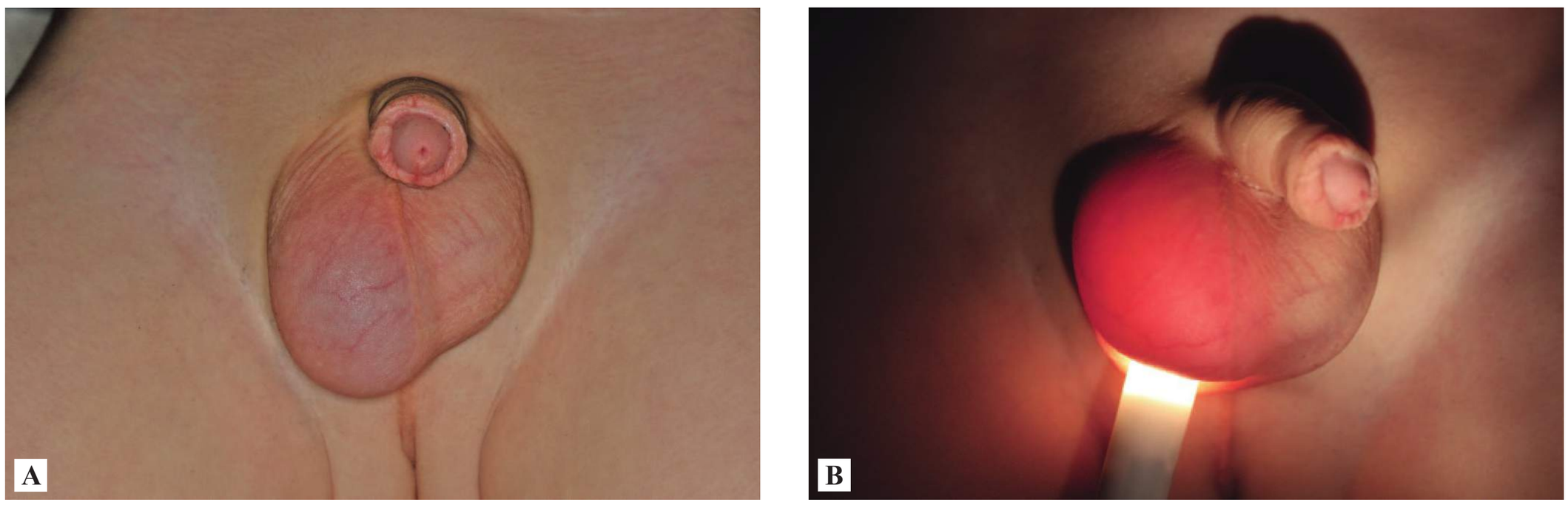


FIGURE 14.109 ■ **Hydrocele.** Painless scrotal swelling (**A**) which transilluminates (**B**). This patient had an inguinal hernia which was repaired electively. (Photo contributor Kevin J. Knoop, MD, MS.)

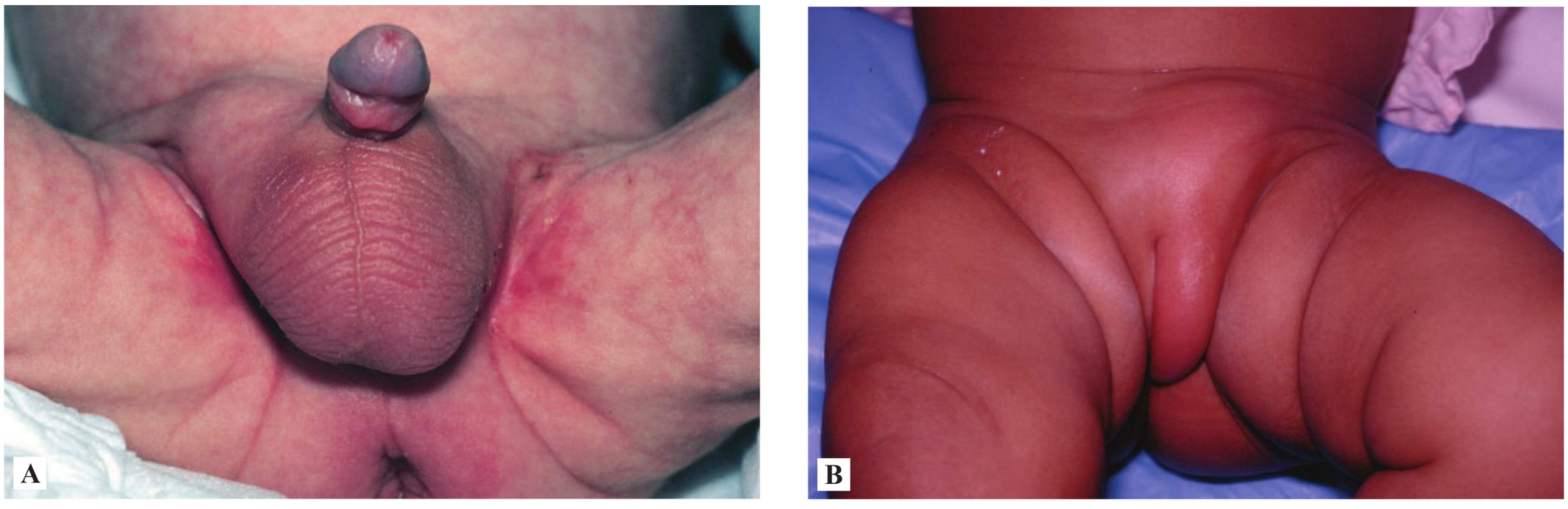


FIGURE 14.110 ■ **Inguinal Hernia.** (**A**)Right inguinal hernia in a male infant (**B**) Left inguinal hernia in a female infant. (Photo contributor: Lawrence B. Stack, MD.)

hernia may be facilitated by adequate pain control and sedation (80% successful). The reduction technique consists of gentle traction inferiorly on the hernia sac with pressure from above to straighten the inguinal canal. There is a high rate of early recurrence of incarceration. Admission to the hospital with repair in 24 to 48 hours after the associated edema has subsided is advisable. Immediate surgical consultation for operative repair is indicated for any incarcerated hernia that cannot be manually reduced.

Pearls

1. Transilluminate the scrotum. Hydroceles will transilluminate, whereas hernias will not.
2. The Trendelenburg position may aid in hernia reduction.
3. Palpate both testicles in the scrotum prior to diagnosing an inguinal hernia.
4. Plain abdominal radiographs are unlikely to be helpful in cases of scrotal swelling where the diagnosis is not apparent. Scrotal ultrasound has a diagnostic accuracy of over 90%.

PINWORM INFECTION (ENTEROBIASIS)

Clinical Summary

Enterobius vermicularis is a threadlike white worm that infects the colon and causes intense nocturnal perianal pruritus when the gravid adult female migrates to deposit eggs at night. Female worms measure 8 to 13 mm in length and can be observed moving about the perianal area at night. On rare occasions, this nematode can lead to vulvovaginitis. The diagnosis can be made by direct visualization of the nematode by the parents or by using a piece of transparent adhesive tape and touching it to the perianal area upon awakening in the morning. This tape is then applied to a glass slide for microscopic examination under low power to look for eggs. The differential diagnosis includes perianal irritation, cellulitis, fissures, hemorrhoids, and contact dermatitis.

Management and Disposition

The treatment of choice is the anthelmintic agent, albendazole. The initial single dose is repeated in 2 weeks to treat secondary Enterobius hatchings. Because of the high frequency of reinfection, families should be treated as a group.

Pearls

1. Reinfection from other infected individuals (daycare cohorts) or autoinfection is necessary to maintain enterobiasis in the individual, since these nematodes usually die after depositing their eggs in the perianal region. Frequent hand washing may reduce chances of infection as transmission occurs by the fecal-oral route.
2. E vermicularis is the most common intestinal nematode in the United States, affecting 5% to 15% of the population. Many infections are asymptomatic.
3. Suspect pinworm infection in children who present with nocturnal restlessness. These patients are often evaluated for urinary tract infection because the scratching of the perineal area is misinterpreted by the parents and treating clinicians as painful urination.



FIGURE 14.111 ■ **Pinworms.** Multiple tiny pearly-white worms are seen at the anus. (Photo contributor: Lawrence E. Heiskell, MD.)

Clinical Summary

There are three varieties of lice specifically parasitic to humans: Pediculus humanus capitis (head louse), which infests the hair and scalp, Pthirus pubis (crab louse), which infests the pubic hair, and Pediculus humanus corporis (body louse). Affected patients may be asymptomatic or may have pruritus. The diagnosis is made via visual examination whereby crawling lice (nymphs and adults) and eggs (nits) are found on hair shafts. The treatment is topical pediculicides (including permethrin, malathion, benzyl alcohol, spinosad, and topical ivermectin) and wet combing to remove lice and nits. Lindane has an association with neurotoxicity, and its use should be avoided in children. Of note, nits may persist for several weeks following therapy. The differential diagnosis includes pseudonits (2-7 mm hair casts), psoriasis, cutaneous fungal infections, scabies, seborrheic dermatitis, and dandruff.

Management and Disposition

The diagnosis is clinical. Patients should be treated with topical pediculicides and educated on signs of resolution. Treatment failure is associated with noncompliance, reinfestation, and drug resistance. Close contacts and those who share bedding should be treated prophylactically. Head lice generally do not survive for more than 2 days when off of a person. The nits die within 1 week if the temperature close to the scalp is not maintained. Clothing and items (eg, pillows, bedding, or stuffed animals) that cannot be washed should be dry-cleaned or sealed for 2 weeks in a plastic bag.

Pearls

1. Children with head lice do not necessarily need to be excluded from school as many have had lice for several weeks and been attending school prior to diagnosis.
2. Combs and brushes can be disinfested by soaking in hot water (130°F or greater) for 5 to 10 minutes.
3. Though shaving hair is anecdotally an effective treatment, it can have adverse psychological effects.



FIGURE 14.112 ■ **Nits.** Several small white cylindrical nits (louse eggs) are seen clinging to dark hair. (Photo contributor: Larry B. Mellick, MD.)



FIGURE 14.113 ■ **Lice Infestation.** Multiple lice are seen in the hair of this patient. (Photo contributor: David Effron, MD.)

TRICHOTILLOMANIA

Clinical Summary

Trichotillomania is an impulse control disorder, which results in compulsive hair pulling causing noticeable hair loss. Hair is repeatedly twisted around the finger then pulled out, or rubbed until it breaks off. There is an irregular-angled border with short hair of different lengths in the affected area. The eyebrows and eyelashes may also be targeted. True alopecia (complete loss of hair) is not present. Young children, adolescents, and women more commonly are affected. The course is chronic with frequent remissions. It is highly distressing to most patients, causing significant psychological duress.

Management and Disposition

Ask if the patient manipulates the hair to confirm the behavior pattern. Perform a Wood lamp exam to rule out noninflammatory tinea capitis. Refer to primary care for initial uncomplicated presentation. Consider referral to dermatology (for biopsy) or mental health in questionable or refractory cases. Additional information for sufferers is at the Trichotillomania Learning Center at www.trich.org.

Pearls

1. Hair density is greatly reduced at the affected site, but it is never bald.
2. There may be an associated mood or anxiety disorder.



FIGURE 14.114 ■ **Trichotillomania.** Patchy hair loss seen in a young child with a history of self-induced hair pulling. Short hair of different lengths are randomly distributed. (Photo contributor: Timothy D. McGuirk, DO.)

Clinical Summary

Oculogyric crisis (OGC) is the most common of the ocular dystonic reactions. It includes blepharospasm, periorbital twitches, and protracted staring episodes. It usually occurs as a side effect of neuroleptic drug treatment. OGC represents approximately 5% of the dystonic reactions. The onset of a crisis may be paroxysmal or stuttering over several hours. Initial symptoms include restlessness, agitation, malaise, or a fixed stare followed by the more characteristically described maximal sustained upward deviation of both eyes. The eyes may also converge, deviate upward and laterally, or deviate downward. The most frequently reported associated findings are backward and lateral flexion of the neck, widely opened mouth, tongue protrusion, and ocular pain. A wave of exhaustion follows some episodes. Other features noted during attacks include eye blinking, lacrimation, pupil dilation, drooling, facial flushing, vertigo, anxiety, and agitation. Several medications have been associated with the occurrence of OGC: cetirizine, neuroleptics, amantadine, benzodiazepines, carbamazepine, chloroquine, levodopa, lithium, metoclopramide, and nifedipine. Careful history and physical examination should exclude the possibility of focal seizures, meningitis, encephalitis, head injury, conversion reaction, Parinaud syndrome, and other types of movement disorders.

Management and Disposition

Treatment in the acute phase in children involves reassurance, discontinuation of the causative agent, and diphenhydramine at a dosage of 1.25 mg/kg initially; this may be repeated if there is no effect. For moderate to severe cases, give the initial dose parenterally. Occasionally, doses up to 5 mg/kg are required. The treatment with diphenhydramine should be continued every 6 hours for 1 to 2 days. Benztropine can also be used; however, it is not approved for children below 3 years of age. Close monitoring is important during treatment as dystonic reactions are occasionally accompanied by fluctuations in blood pressure and arrhythmias.

Pearls

1. The abrupt termination of the symptoms at the conclusion of the crisis or after the use of diphenhydramine is diagnostic and most striking.
2. In infants presenting with "seizures," unusual behavior, eye deviation, and a history of reflux treated with metoclopramide, the possibility of OGC should be considered. Although the overall incidence of extrapyramidal effects associated with metoclopramide is 0.2%, pediatric and geriatric patients are affected more commonly, with an incidence as high as 10%. These side effects usually occur within a few days of initiation of the medication and are more common at higher doses.



FIGURE 14.115 ■ **Oculogyric Crisis.** This 6-year-old boy developed extrapyramidal symptoms, including opisthotonos and oculogyric crisis, after his dosage of risperidone was increased. Benadryl 12.5 mg PO given at home resolved the opisthotonos. Persistent oculogyric crisis (upward gaze deviation) and hypertonia resolved completely after Benadryl 25 mg IM. (Photo contributor: Mark Ralston, MD.)

SETTING-SUN PHENOMENON (SUNDOWNING)

Clinical Summary

The setting-sun phenomenon also known as "sundowning" is a concerning sign that may represent a pathologic increase in intracranial pressure in the young infant. The clinical presentation consists of an upward-gaze paresis with eyes that appear to be driven downward. There is usually sclera showing between the upper eyelid and the iris. Retraction of the upper eyelids, sometimes accompanied by raising of the brow, may be seen. Although this sign is commonly seen in children with obstructive hydrocephalus, it may also be seen as a result of intracranial hypertension of other causes (trauma or ventriculoperitoneal shunt dysfunction) and occasionally in normal infants. It is thought to result from compression of the periaqueductal structures.

The phenomena could be an important sign in early detection of elevated intracranial pressure, appearing sooner than enlarged head circumference, full fontanelle, separation of sutures, irritability, or vomiting.

Management and Disposition

Suspect increased intracranial pressure and obtain neuroimaging urgently. Consult neurosurgery for guidance in initial management and for definitive treatment in the operating room.

Pearls

1. This sign is a valuable cue for obtaining prompt neuroimaging and urgent surgical intervention.
2. When persistent, this sign is a frequent marker of elevated intracranial pressure, appearing in 40% of children with hydrocephalus (of any cause) and in 13% of patients with shunt dysfunction.
3. Ask parents to share photographs of their child taken since birth to assess for changes to head size and for the presence of sundowning.

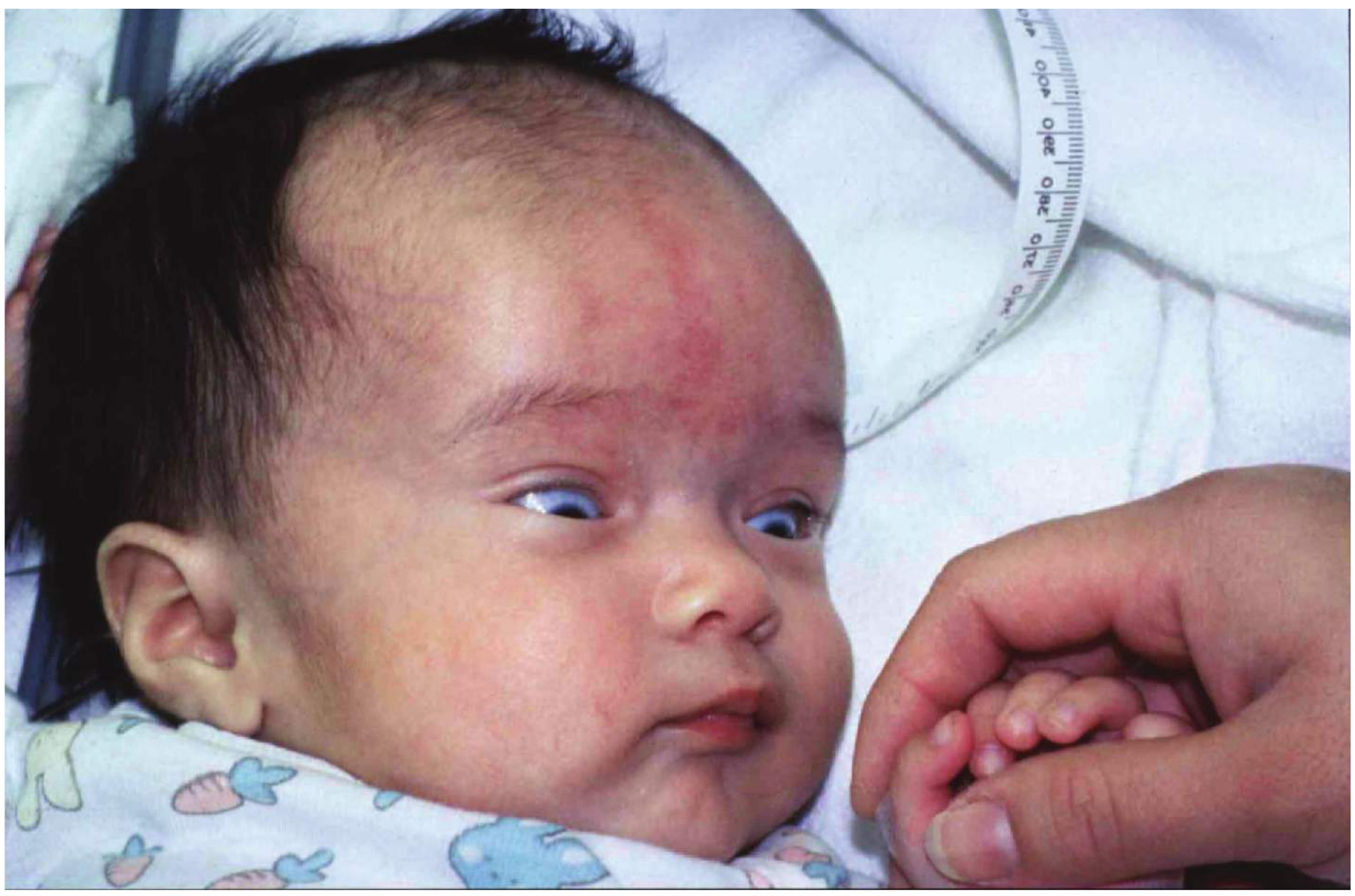


FIGURE 14.116 ■ **Setting-Sun Phenomenon (Sundowning).** The eyes appear driven downward in this infant with hydrocephalus. (Photo contributor: Stephen W. Corbett, MD.)

Clinical Summary

Kawasaki disease (KD), also known as mucocutaneous lymph node syndrome, is an acute self-limited medium to small vessel vasculitis of unknown etiology occurring most commonly in young children with a peak incidence between 1 and 2 years of age. The classic diagnosis is made clinically and is based on the presence of more than 5 days of fever and at least four of the following five principal clinical features:

1. Generalized polymorphous rash usually appearing within 5 days of the onset of fever and most commonly is a nonspecific, diffuse maculopapular eruption. Early desquamation may occur in the perineal region, especially in infants.
2. Bilateral bulbar conjunctivitis sparing the limbus and nonexudative.
3. Changes to the lips and oral mucosa including dry, red, cracked lips, strawberry tongue, and diffuse erythema of the oropharyngeal mucosa.
4. Peripheral extremity changes: acute erythema and induration of the palms and soles followed in 2 to 3 weeks by desquamation of the fingers and toes beginning in the periungual region. One to two months after the onset of the fever, deep transverse grooves across the nails (Beau lines) may appear.
5. Cervical lymphadenopathy that is usually unilateral, confined to the anterior cervical triangle, and is greater than or equal to 1.5 cm in diameter.

The fever of KD is typically spiking and unremitting with peak temperatures often greater than 40°C (104°F). Incomplete (atypical) KD, which is more common in infants, does not meet all of the classic criteria but should be suspected in any child with unexplained fever for more than 7 days or unexplained fever for more than 5 days with less than three KD-associated clinical criteria. Laboratory evaluation is necessary to support the diagnosis of incomplete KD and includes elevated ESR (≥ 40 mm/h) or CRP (≥ 3 mg/dL), and ≥3 supplemental laboratory criteria (albumin ≤ 3 g/dL, anemia for age, elevation of alanine aminotransferase, platelet count after 7 days ≥ 450,000/mm^3, white blood cell count ≥ 15,000/mm^3, and urine ≥ 10 white blood cells/high-power field). An abnormal echocardiogram may also aid in the diagnosis of incomplete KD.

Coronary artery aneurysms or ectasias are the most important complication of KD and develop in 15% to 25% of untreated children and may lead to ischemic heart disease, myocardial infarction, or sudden death. KD may also cause aseptic meningitis, urethritis, myocarditis, arthritis, liver



FIGURE 14.117 ■ **Kawasaki Disease.** Irritability in an infant with Kawasaki disease. Note also the conjunctivitis, and red, cracked lips. (Photo contributor: Tomisaku Kawasaki, MD.)

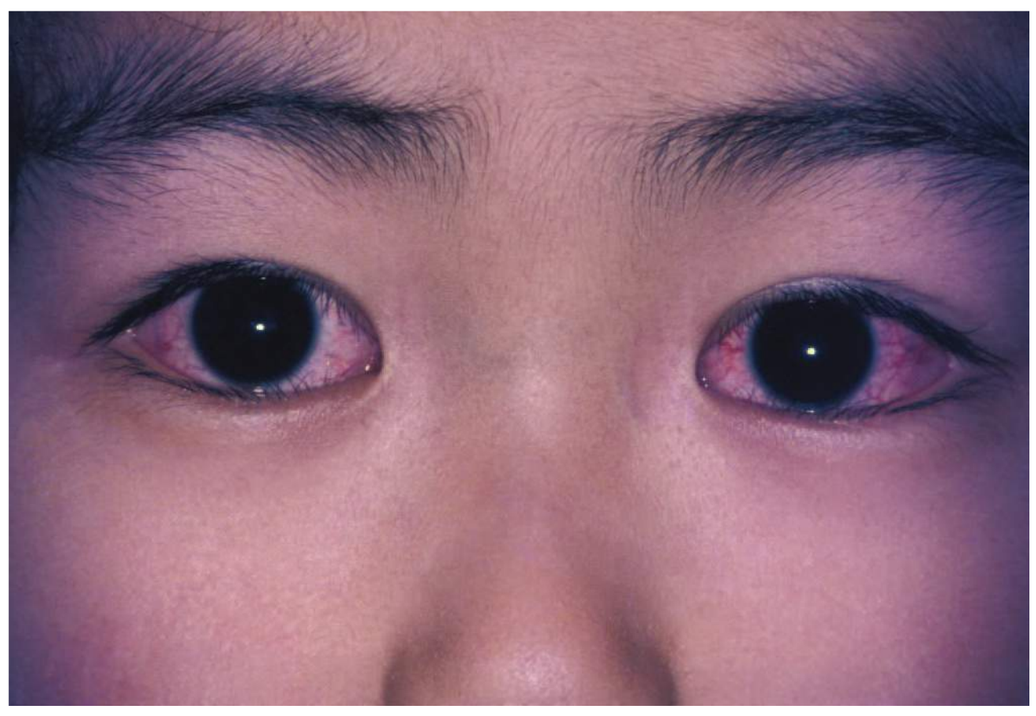


FIGURE 14.118 ■ **Conjunctivitis.** Note the intense redness of the bulbar conjunctiva seen in this patient with Kawasaki disease. (Photo contributor: Tomisaku Kawasaki, MD.)

dysfunction, abdominal pain, and gallbladder hydrops. The differential diagnosis of KD includes scarlet fever, staphylococcal scalded skin syndrome, toxic shock syndrome, viral infections (measles, adenovirus, enterovirus, Epstein-Barr virus), bacterial cervical lymphadenitis, drug hypersensitivity reaction, Stevens-Johnson syndrome, and acrodynia.

Management and Disposition

If suspected, consultation with a cardiologist or other specialist in KD is highly recommended for confirmation of diagnosis as well as treatment. A baseline echocardiogram should be obtained to document the status of the coronary arteries. Treatment includes hospitalization, administration of IVIG 2 g/kg infused over 8 to 10 hours, and initiation of high-dose aspirin 80 to 100 mg/kg/day in four divided doses. Approximately 90% of patients will defervesce with this regimen. Failure to respond is usually defined as persistent or recurrent fever greater than or equal to 36 hours after completion of the initial IVIG infusion. Retreatment with IVIG 2 g/kg is recommended. With IVIG treatment in the acute phase of the disease, the risk of coronary artery abnormalities is decreased to less than 5%.



FIGURE 14.119 ■ **Lymphadenopathy.** Visible cervical lymphadenopathy is seen in this child with Kawasaki disease. (Photo contributor: Tomisaku Kawasaki, MD.)



FIGURE 14.121 ■ **Periungual Desquamation.** This finding typically begins 2 to 3 weeks after the onset of Kawasaki disease, in contrast to perineal desquamation that occurs during the early course of the disease in infants. (Photo contributor: Tomisaku Kawasaki, MD.)



FIGURE 14.120 ■ **Extremity Findings.** Red swollen hands are present in this patient with Kawasaki disease. (Photo contributor: Tomisaku Kawasaki, MD.)



FIGURE 14.122 ■ **Beau Lines.** Deep transverse grooves across the nails may appear 1 to 2 months after the onset of fever. (Photo contributor: Tomisaku Kawasaki, MD.)

Pearls

1. The diagnosis of typical KD is clinical, but laboratory studies may be helpful in cases of suspected incomplete KD.
2. Children with KD are commonly irritable and difficult to console.
3. KD is the leading cause of acquired heart disease in children in the United States. Echocardiography should be considered in any infant less than 6 months of age with fever of greater than or equal to 7 days duration, laboratory evidence of inflammation, and no other explanation for the febrile illness.
4. Even when treated with high-dose IVIG regimens within the first 10 days of illness, approximately 5% of children with KD develop at least transient coronary artery dilation.
5. Vaccination against influenza should be provided to all patients receiving aspirin therapy to avoid Reye syndrome.

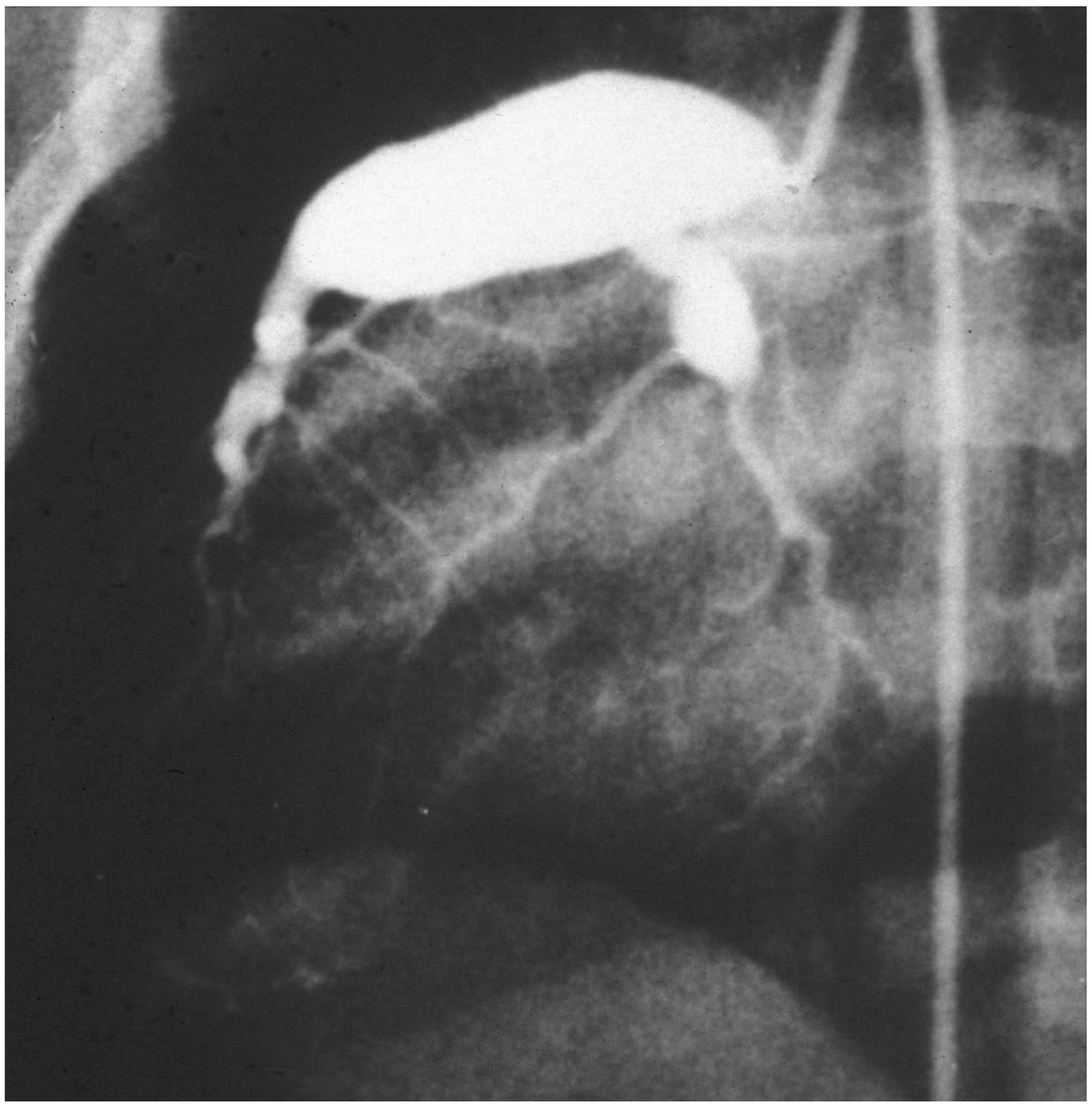


FIGURE 14.123 ■ **Coronary Artery Aneurysms.** A large coronary artery aneurysm is seen in this patient with Kawasaki disease. (Photo contributor: Tomisaku Kawasaki, MD.)

Chapter 15

CHILD ABUSE

Kathi L. Makoroff
Daniel M. Lindberg
Marguerite M. Care



Inflicted Scald Burn. Partial thickness burn on the lower extremity with sharp demarcation is suspicious for intentional injury. (Photo contributor: Kathi Makoroff, MD.)

The authors acknowledge the special contributions of Robert A. Shapiro, Charles J. Schubert, and Megan L. McGraw for contributions to prior editions.

Physical Abuse
CUTANEOUS FINDINGS

Clinical Summary

Bruises are the most common manifestation of child physical abuse. Children younger than 6 months and those that are not yet "mobile" rarely have any bruising from accidental injury. In older children, bruising to the abdomen, ear, neck, cheek, buttocks, or genitals is also less likely to be the result of accidental injury. Child abuse should also be strongly considered when a bruise takes the shape of an object (belt, cord, hand).

Certain clues may assist in differentiating abusive from accidental burns, but, often, doubt remains even after careful evaluation. With an immersion burn, the child is held into or under hot liquid and the burn margins are usually distinct. A "splash" pattern, conversely, will occur when a child attempts to get out of the way of the hot liquid; the burn margins are less distinct and there may be additional adjacent small burns. Contact burns usually have distinct and recognizable shapes but it is sometimes difficult to determine how the burn occurred. A child who has multiple contact burns or burns to areas that are unlikely to come in contact with the hot object accidentally should be evaluated for abuse.

While certain findings (bite marks, patterned bruises) are very specific for abuse, cutaneous findings are not sensitive for predicting other injuries such as fractures, brain injury, or abdominal injury. The absence of bruising should not preclude screening for other injuries.

Management and Disposition

With the exception of significant burns, abusive cutaneous injuries rarely require significant treatment. Rather, children with concerning cutaneous findings should be screened for other abusive injuries and medical mimics of abuse. Order neuroimaging (CT or MR) for children less than 6 months of age, skeletal survey for children less than 2 years old, and hepatic transaminases (alanine transferase [AST]/aspartate aminotransferase [ALT]) for children less than 5 years old. Order prothrombin time/partial thromboplastin time (PT/PTT) and complete blood count (CBC) for children with concerning bruises or other findings that could indicate a coagulopathy and consider hematology consultation for other coagulopathy testing.

If a human bite is suspected, photo document and swab for potential DNA analysis. Run a moistened swab followed by a new dry swab over the area and clearly follow and document chain of evidence. If available, consult with a forensic dentist.

Report suspected abuse to the legally mandated child protection agency before the child is discharged from the emergency department (ED). In the United States, medical professionals have a legal mandate to report in cases concerning for child abuse. When available, consult with the Child Abuse Team.



FIGURE 15.1 ■ **Loop Marks.** Subtle loop and linear marks are seen on the thigh and buttock of this child. (Photo contributor: Robert A. Shapiro, MD.)



FIGURE 15.2 ■ **Loop Marks.** Loop and linear marks signify use of a cord or other similar object. (Photo contributor: Alan B. Storrow, MD.)

Pearls

1. Use the distance between the maxillary canines to determine if the biter was someone with primary teeth (child) or secondary teeth (adult). Most adults have greater than 29 mm distance.
2. Do not use the color or appearance of a bruise to estimate its age. Despite a widely cited dating schema, there is no evidence to support this.
3. Cigarettes can burn as hot as 400 to 700°C. Inflicted cigarette burns are therefore usually ulcerated and full-thickness, rarely superficial.
4. Burn mimics to consider include impetigo, phytophotodermatitis, and contact dermatitis. Bruise mimics to consider include hemophilia, von Willebrand disease, Henoch-Schönlein purpura (HSP), and idiopathic thrombocytopenic purpura (ITP).
5. Place a millimeter ruler or a coin in the field when photographing injuries, and photograph at a 90 degree angle to the plane of the finding.
6. Genital bruising is always concerning for child abuse. Physical abuse as well as sexual abuse should be considered.



FIGURE 15.3 ■ **Belt Pattern.** This patterned bruise was inflicted by a woven belt. (Photo contributor: Daniel M. Lindberg, MD.)



FIGURE 15.5 ■ **Adult Bite Marks.** This adult bite mark demonstrates evidence of suction (hickey) within the bite mark itself. The size of the facing arches indicates an adult bite. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.4 ■ **Buttocks Bruising.** Forceful spanking can cause buttocks bruising and longitudinal bruises parallel to the gluteal fold. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.6 ■ **Adult Bite Marks.** This bite mark on a child's arm is consistent with an adult bite. While marks from individual teeth are not visible, facing arches of appropriate size are indicative of a bite mark. (Photo contributor: Megan McGraw, MD.)



FIGURE 15.7 ■ **Child Bite.** This bite mark, with a maxillary intercanine distance less than 29 mm, is more consistent with a child biter. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.9 ■ **Child Bite.** Distinct impressions of teeth are seen in this injury with the outlines of the upper and lower oral arches. Note the size of the mother's mouth in relation to the size of the bite on the neck, making the adult an unlikely source. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 15.8 ■ **Child Bites.** Multiple bite marks were inflicted on this child's face by another child. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.10 ■ **Ligature Bruise.** This child survived beating and attempted strangulation that resulted in circumferential, linear neck abrasions and occipital ecchymosis. (Photo contributor: Barbara R. Craig, MD.)



FIGURE 15.11 ■ **Ligature Bruise.** This bruises around the neck of this child represent ligature marks. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.12 ■ **Folk Remedies: Coining.** This child has linear petechiae along her spine from the Southeast Asian practice of coining (Cheut Sah or Cao Gio) where a coin is rubbed along the skin to heal illness. This practice is neither painful nor dangerous and is not abusive. Another folk remedy, cupping or moxibustion (not shown), produces circular bruising when a match is used to create a vacuum inside a glass placed on the skin. (Photo contributor: Charles Schubert, MD.)



FIGURE 15.13 ■ **Folk Remedies: Cupping.** Circular "burns" from the practice of "cupping" result when warm cups are placed on the skin to draw out illness. (Reproduced with permission from American Academy of Pediatrics. Visual Diagnosis of Child Abuse. 3rd ed. Elk Grove Village, IL: American Academy of Pediatrics; 1994.)



FIGURE 15.14 ■ **Henoch-Schönlein Purpura (HSP).** This child has palpable purpura on the extensor surfaces of the legs. HSP should be considered whenever there is symmetric ecchymosis along the extensor surfaces of the extremities and buttocks. Migratory arthritis and abdominal pain may be present. (Photo contributor: Ralph A. Gruppo, MD.)



FIGURE 15.15 ■ **Dermal Melanosis (Mongolian Spots).** These blue-gray congenital marks are more common in darker-skinned children. Frequently found on the buttocks or low back, they can be seen anywhere. They are distinguished from bruising because they fail to resolve in days or weeks. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.16 ■ **Pattern Burn.** Multiple contact burns on the chest and abdomen from a curling iron. Accidental curling iron burns occur, but this number of injuries is very concerning for abuse. (Photo contributor: Robert A. Shapiro, MD.)



FIGURE 15.17 ■ **Pattern Burn.** This child was held against a heater grate. The pattern indicates that the child's leg was flexed at the time of injury. (Photo contributor: David W. Munter, MD.)



FIGURE 15.18 ■ **Immersion Burn.** Immersion burns are often associated with toilet-training accidents. This girl was plunged into hot water after soiling herself. She shows sparing of the buttocks, which contacted the surface of the bathtub and avoided being burned. (Reproduced with permission from American Academy of Pediatrics. Visual Diagnosis of Child Abuse. 3rd ed. Elk Grove Village, IL: American Academy of Pediatrics; 1994.)



FIGURE 15.19 ■ **Immersion Burn.** A child's extremities may also be immersed. Burns in a "stocking" or "glove" pattern are concerning for an immersion mechanism. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.20 ■ **Splash Burn.** This toddler pulled a cup of hot liquid on to her. Note the "flowing" pattern that starts on her face and extends down her arm with burn depth decreasing as the liquid flows and cools. Also characteristic are "skip" areas of skin that are not affected. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.21 ■ **Laxative "Burn."** Laxatives can cause skin irritation or sloughing, and often results in a diamond-shaped pattern. Blistering can also occur. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.22 ■ **Scald Burn with Sparing.** Superficial and partial-thickness burns were noted on the patient's anterior surface only. The areas of abdominal sparing indicate that the victim was flexed and curled at the time of injury. The child's caretaker, the mother's boyfriend, admitted to holding the child under a running hot-water tap. Partial-thickness burns on the penis and medial thighs are indicative of pooling of the liquid in those areas, resulting in a time-dependent injury. (Photo contributor: William S. Smock, MD.)



FIGURE 15.23 ■ **Cigarette Burn—Inflicted.** Cigarette burns are circular injuries with a diameter of about 8 mm. Children who accidentally run into a lit cigarette often have burns to the face or distal extremities. Accidental burns may be less distinct or deep compared with inflicted burns. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.24 ■ **Cigarette Burn—Inflicted.** This child suffered an inflicted cigarette burn to the ear. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.25 ■ **Impetigo.** These circular lesions of impetigo may be confused for healing cigarette burns. (Photo contributor: Michael J. Nowicki, MD.)

Clinical Summary

Abusive head trauma (AHT) (formerly shaken baby syndrome) is an important cause of morbidity and mortality from child physical abuse. Subdural hematoma is the most common finding. Skull fractures and/or cerebral edema can also be present. Retinal hemorrhages do not have to be present to make the diagnosis of AHT. Retinal hemorrhages that are numerous, multilayered, and extend to the periphery have only been described in infants and children with significant trauma including AHT. Fewer hemorrhages clustered around the posterior pole of the retina have been described with less severe trauma and in critically ill children with no concern for abuse and are, therefore, less specific. Retinal examinations without dilation, or by nonspecialists, are limited. All children with traumatic brain injury and concern for abuse should be examined by an ophthalmologist with pediatric experience.

Management and Disposition

Infants and children with head injury should be treated as medically appropriate. Subdural hemorrhages can be seen in infants and children with nonspecific (fussy, vomiting, less active) or no symptoms. Therefore, have a low threshold for ordering neuroimaging in an infant or child who has other concerning signs of abuse (bruising, fractures).

In the absence of major trauma, subdural hematomas are very concerning for abuse, and should trigger a thorough search for other abusive injuries, coagulopathy, or a history of abuse. A report of suspected child abuse should be made to children's services if there is a concern for AHT.

Pearls

1. A history that a short fall or other minor injury caused a significant intracranial injury should be highly suspected.
2. Infants and children with AHT may have no external signs of trauma and nonspecific or no neurologic deficits.
3. Minor skull fractures can occur accidentally and are impossible to date.
4. CT can identify significant soft tissue injury that is missed on physical examination.
5. Significant retinal hemorrhages are exceedingly rare if neuroimaging is negative. Dedicated retinal exam may be omitted if traumatic brain injury is absent.

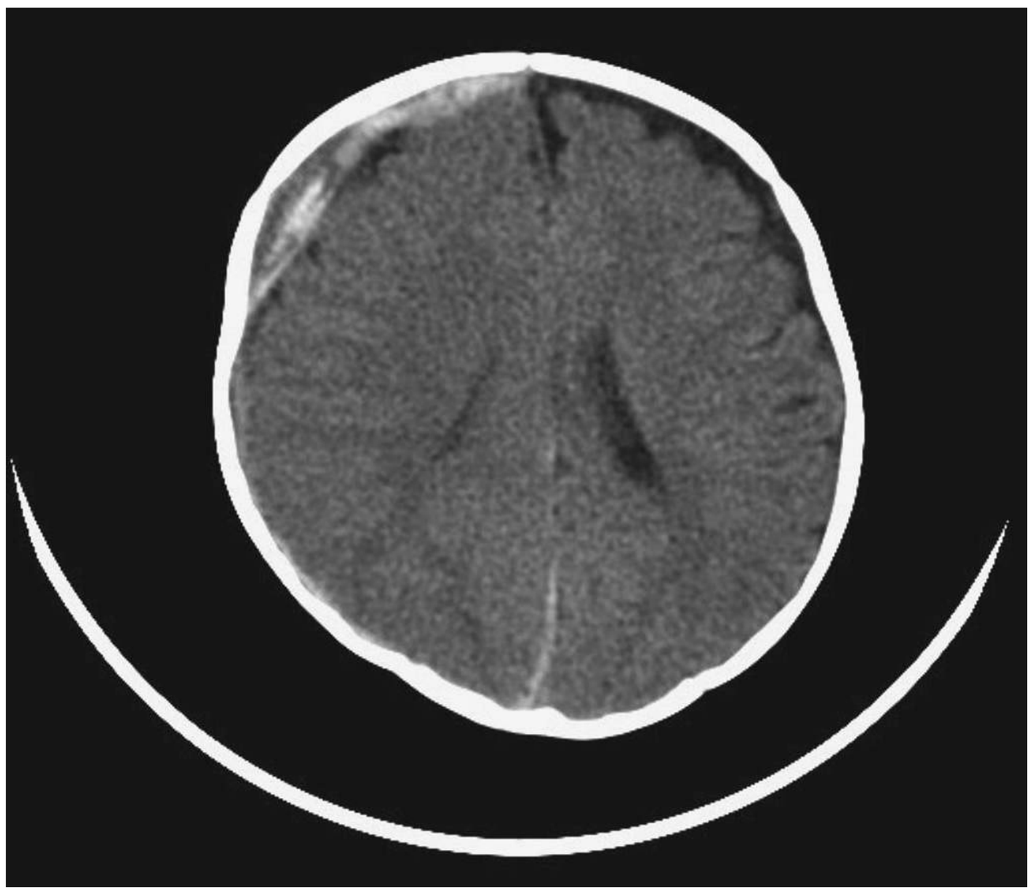


FIGURE 15.26 ■ **Acute Subdural Hematoma.** There is a crescent-shaped, hyperdense collection, indicating an acute subdural hematoma over the right cerebral hemisphere. In addition, the right side of the brain demonstrates mass effect. (Photo contributor: Cincinnati Children's Hospital Medical Center.)

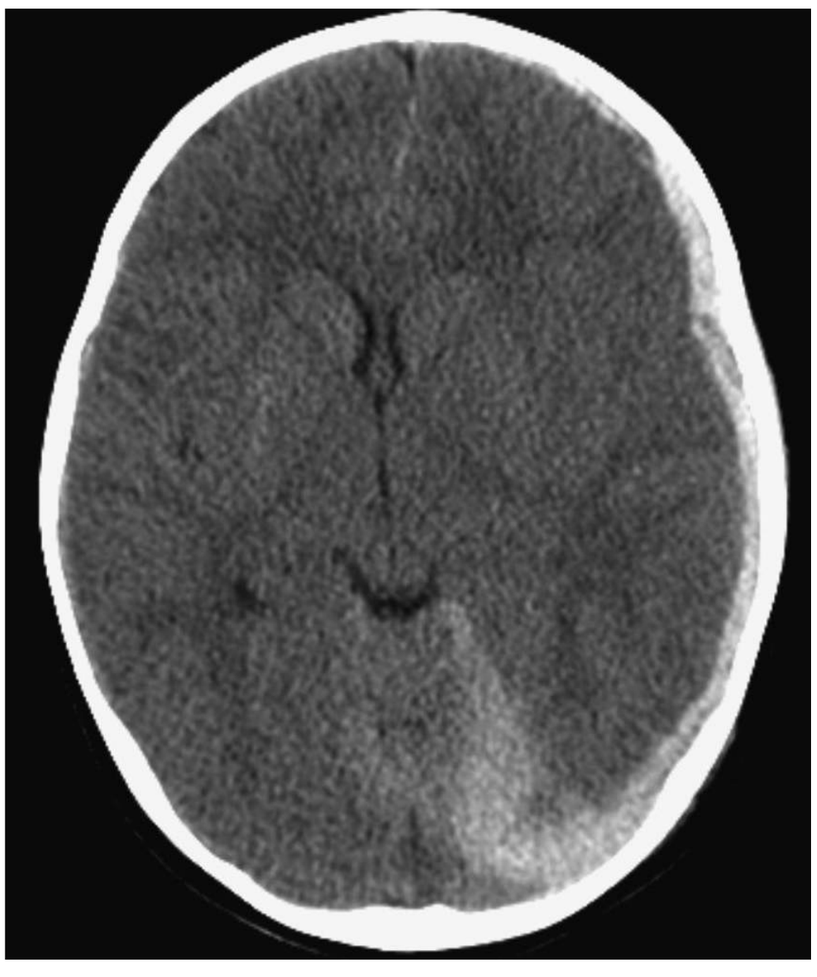


FIGURE 15.27 ■ **Left Subdural Hematoma.** There is a thin hyperdense collection that extends along the entire left hemisphere as well as along the tentorium indicating an acute subdural. (Photo contributor: Marguerite Care, MD.)

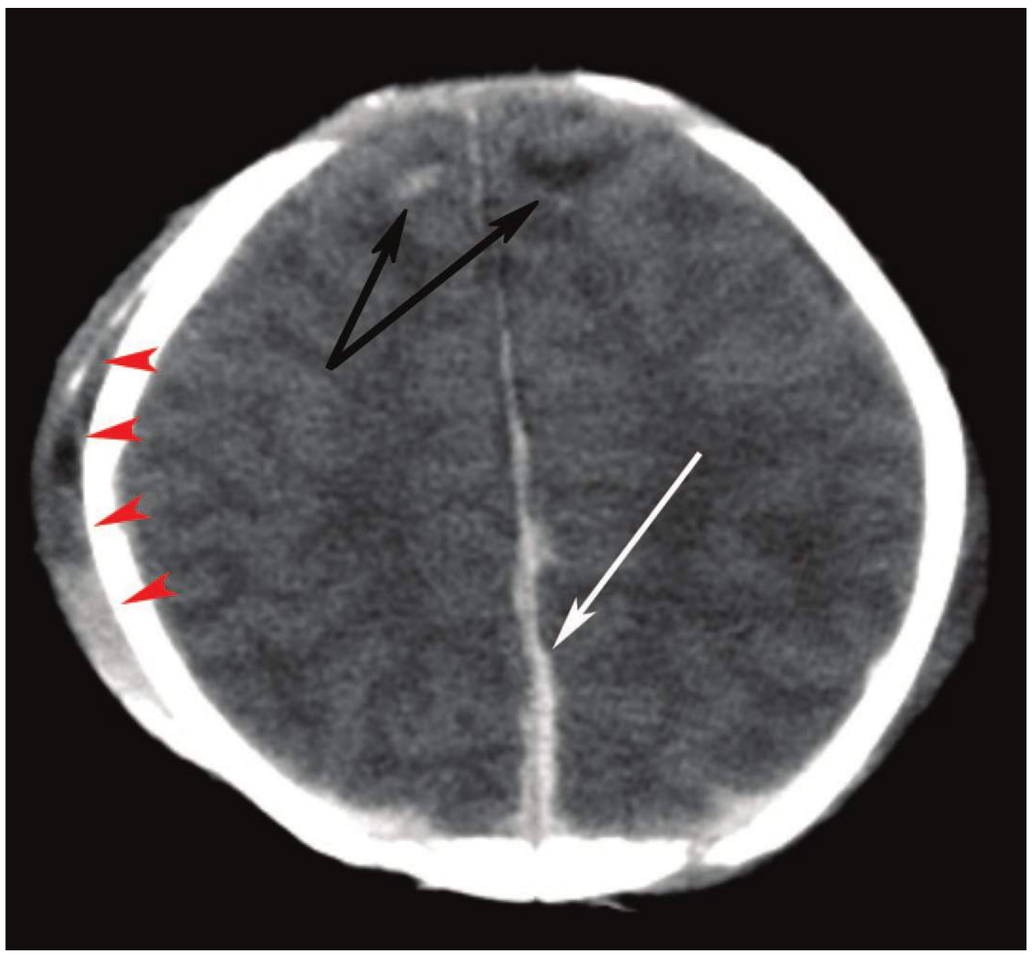


FIGURE 15.28 ■ **Subdural Hematoma and Extra-Axial Swelling.** This image shows intrahemispheric acute subdural blood (large arrows) and subtle contusions (black arrows) of the frontal lobes. Extracranial soft tissue swelling (arrow heads) is also present; sometimes this is visible on CT but not on physical examination. (Photo contributor: Marguerite Care, MD.)

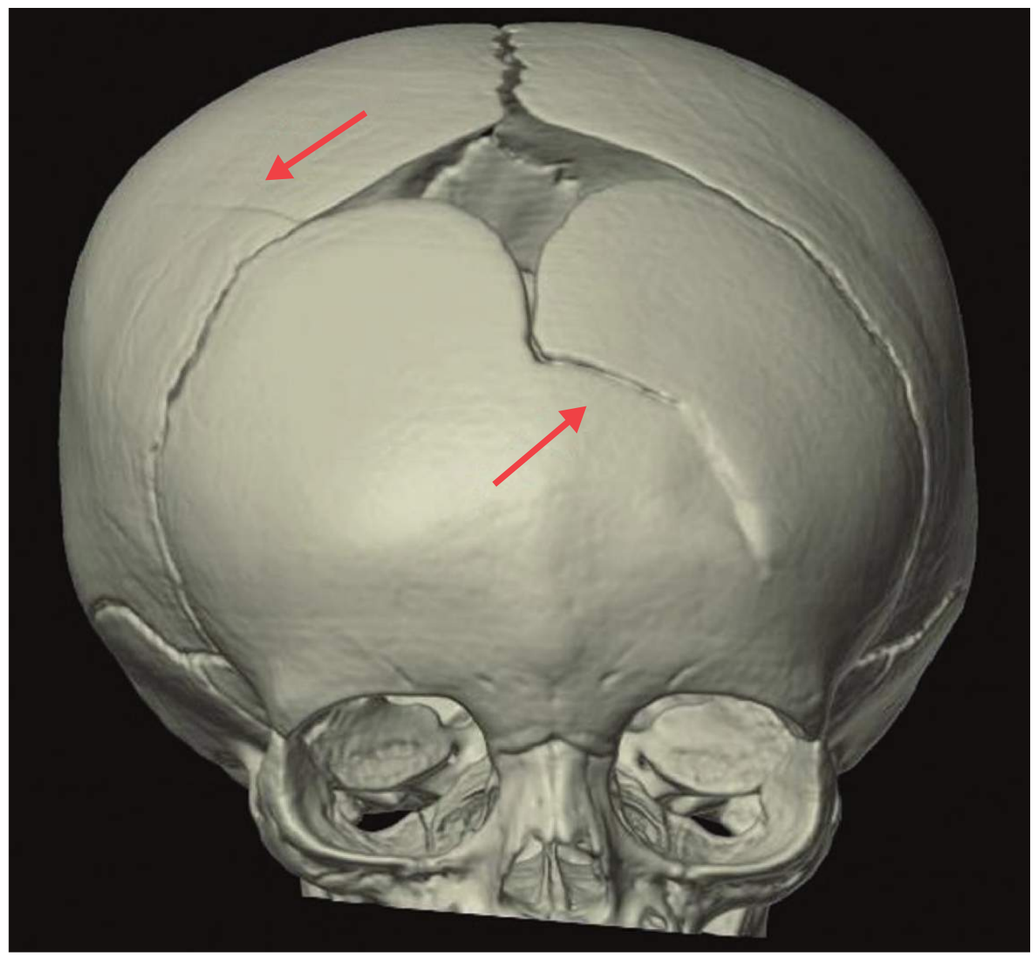


FIGURE 15.30 ■ **Vertex Skull Fracture.** This infant was accidentally dropped on her head. The 3D reconstructed image shows two fractures that originated from the anterior fontanelle. It is possible that two distinct fractures resulted from a single impact to the vertex of the head. (Photo contributor: Cincinnati Children's Hospital Medical Center.)

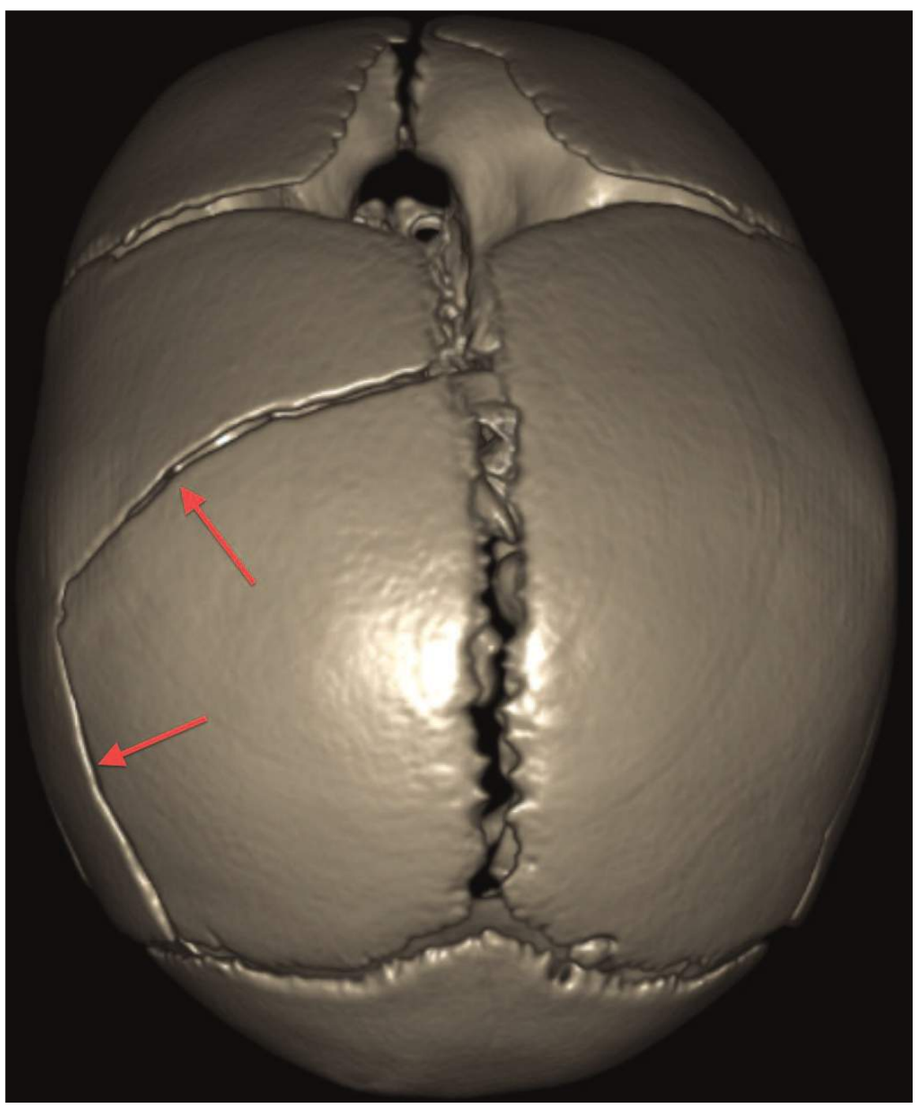


FIGURE 15.29 ■ **Skull Fracture.** This 3D reconstructed image shows a left parietal skull fracture (arrows). Skull fracture can result from inflicted or accidental mechanisms. (Photo contributor: Marguerite Care, MD.)

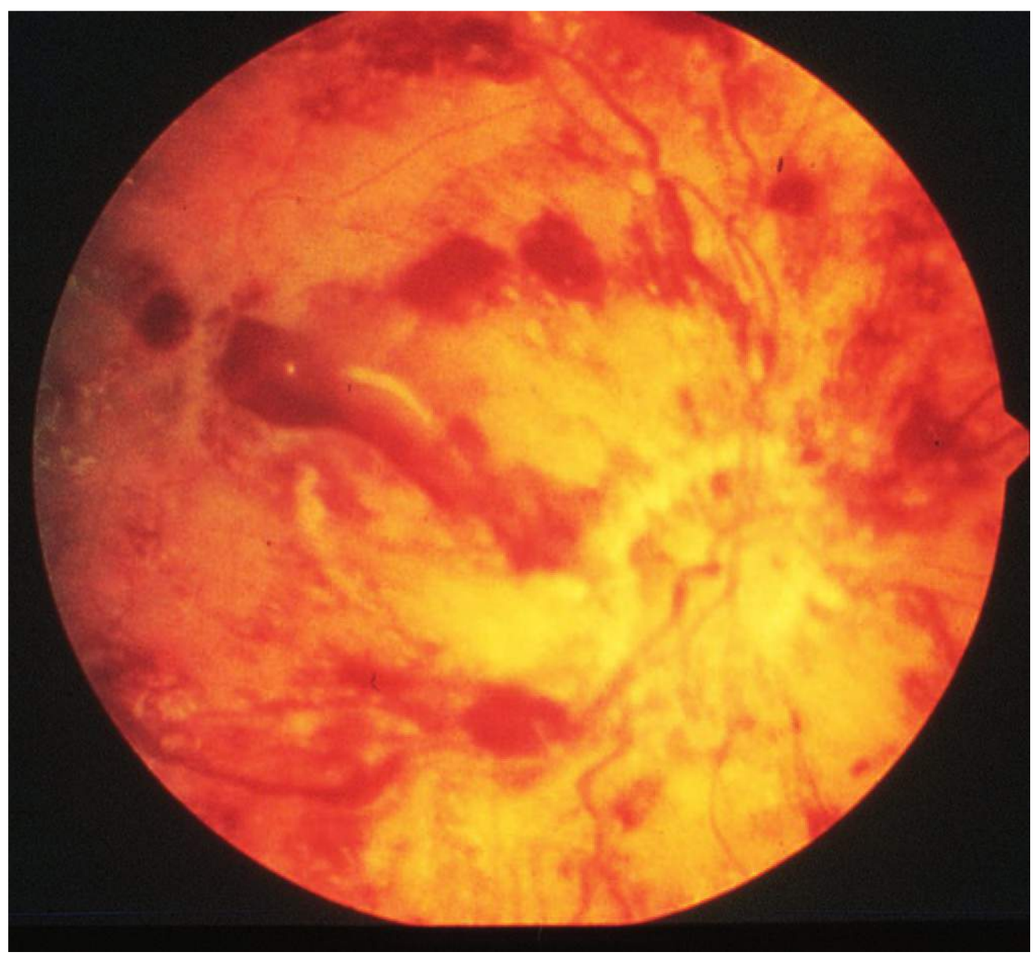


FIGURE 15.31 ■ **Retinal Hemorrhages.** Multiple retinal hemorrhages (too numerous to count) are present in this image. A pediatric or general ophthalmologist can obtain a more complete view of the retina with dilated direct ophthalmoscopy and should be consulted to evaluate for retinal hemorrhages. (Photo contributor: Rees W. Sheppard, MD.)

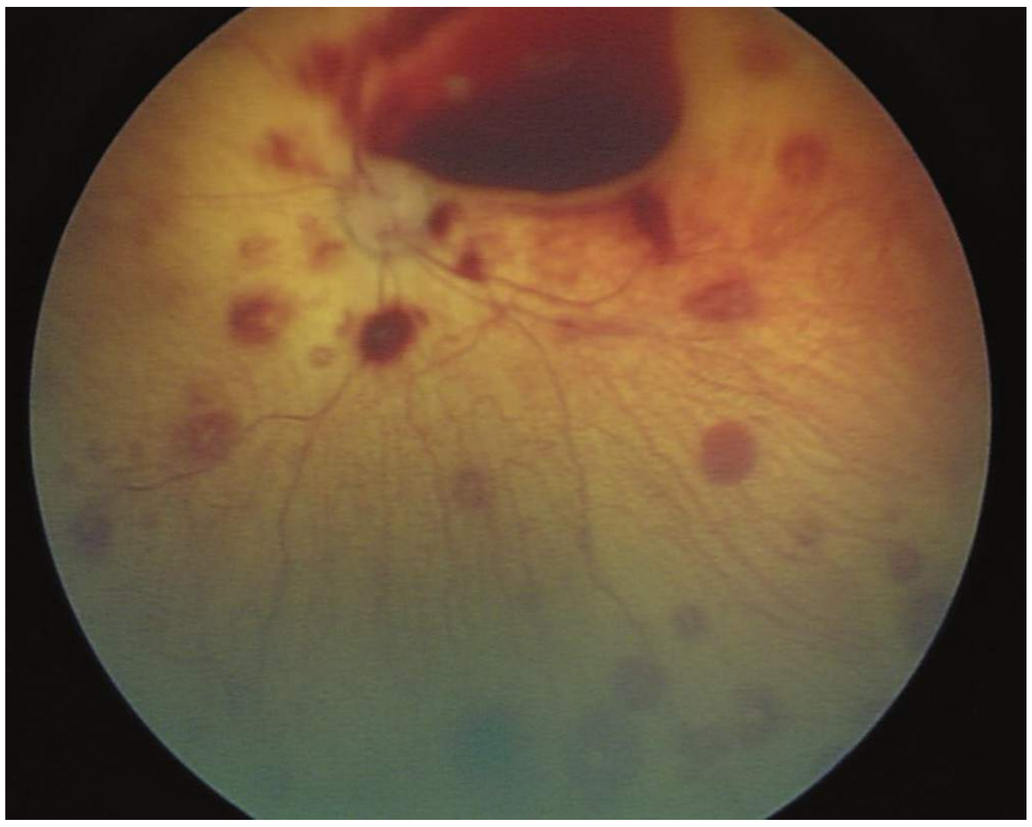


FIGURE 15.32 ■ **Retinal Hemorrhages.** Multiple discreet subhyaloid hemorrhages are seen. (Photo contributor: John D. Baker, MD, and Massie Research Laboratories, Inc.)



FIGURE 15.34 ■ **Hair Pulling Subgaleal Injury.** This child has a large posterior subgaleal hemorrhage without associated skull fracture. It resulted from abusive hair yanking. (Photo contributor: Cincinnati Children's Hospital Medical Center.)

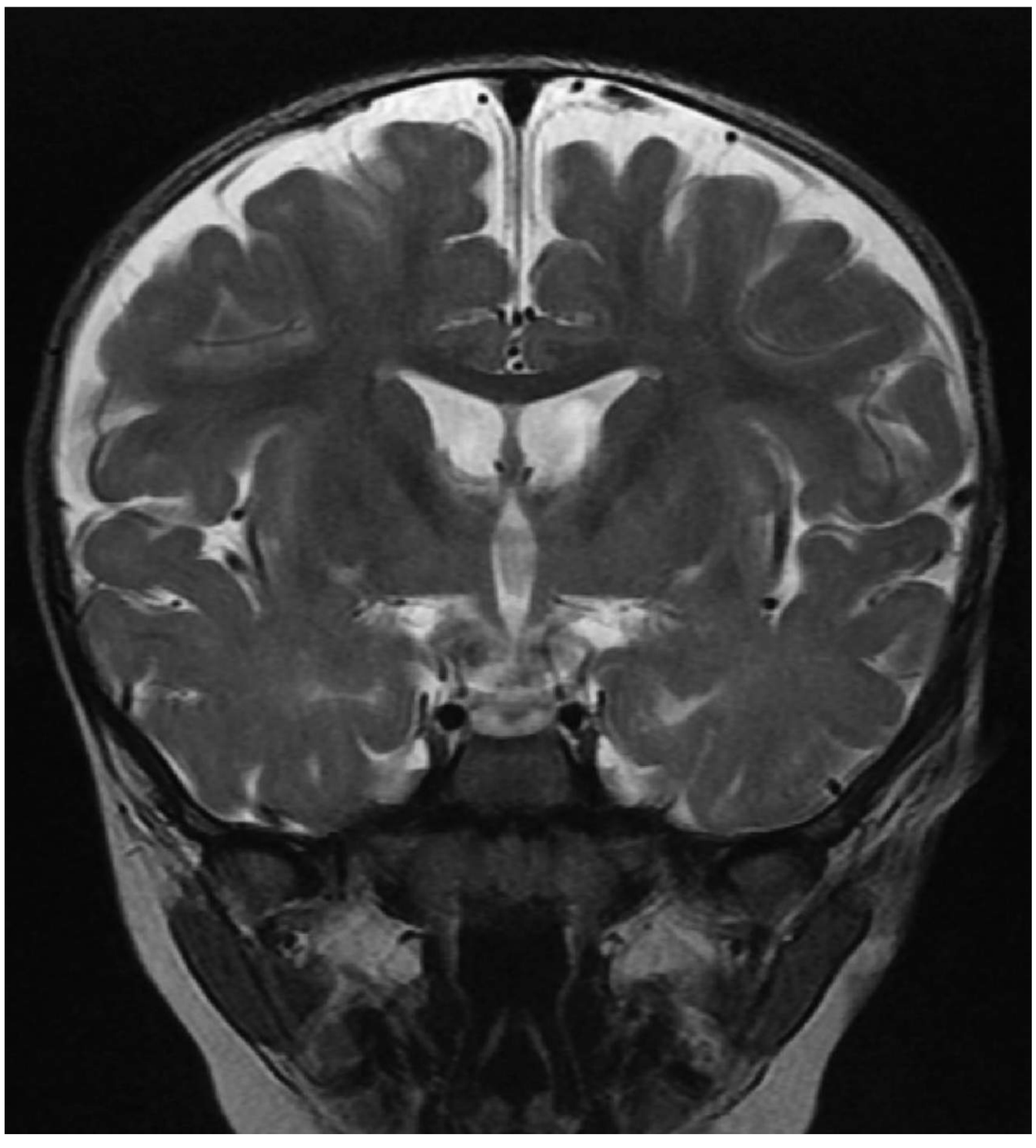


FIGURE 15.33 ■ **Benign Extra-Axial Fluid.** This coronal T2-weighted MRI shows enlarged subarachnoid spaces (vessels running through). This can be a common finding in children with macrocraina. The enlarged spaces can be confused for subdural collections which will then raise the concern of abusive head injury. (Photo contributor: Marguerite Care, MD.)

Clinical Summary

Because crying and feeding difficulties can serve as the trigger for physical abuse, the face is frequently the site of the most forensically significant, even if subtle, signs of abuse. Carefully examine the oral cavity, ears, and fontanel of all children with concern for abuse. Force-feeding or forceful insertion of utensils or fingers into a child's mouth can result in injury to lips, frenula, or palate, as well as deeper oropharyngeal injury. Accidental oropharyngeal injuries are commonly caused in older children by toothbrushes, lollipops, or popsicle sticks, but these findings are concerning for abuse in children who are not yet ambulatory.

Management and Disposition

Most abusive facial injuries are minor or self-limited, and are most important as an indication for further screening for other occult injuries, especially AHT. Bruising to the ear is very concerning for abuse, as are injuries to the labial or lingular frenula, or palate in children who are not yet walking. All injuries concerning for abuse should be photographed and carefully documented in the medical record.

Children with abusive oropharyngeal injuries should be evaluated as other children with accidental oropharyngeal injuries, with low threshold to image (MRA or CTA) to identify vascular injury or deep space infection or abscess.

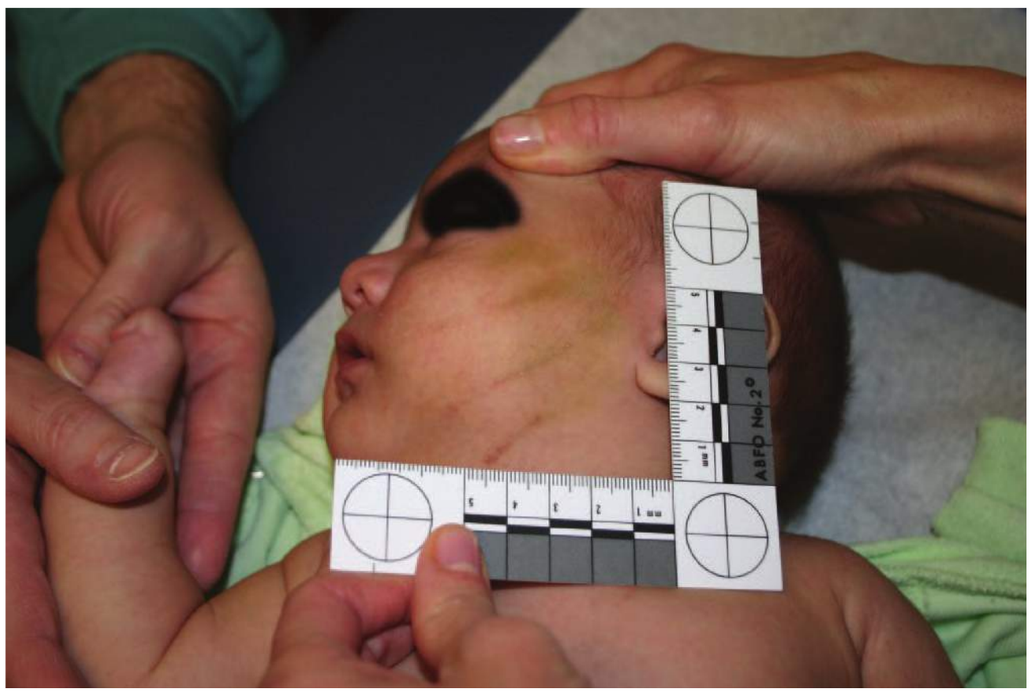


FIGURE 15.35 ■ **Hand-Print Bruise.** Note that the bruises run between and around the fingers, creating parallel lines of bruising. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.36 ■ **Pinna Bruise.** Any bruising to the ear is concerning for abuse and should trigger a thorough evaluation for other abusive injuries. It is important to examine the entire ear. In this case, there is bruising to the posterior part of the pinna. (Photo contributor: Robert A. Shapiro, MD.)



FIGURE 15.37 ■ **Pinna Bruise.** Any bruising to the ear is concerning for abuse and should trigger a thorough evaluation for other abusive injuries. (Photo contributor: Daniel M. Lindberg, MD.)

Pearls

1. Identification of injury to the oral cavity in children with other concerns for abuse should substantially increase the level of concern for abuse.
2. The physical examination of a fussy infant or any patient with injuries should include a good look at the mouth and ears.



FIGURE 15.38 ■ **Forehead Hematoma with Eye Bruising.** This child with hemophilia demonstrates the tendency of blood from a forehead hematoma to track downward with gravity and become visible in the periorbital region. In some cases, evidence of the forehead injury may be subtle. (Photo contributor: Ralph A. Gruppo, MD.)



FIGURE 15.39 ■ **Gag Ligature.** This child had a sock stuffed into his mouth and tied around his head. The bruises in the corners of the child's mouth are indicative of gagging. (Photo contributor: Robert A. Shapiro, MD.)

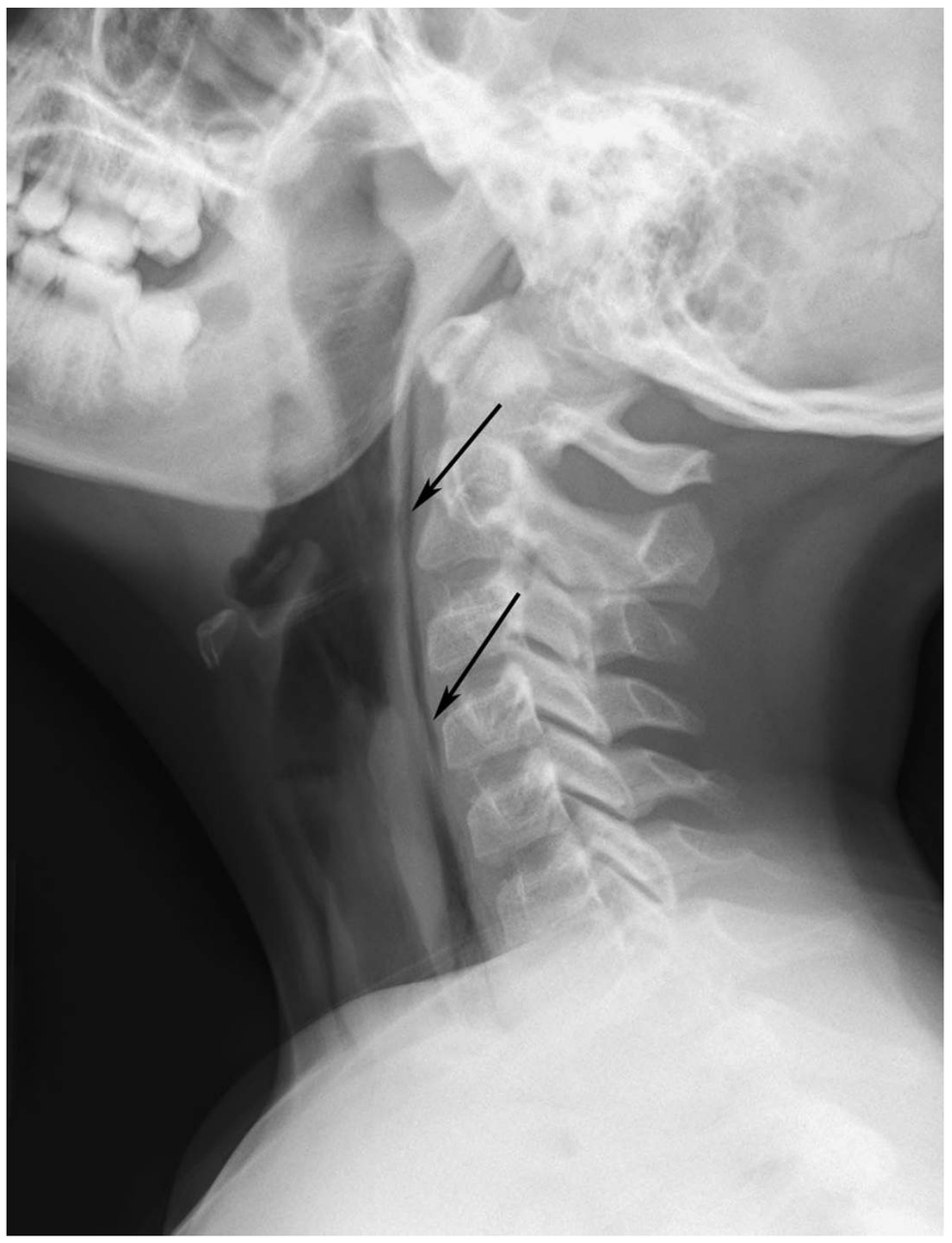


FIGURE 15.40 ■ **Oropharyngeal Injury.** Fingers, utensils, or other objects can perforate the oropharynx when shoved into the mouth of young children in the course of feeding or in an attempt to pacify an infant. This should trigger a thorough evaluation for other abusive injuries. This x-ray demonstrates retropharyngeal air (arrows) indicating oropharyngeal perforation. (Photo contributor: Marguerite Care, MD.)



FIGURE 15.41 ■ **Labial Frenulum Tear.** Injuries of the labial or lingular frenula are concerning for abuse, especially in nonmobile children. The frenula should be specifically examined when there are other concerns for abuse, especially in infants. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.42 ■ **Lip Injury.** This subtle injury was identified in a child with facial bruising attributed to a short fall. Ultimately, several abusive injuries were identified and the child's mother was found to have struck the child with a blunt object. (Photo contributor: Daniel M. Lindberg, MD.)



FIGURE 15.43 ■ **Dental Injury.** This school-aged child had multiple abusive injuries, including this chipped tooth (left, lower incisor) from blunt trauma. (Photo contributor: Kathi L. Makoroff, MD.)

Clinical Summary

Radiographic signs remain among the most specific and well-supported indicators of an abusive mechanism for injury. Signs of fracture healing can be used to determine the age of an injury, and can therefore be used to help determine the perpetrator of abuse, or establish that a child has been injured on multiple occasions. Signs of fracture healing, including periosteal reaction, callous formation, or remodeling are first seen approximately 7 to 10 days from the time of the injury.

The fractures that are most specific for abuse include rib fractures in children of any age, especially at the posterior portion of the rib, classic metaphyseal fractures, fractures of multiple ages, fractures of the hands and/or feet, and fractures of the scapula, spinous processes, or sternum.

Management Disposition

Fractures that raise a reasonable concern for abuse should be reported to child protective services. Rib fractures and metaphyseal fractures, though very specific for abuse, are frequently self-limited and do not require immobilization. Children with inflicted fractures require identification of a safe care environment before discharge.

Pearls

1. The physical examination is not sensitive for bony injury, especially in young infants. Bruising is absent in the majority of abusive fractures.
2. Skeletal survey is mandatory for children less than 24 months old with concern for physical abuse. Skeletal surveys are less likely to be helpful in children older than 60 months (5 years).
3. Radiographic signs of healing (periosteal reaction, callous) are typically seen approximately 7 to 10 days from the time of injury.
4. A follow-up skeletal survey, obtained 10 to 14 days after the initial survey, can identify additional fractures, especially of the ribs, and long bones. Films of the skull are omitted from these follow-up surveys.
5. Suspect abuse when a child has metaphyseal fractures, rib fractures, fractures at different stages of healing, or unsuspected fractures.
6. Toddler's fractures (spiral fractures of the tibia) are nonabusive fractures common in children learning to walk.
7. Many infants with rib fractures will have minor or nonspecific (fussy, decreased appetite) symptoms.

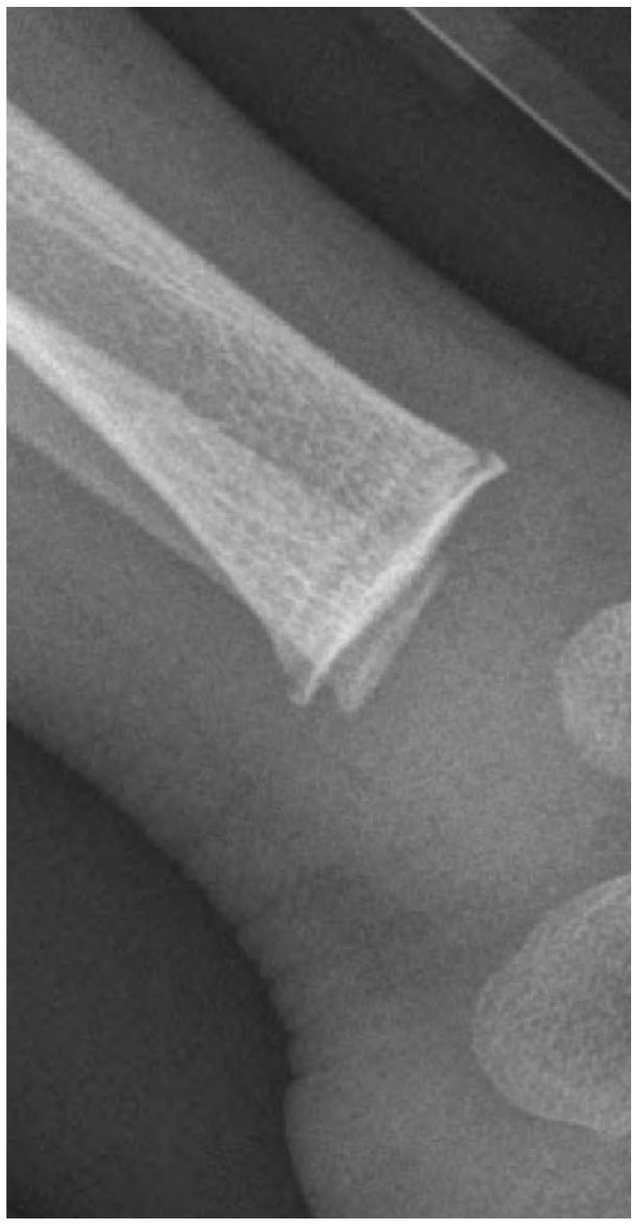


FIGURE 15.44 ■ **Classic Metaphyseal Lesion (CML).** High-detail imaging demonstrates the corner fracture morphology of this metaphyseal fracture. These fractures are rarely symptomatic. (Photo contributor: Marguerite Care, MD.)

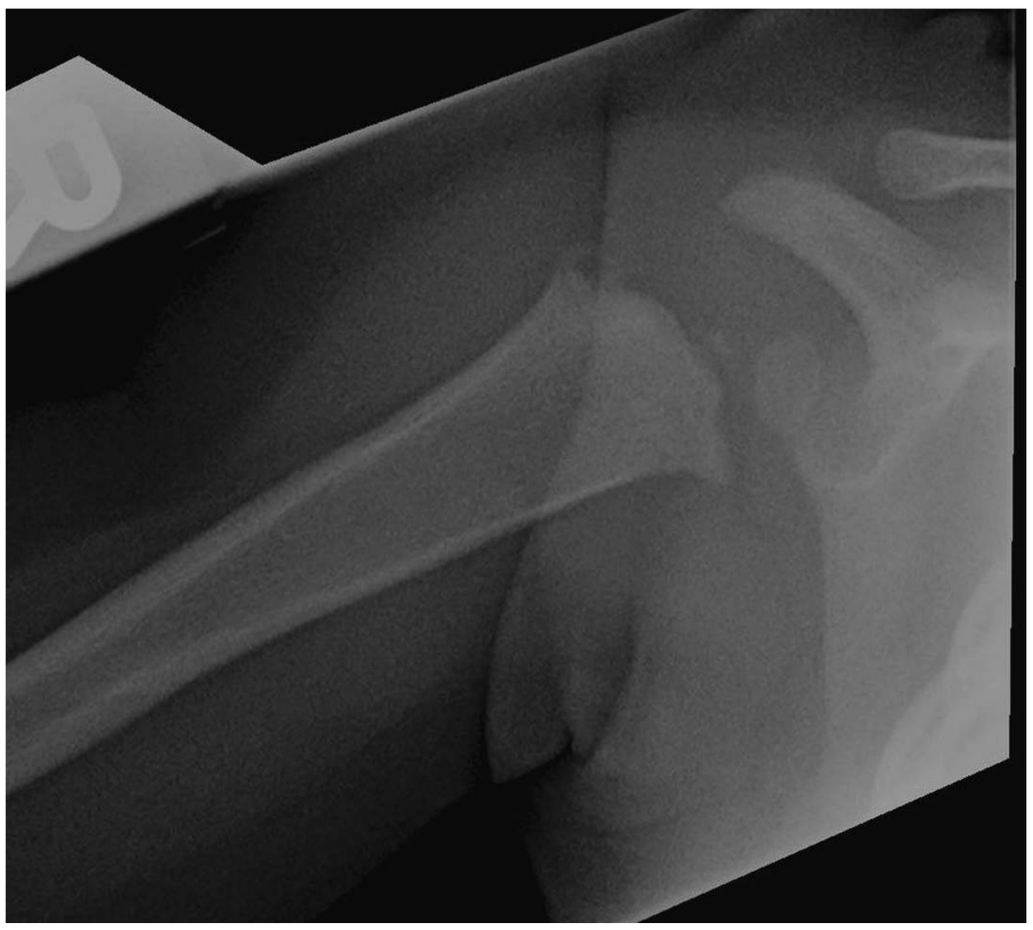


FIGURE 15.45 ■ **Classic Metaphyseal Lesion (CML).** Skeletal survey demonstrates a bucket-handle morphology of this metaphyseal fracture of the proximal humerus. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.46 ■ **Classic Metaphyseal Lesion (CML).** Two views of this metaphyseal fracture (**A**, **B**) demonstrate that the "corner fracture" or "bucket handle" morphology of these fractures is a function of the view obtained, rather than properties intrinsic to the fracture itself. (Photo contributor: Cincinnati Children's Hospital Medical Center.)

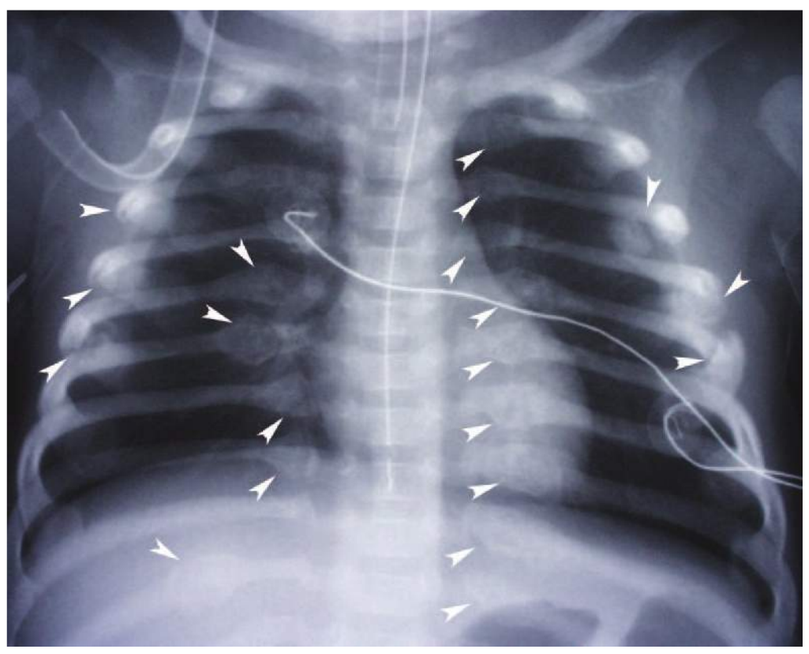


FIGURE 15.47 ■ **Posterior Rib Fractures—Healing.** This A/P chest x-ray demonstrates multiple, bilateral, acute, and healing rib fractures in the posterior portion of the ribs adjacent to the spine (arrowheads). Posterior fractures result from levering of the ribs against the transverse processes of the vertebrae. (Photo contributor: Cincinnati Children's Hospital Medical Center.)

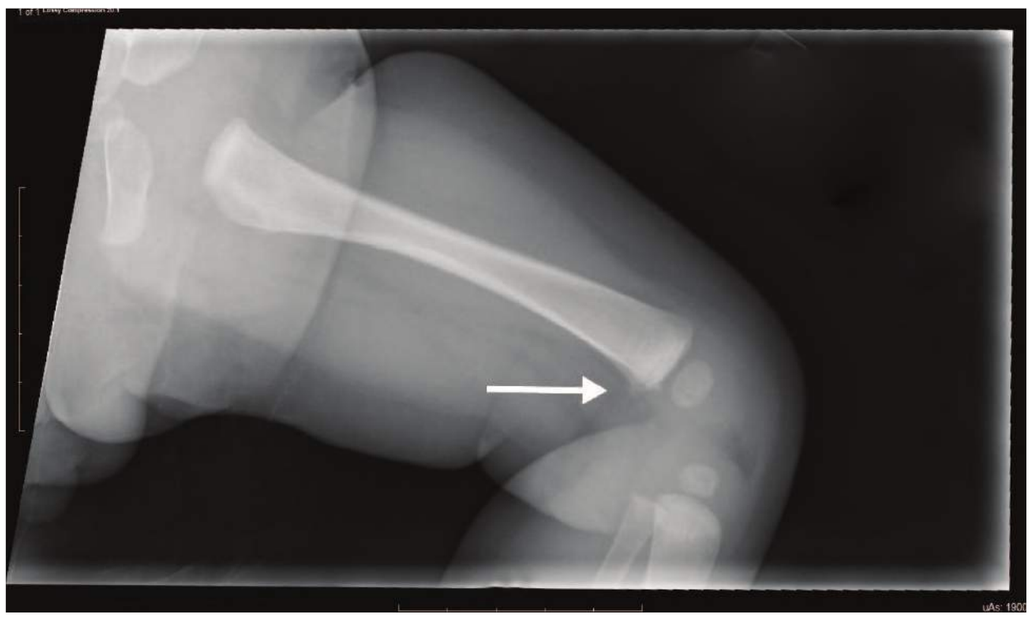


FIGURE 15.48 ■ **Classic Metaphyseal Lesion (CML).** This CML of the distal femur appears as a chip at the posterior corner of the metaphysis. (Photo contributor: Cincinnati Children's Hospital Medical Center.)

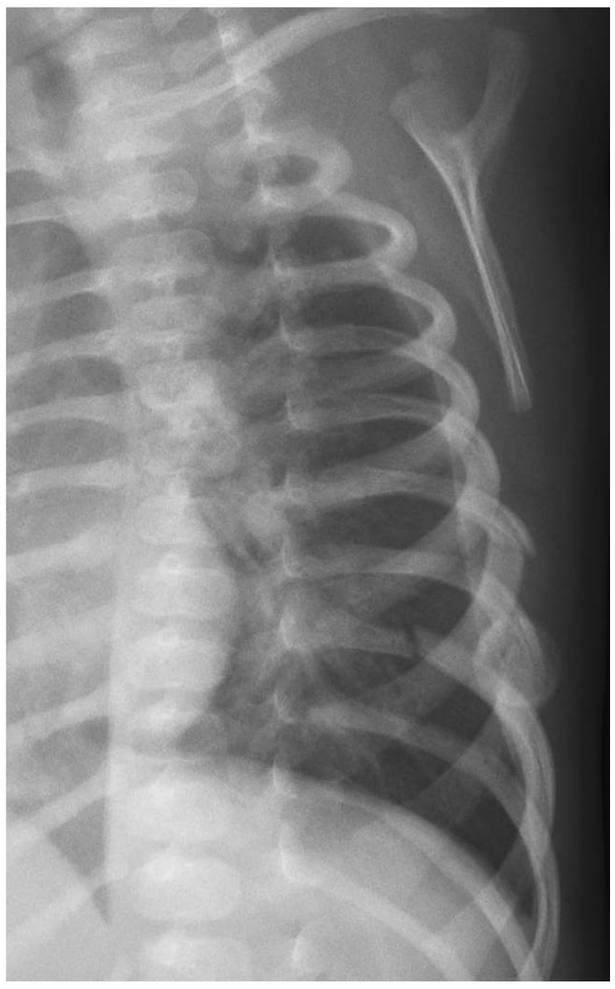


FIGURE 15.49 ■ **Rib Fractures—Acute and Healing.** This oblique radiograph demonstrates acute fractures of ribs 6 and 8 and a healing fracture of rib 7, implying multiple episodes of trauma. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.50 ■ **Blue-Gray Sclera.** This school-aged child has osteogenesis imperfecta and a history of multiple fractures. While some patients demonstrate the classic "robin's egg blue" sclera, a blue-gray tint is more common, and some patients have normal scleral pigmentation. (Photo contributor: Daniel M. Lindberg, MD.)

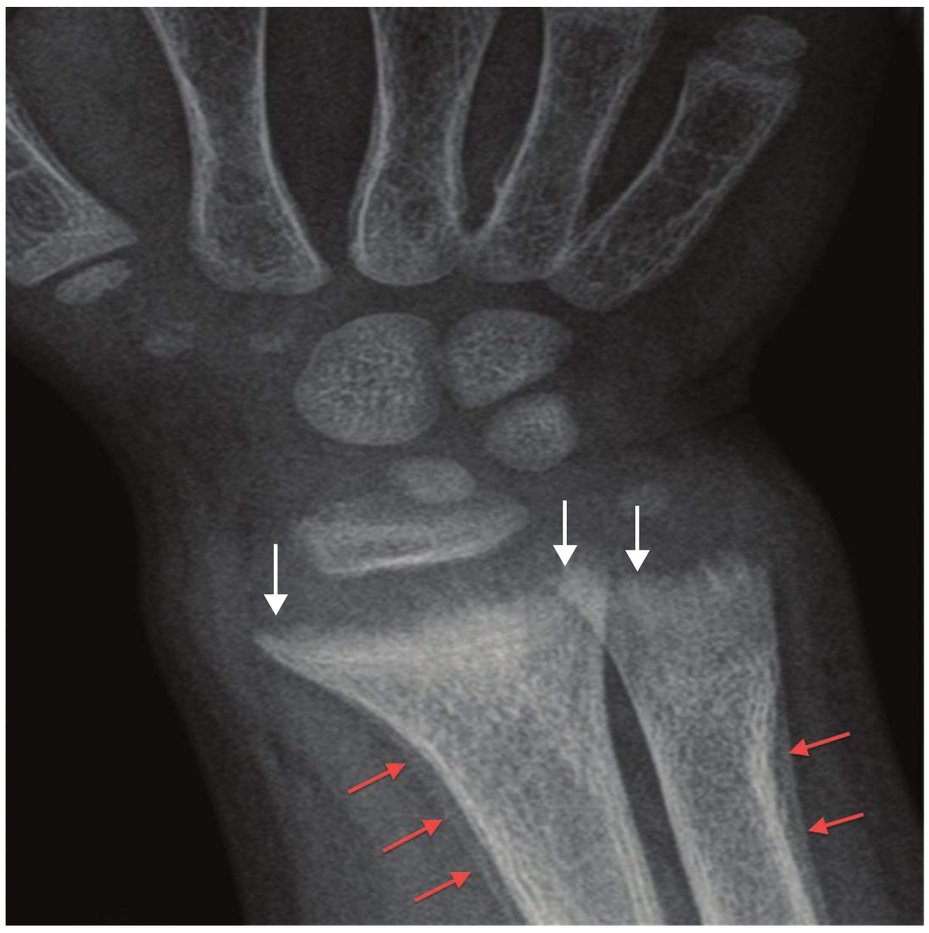


FIGURE 15.51 ■ **Skeletal Changes with Ricketts.** Cupping of the metaphysis (proximal to the growth plate), as shown here in both the radius and ulna, is one of the earliest signs of Rickets or calcium deficiency (open arrows). Periosteal reaction is demonstrated along the shaft of both bones (arrows). (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.52 ■ **Toddler's Fracture.** The toddler's fracture is a commonly seen, nonabusive, spiral fracture of the distal tibia that often presents in children learning to walk, sometimes without a specific history of trauma beyond the normal falls associated with this developmental stage. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.53 ■ **Phalanx Fracture (Second, Third Metacarpal).** Fractures to the phalanges of the fingers or toes are rare in both accidents and abuse. Without a history of significant trauma, identification of these fractures, which may be very subtle, should prompt a thorough evaluation for child abuse. (Photo contributor: Cincinnati Children's Hospital Medical Center.)

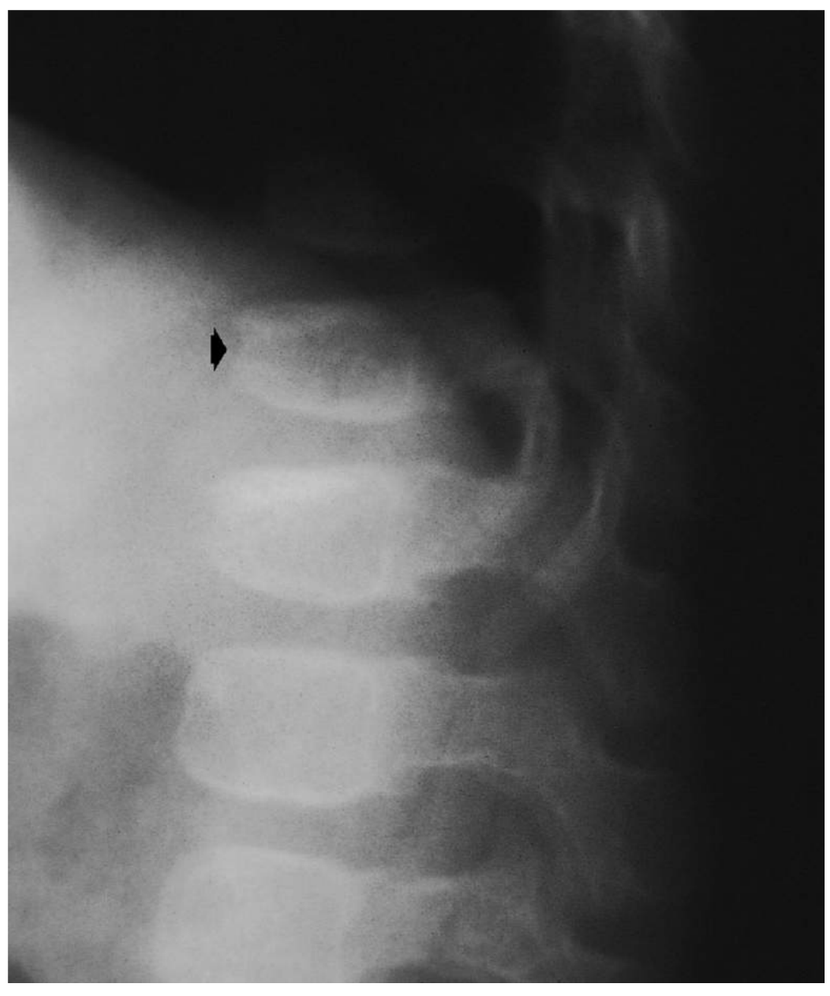


FIGURE 15.54 ■ **Vertebral Fracture.** The wedging of T-12 (arrow) and probably L-1 indicates vertebral compression fractures. These fractures are the result of significant forces applied to the spinal column and are often indicative of child abuse. (Photo contributor: Alan E. Oestreich, MD.)

Clinical Summary

Intra-abdominal and intrathoracic injuries are identified in a small but important fraction of abused children, but can be an important source of morbidity and mortality, and can significantly affect the plausibility of an offered history for other injuries. Clinical signs of abdominal injury (tenderness, bruising, distention, altered bowel sounds), though relatively specific for injury, are relatively insensitive. Hepatic transaminases (AST and ALT) can identify abdominal injuries that are missed by clinical examination alone and should be obtained in all children less than 5 years old with concern for physical abuse. When AST or ALT are more than 80 IU/L, abdominal CT with IV contrast should be obtained to identify intra-abdominal injury.

Significant intra-abdominal injuries are uncommonly the result of short falls or stairway falls, though relatively low-energy injuries with a direct blow to the abdomen (such as a fall onto handlebars) can produce significant injuries.

Most solid-organ injuries in well-appearing children are self-limited, but hollow-viscus injuries, pancreatic injuries, and vascular injuries have high likelihood of deterioration, and require early surgical consultation.

Management and Disposition

Children with identified intra-abdominal or intrathoracic injuries require admission for observation and surgical consultation. The identification of abusive intra-abdominal or intrathoracic injuries should prompt screening for other abusive injuries with a careful physical examination, skeletal survey (for children less than 24 months old), and neuroimaging (for children less than 6 months old or with signs of brain injury).

Pearls

1. Abdominal CT with IV contrast is the first choice for imaging in children with suspicion of abusive abdominal injuries as ultrasound has limited sensitivity.
2. Hollow-viscus injuries, vascular injuries, and pancreatic injuries have a high likelihood for decompensation and require early surgical consultation.
3. Neither a normal ultrasound nor declining liver function tests obviate the need for a CT—if concerned, get a contrast-enhanced CT without delay.
4. Many children with significant abusive abdominal injury will have no bruising on physical examination.



FIGURE 15.55 ■ **Abdominal Bruises.** Abdominal bruising is unlikely in accidental injury and frequently indicates intra-abdominal injury. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.56 ■ **Positive FAST Examination.** This anechoic (dark) fluid in Morrison pouch represented blood in this patient. As with nonabusive trauma, ultrasound is a rapid, noninvasive way to screen for abdominal injury in an unstable patient, but is not sufficiently sensitive to detect smaller abdominal injuries that may, nevertheless, have important forensic significance. (Photo contributor: Jason W. Fischer, MD, MSc.)

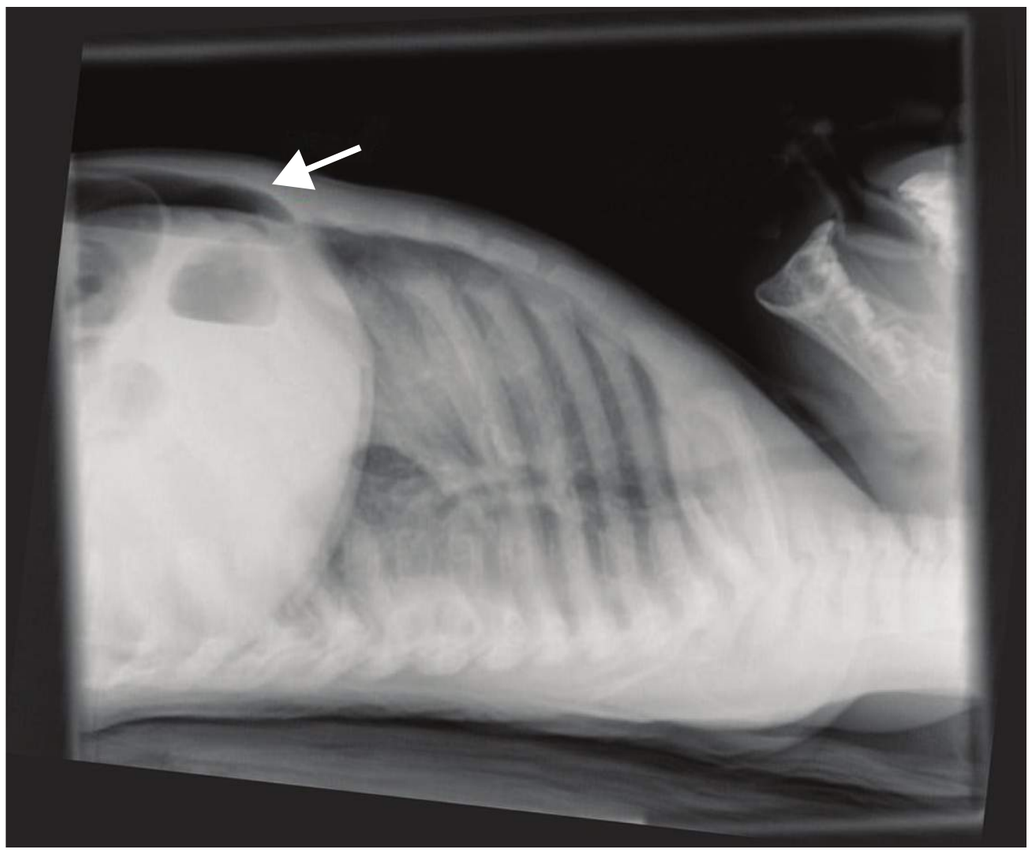


FIGURE 15.57 ■ **Hollow Viscus Perforation.** This lateral chest x-ray demonstrates free air anteriorly, inferior to the diaphragm, suggesting hollow viscus perforation, an injury that, like pancreatic injuries, is overrepresented in abused children. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.59 ■ **Splenic Laceration.** This splenic laceration was identified in a child with abusive head trauma and retinal hemorrhages in the course of an investigation for unexplained anemia. (Photo contributor: Daniel M. Lindberg, MD.)

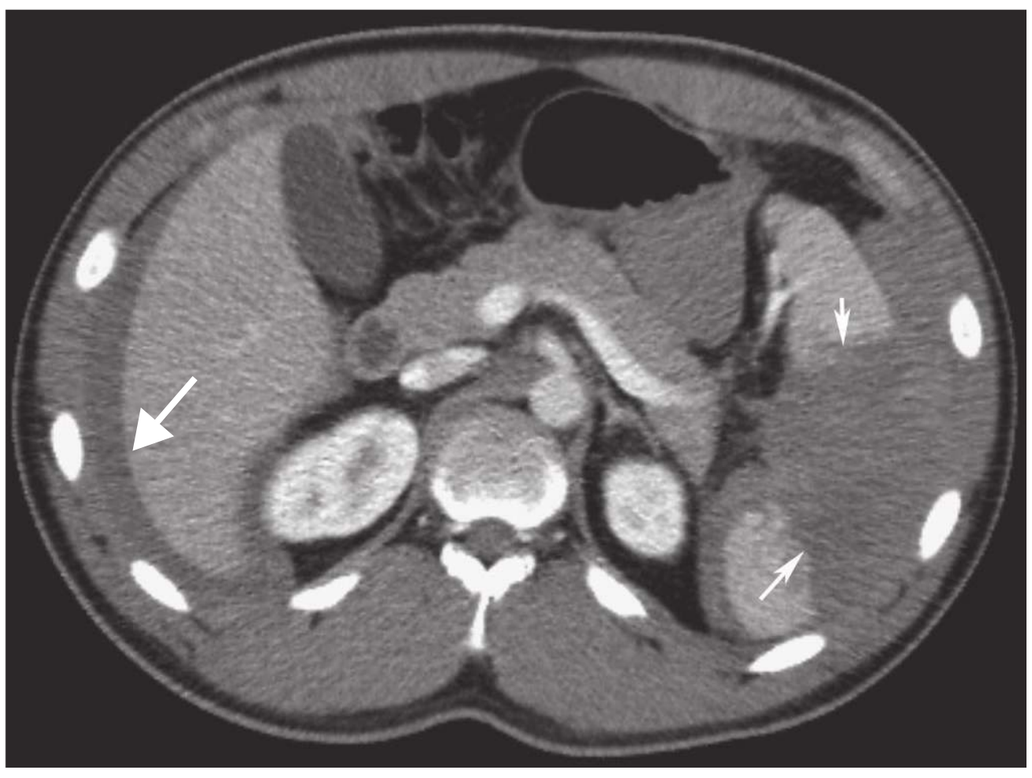


FIGURE 15.58 ■ **Splenic Laceration.** A severe laceration of the spleen is seen. Note the large separation of the splenic parenchyma with fluid density (blood) visible between the sections of the spleen (white arrows) and free fluid within the peritoneum (black arrow). In young children, motor vehicle collisions and abuse account for the large majority of such injuries. (Photo contributor: Marguerite Care, MD.)

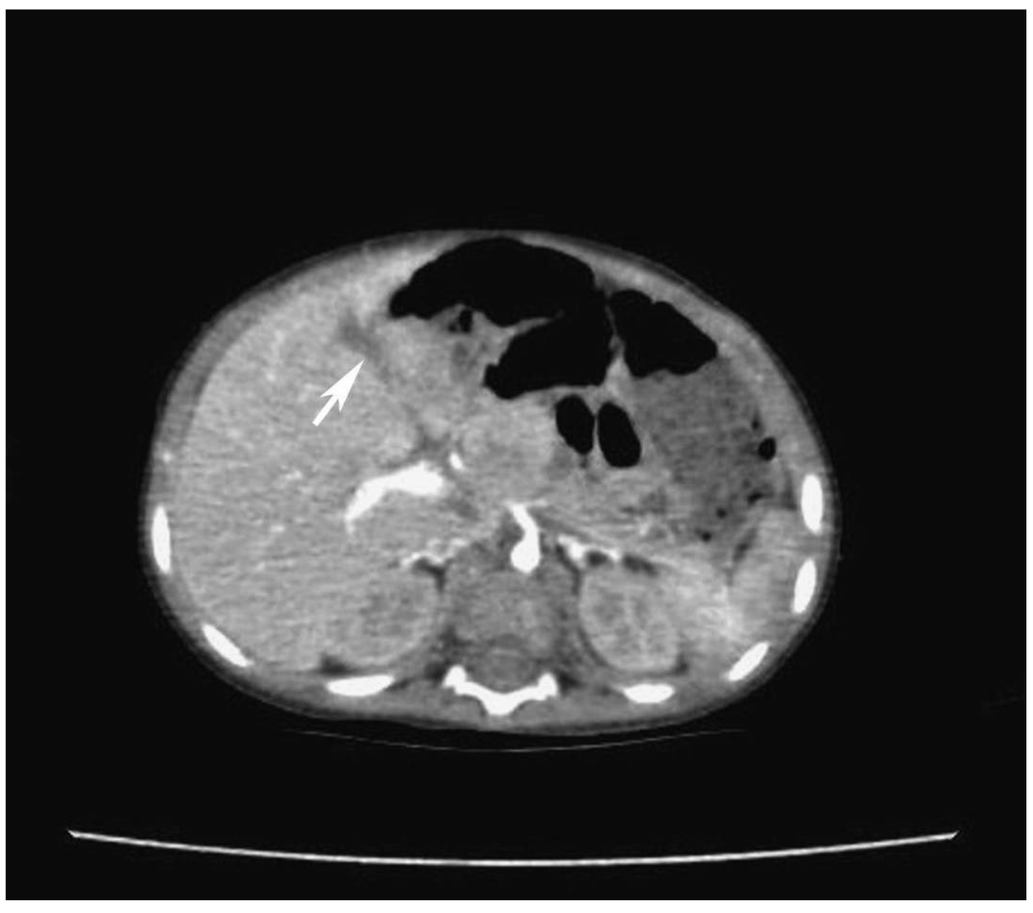


FIGURE 15.60 ■ **Abusive Liver Laceration.** This CT demonstrates a clinically occult hepatic laceration (arrow) identified in a child with other abusive injuries. (Photo contributor: Cincinnati Children's Hospital Medical Center.)

CHILD SEXUAL ABUSE EXAM AND GENITAL FINDINGS

Clinical Summary

The history is often most important to make a diagnosis of sexual abuse. An ED physician may ask questions as necessary for the medical evaluation of the patient. However, forensic interviews should be performed by professionals trained in interviewing children for possible sexual abuse.

A pubertal female can be examined in the supine position with feet in stirrups. A prepubertal female should be examined in the supine frog-leg position (hips and knees bent; soles of feet touching) and can be examined on the examination table or while sitting on a caregiver's lap. Gentle even labial traction will allow visualization of the vulva and the hymen. Attention should be paid to all areas, but special attention should be given to the hymen and posterior fourchette, the most common sites of injury in cases of sexual abuse. Use the clock-face designation when documenting locations around the hymen. A cotton-tipped applicator can be used in the postpubertal female to unfold and examine the edges of the hymen. The prepubescent hymen is sensitive to touch. A speculum is never used in the examination of a prepubertal girl; the only time a speculum is used in this population is during examination under anesthesia.

A male patient can be examined in the supine position, with care to inspect the penis and scrotum in entirety. The anus can be examined in the prone or lateral decubitus position. A colposcope can be used, if available, to magnify the genital and anal areas to look for injury.

Management and Disposition

When patients with a concern of sexual abuse present to the ED, Social Work should be notified. If sexual abuse is suspected, a report of alleged sexual abuse should be made to child protective services and law enforcement.

All children who are seen in the emergency room for acute sexual assault should receive a complete physical examination including genital and anal examination. Any injuries or skin lesions concerning for injuries on the body should be described in detail with the aid of full body diagrams and, when available, photodocumentation. When performing a genital examination, a chaperone (hospital personnel) should be present at all times.

Evidence collection should be performed if the contact occurred within the last 72 hours (for all males and prepubertal females), and can be considered up to 96 hours in pubertal females, as there is the potential for the recovery of trace forensic evidence.

In most cases, an anxious patient will cooperate fully with the examination if a parent or other support person remains for the examination. Reassurance and distraction techniques are helpful. Sedation is rarely needed and can be ineffective. An examination should never be forced upon an unwilling patient. When an examination is deemed medically necessary (ie, vaginal bleeding without a known source, and the patient is not cooperative), examination under anesthesia should be considered.

Sexual assault can coincide with intentional or covertly administered substance use. Do not begin the evidence collection or genital/anal examination until the patient is coherent and can consent or assent to the process and examination.

Pearls

1. A normal examination does not exclude the diagnosis of sexual abuse, and a normal examination is the most likely finding, even in abused children. Examination findings (genital and anal) specific for sexual abuse are found in only a small percentage of patients who report a history of sexual abuse.
2. The shape and appearance of the prepubertal hymen is variable. Crescentic and annular configurations are most common. Hymens can also be sleeve-like, septate, imperforate (which eventually requires surgical opening), and cribriform (multiple small openings).

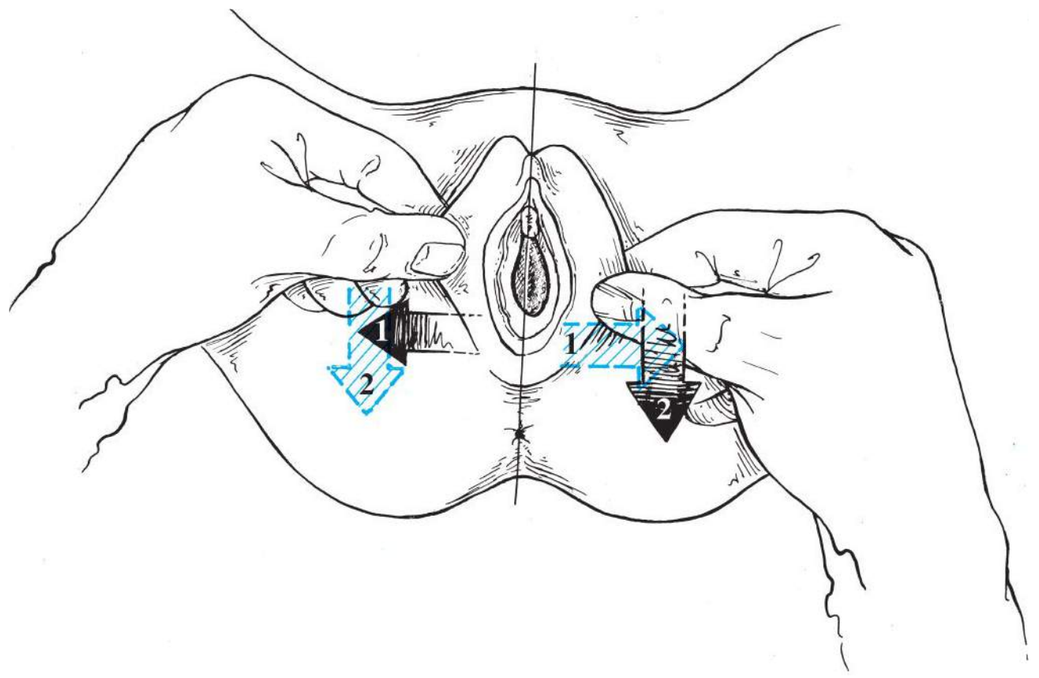


FIGURE 15.61 ■ **Labial Traction Examination Technique.** Position the child in a supine position with her knees out and soles together. Hymenal inspection in prepubertal girls is best accomplished when lateral (**1**) and posterior (**2**) traction to the labia is applied as shown here. (Adapted from Giardino AP et al. A Practical Guide to the Evaluation of Sexual Abuse in the Prepubertal Child. Sage Publications; 1992.)

3. The inner edge of the prepubertal hymen is usually smooth and uninterrupted. Notches at the 3- and 9-o'clock positions are normal. Minor irregularities are most likely insignificant.
4. Presumptive treatment for gonorrhea and chlamydia is not recommended for a **prepubertal** patient because of the very low incidence of infection in these patients and also because of the low risk of ascending infection (infection is a lower-tract disease in prepubertal females) and the need for confirmatory testing.
5. A history of sexual abuse is strongly associated with increased risk of suicidal thinking and suicide and self-harm attempts among older children and adolescents. All patients evaluated for sexual abuse should be carefully assessed for current suicidal or self-harm thoughts.
6. Before the patient is discharged from the ED, it is necessary to also determine what access the alleged perpetrator has to the patient, family, or other individuals and to ensure a safe discharge plan.

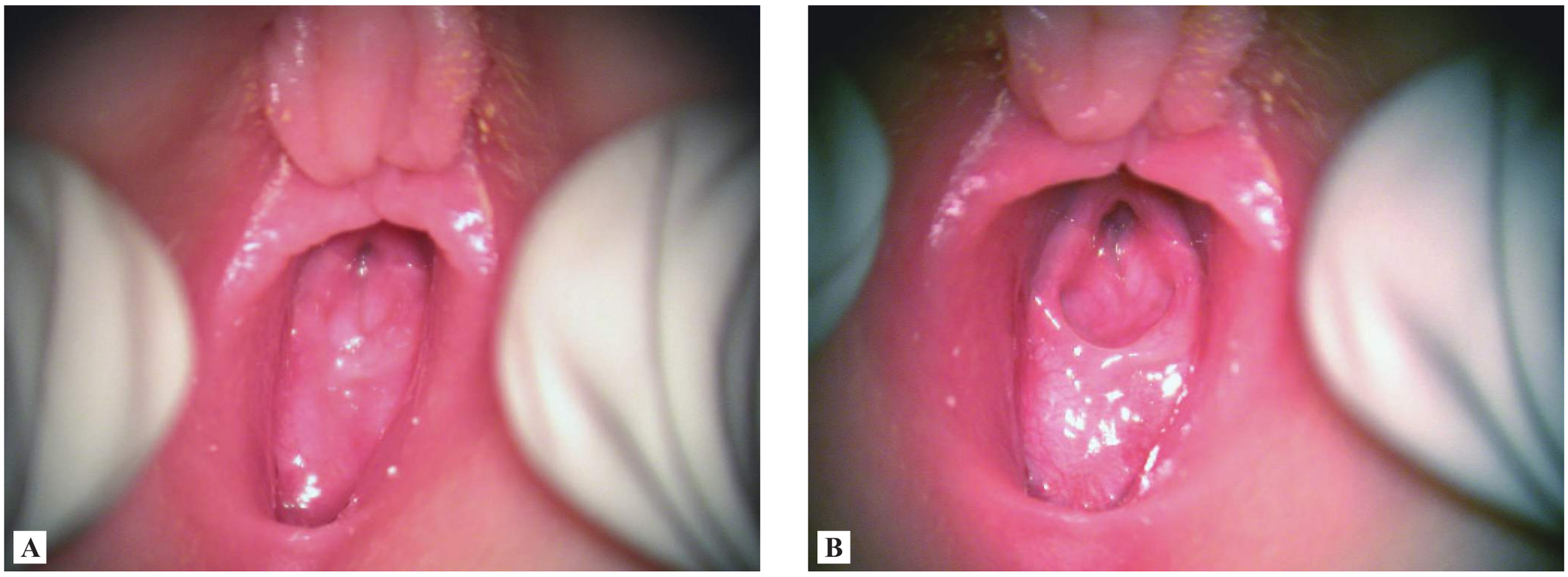


FIGURE 15.62 ■ **Proper Labial Traction Allows Visualization of the Prepubertal Hymen.** (**A**) Lateral labial traction only. The hymenal margins cannot be visualized. (**B**) Lateral, posterior, and caudal labial traction provides complete visualization of the hymenal rim. (Photo contributor: Cincinnati Children's Hospital Medical Center.)

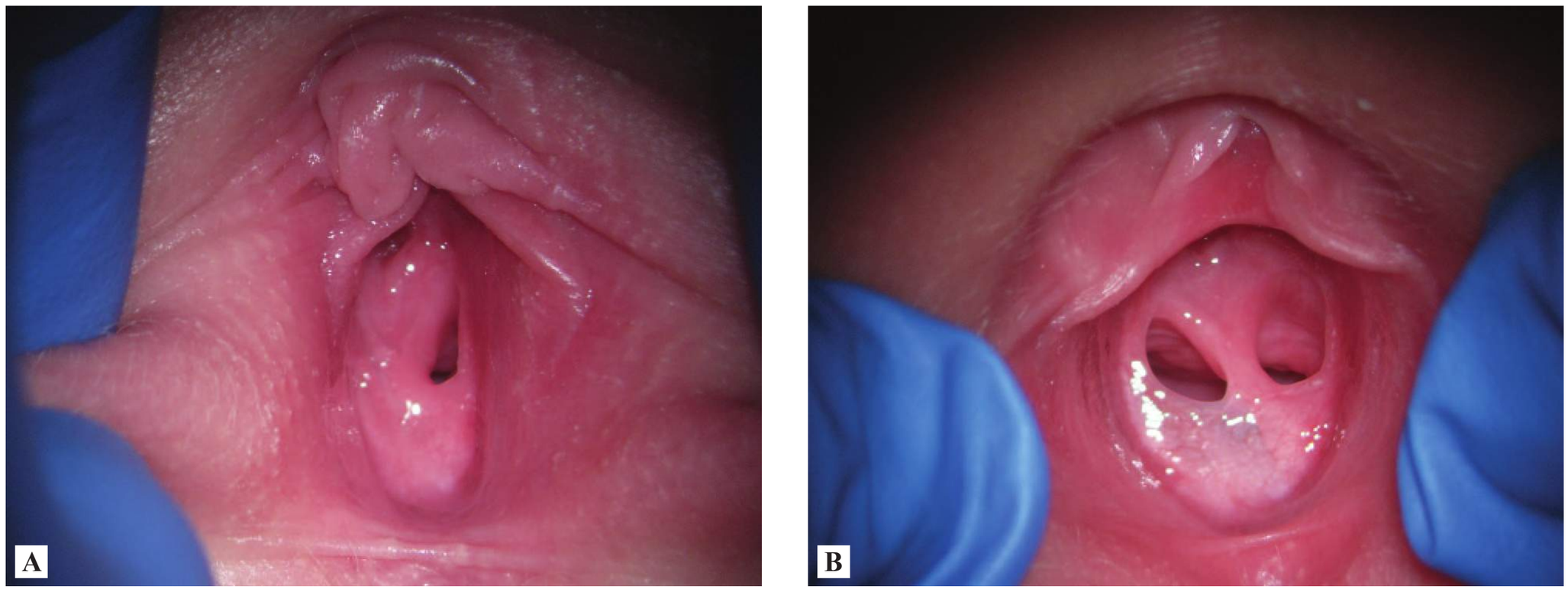


FIGURE 15.63 ■ **Proper Labial Traction Allows Visualization of the Prepubertal Hymen.** The hymen is partially visible in the first photo (**A**). When traction is applied, a hymenal septum is found which creates the appearance of two vaginal openings (**B**). A septum is a normal hymenal variant. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.64 ■ **Normal Estrogenized Newborn Hymen.** Infants often have thickened annular hymens. Maternal estrogens cause this effect. Do not confuse with traumatic swelling. (Photo contributor: Cincinnati Children's Hospital Medical Center.)

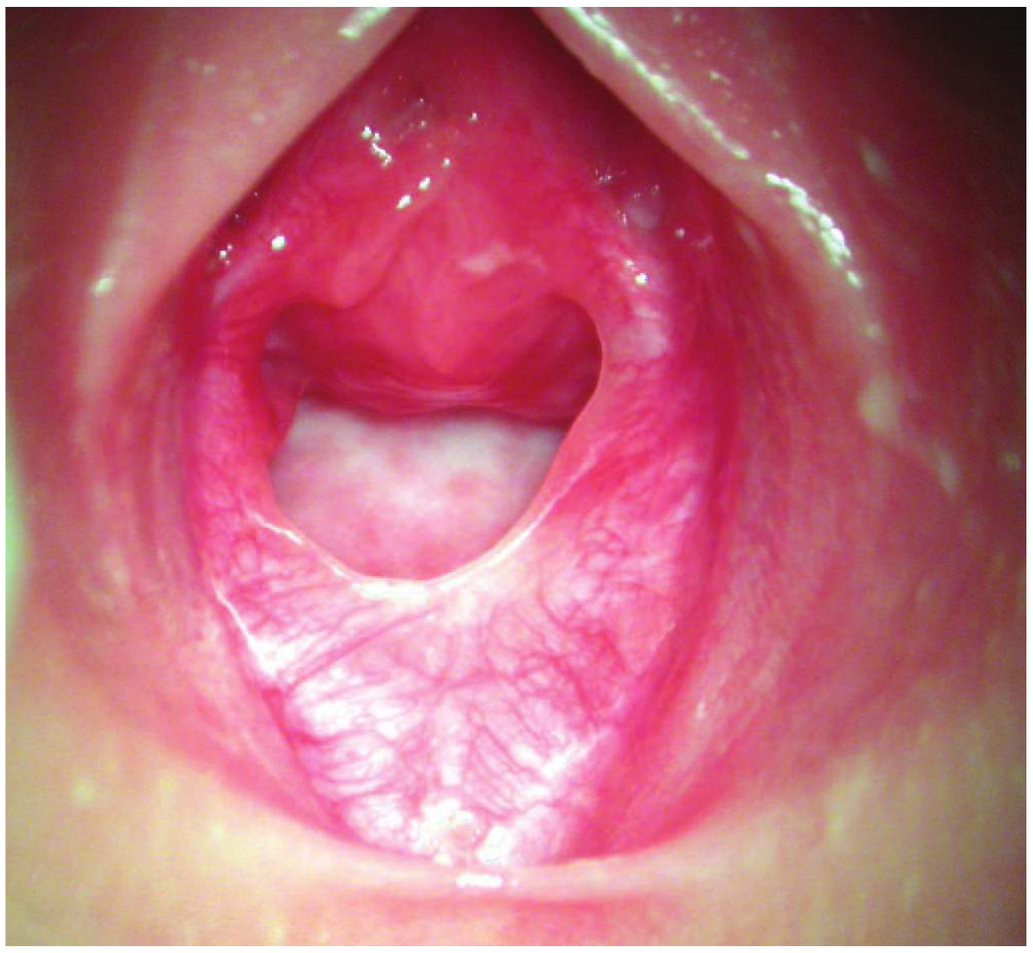


FIGURE 15.66 ■ **Normal Crescentic Hymen.** The hymen is crescentic shaped without any hymen present from the 11- to 1-o'clock position. This is a normal hymen and the most common configuration in prepubertal girls. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.65 ■ **Normal Annular Hymen.** The hymen is doughnut shaped without any defects. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.67 ■ **Normal Hymenal Mound—Crescentic Hymen.** The hymen is crescent shaped, tissue is absent near the urethra between 11- and 1-o'clock position. A nonspecific mound of hymenal tissue is noted at 3-o'clock position. (Photo contributor: Cincinnati Children's Hospital Medical Center.)

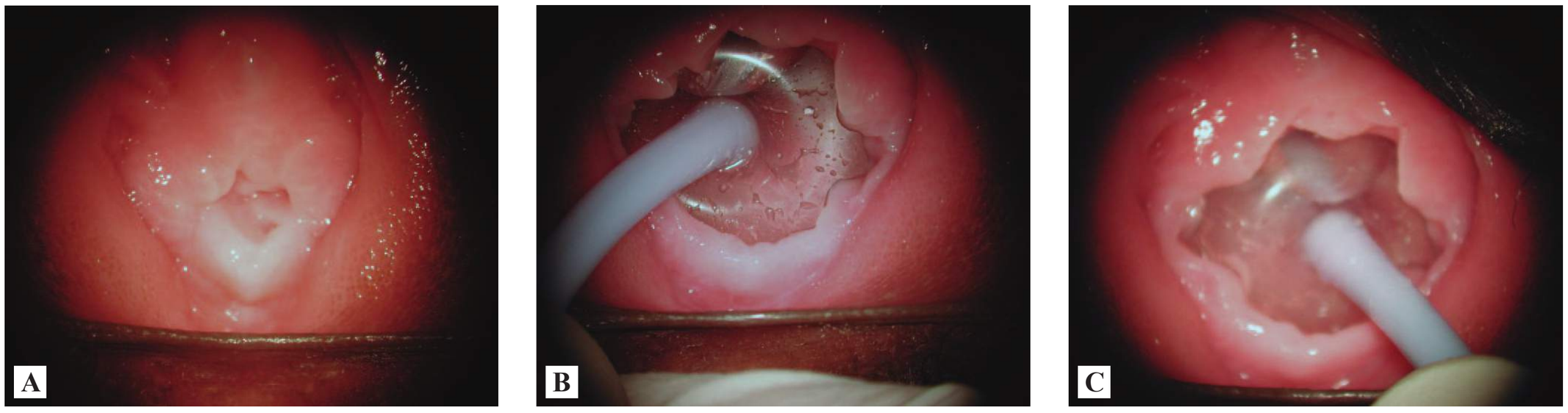


FIGURE 15.68 ■ **Foley Catheter Technique to Better Visualize the Adolescent Hymen.** In addition to using a cotton-tipped applicator (**A**). A Foley catheter can be inserted into the adolescent vagina, filled a mixture of air and water and gently retracted (**B**). Side-to-side displacement of the catheter exposes different hymenal sections for inspection (**C**). (Photo contributor: Cincinnati Children's Hospital Medical Center.)

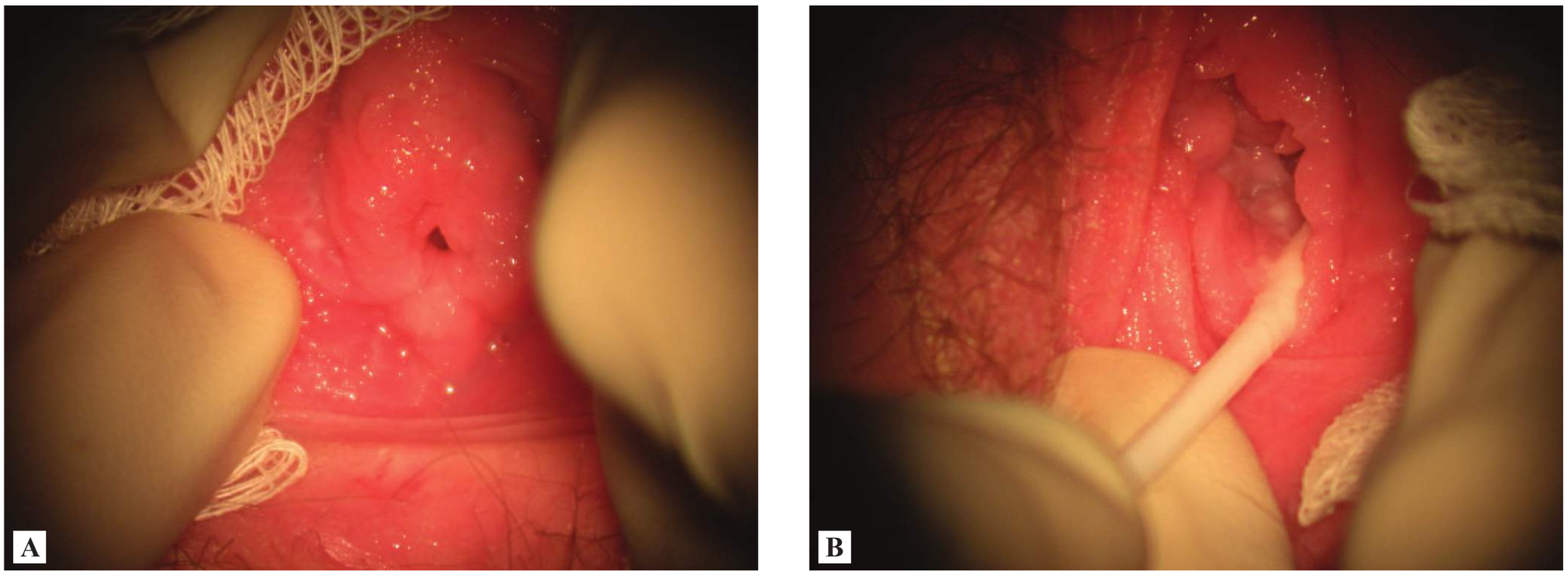


FIGURE 15.69 ■ **Cotton Swab Technique.** These images (**A**, **B**, **C**) show how a cotton-tipped applicator is used to ensure that all parts of the hymen are visualized during the examination of a pubertal patient. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.70 ■ **Hymenal Septum.** This picture shows a common hymenal variant—hymenal septum. A septum that extends into the vagina must be excluded either by examination (demonstrating that a cotton-tipped applicator can pass behind the septum) or with an ultrasound. (Photo contributor: Kathi L. Makoroff, MD.)

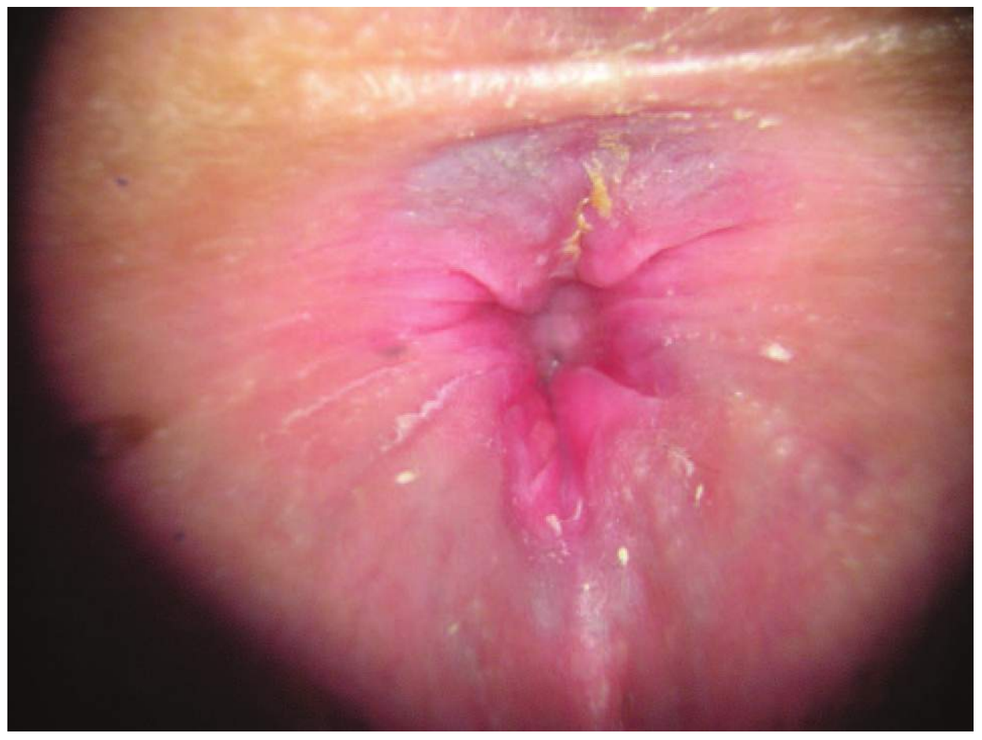


FIGURE 15.71 ■ **Normal Anal Dilation.** A relaxed external sphincter causes anal dilation in all children. Stool in the rectal vault contributes to dilation. This is not a finding of sexual abuse. Other conditions that can cause anal dilatation include chemical sedation, death, or a prolonged period of hypoxia. (Photo contributor: Cincinnati Children's Hospital Medical Center.)

INJURIES AND FINDINGS INDICATIVE OF GENITAL OR ANAL TRAUMA

Clinical Summary

Most physical examinations in cases of sexual abuse or assault will be normal. Findings specific for acute trauma, although not necessarily diagnostic of sexual abuse or assault, include bruising, lacerations, and abrasions. Most of these injuries will heal quickly, usually within a few days, and will heal completely without scarring. Nonacute examination findings caused by trauma can be difficult to diagnose and should be interpreted by a qualified child abuse expert.

Sexually transmitted infections diagnosed in a prepubertal child may indicate sexual abuse. Neisseria gonorrhoeae, Chlamydia trachomatis, Trichomonas, and syphilis are almost always transmitted by intimate sexual contact (intimate contact with infected secretions or lesions) unless acquired perinatally. Human immunodeficiency virus (unless acquired perinatally) and hepatitis are transmitted by intimate sexual contact or contact with infected blood. Condylomata acumintata (human papillomavirus or genital warts) and herpes simplex virus may be transmitted to the prepubertal child through sexual or nonsexual contact.

Management and Disposition

Victims of an acute sexual assault may have genital and/or anal bleeding. Patients who have active genital and/or anal bleeding need immediate attention and evaluation.

Also see "The Child Sexual Abuse Examination."

Pearls

1. It is not necessary to measure the vaginal opening of prepubertal girls. The size of the opening is dependant on examination technique and degree of patient relaxation. There is no consensus of normal size opening among experts.
2. Eyelash lice are pubic lice, and sexual abuse must be considered.
3. Rectal abuse often causes no visible trauma. Superficial anal fissures can be common findings in children; consider abusive trauma, however, if deep fissures are present.



FIGURE 15.73 ■ **Posterior Fourchette Laceration.** A laceration is present in the posterior fourchette, after sexual assault. This is a common location for injury after sexual assault/abuse. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.72 ■ **Hymenal Bruise.** The hymen has bruising from 3- to 5-o'clock position after an acute sexual assault. (Photo contributor: Cincinnati Children's Hospital Medical Center.)

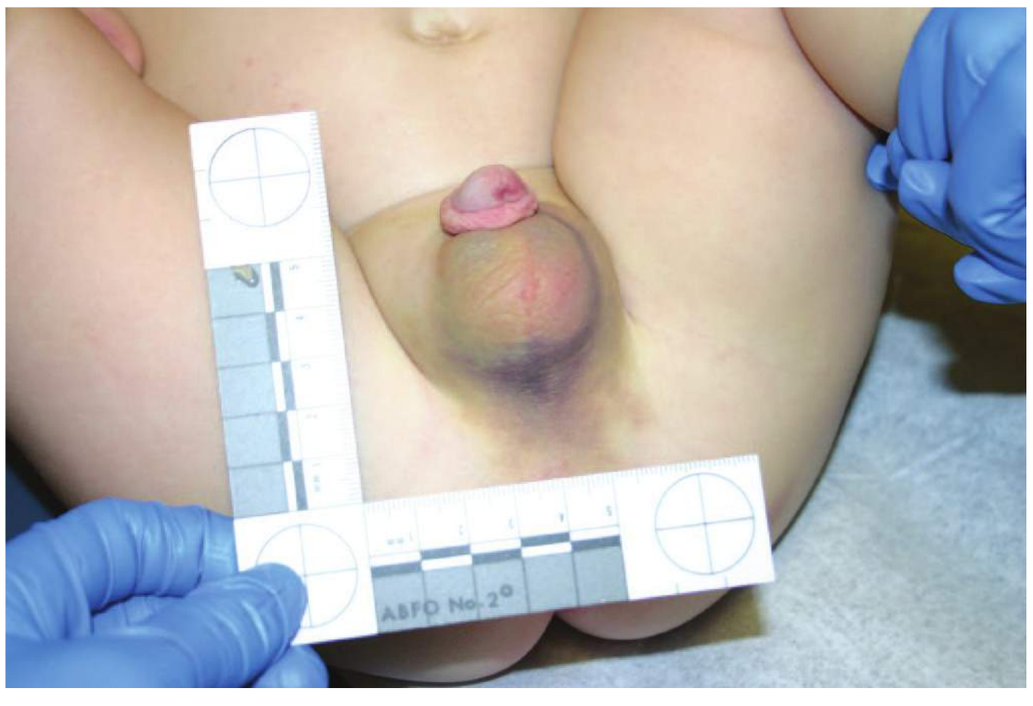


FIGURE 15.74 ■ **Scrotal Bruise.** Bruising to the scrotum is present as well as an abrasion to the tip of the penis. Physical abuse as well as sexual abuse should be considered when genital injury is noted. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.75 ■ **Penile Abrasion.** Abrasion to the underside of the penis illustrates that it is important to examine all parts of the genitalia. (Photo contributor: Daniel M. Lindberg, MD.)

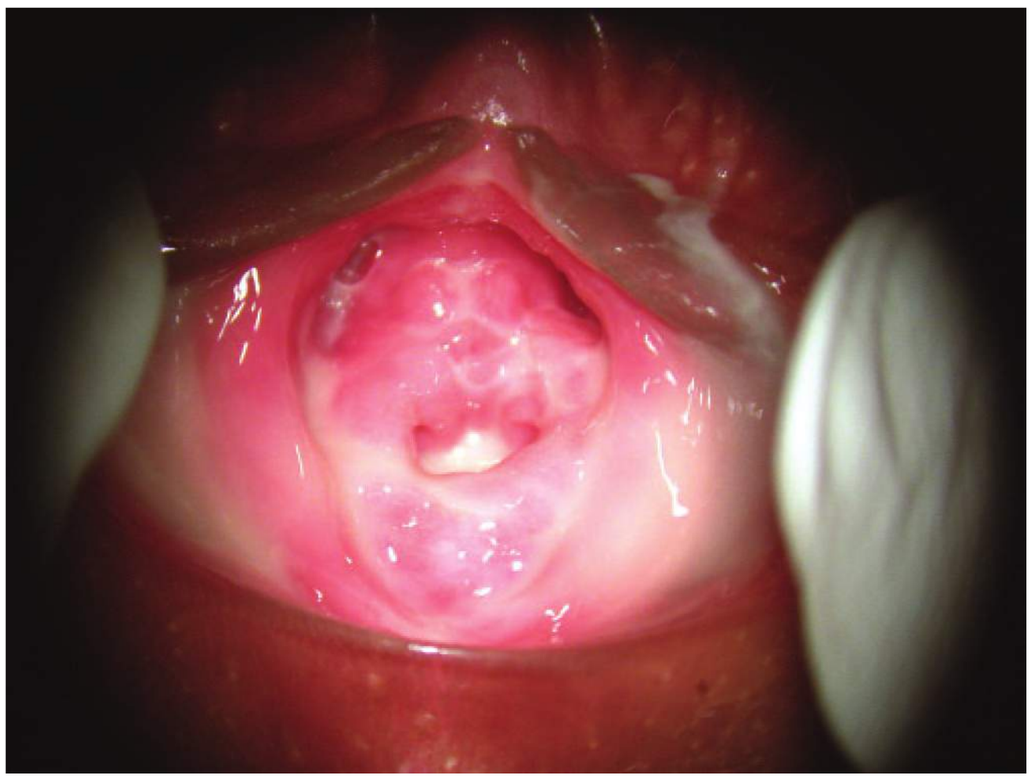


FIGURE 15.77 ■ **Vaginal Discharge—Neisseria gonorrhoeae.** Vaginal discharge in a prepubertal child may be an indication of an STI even when a history of abuse is denied. All children with vaginal discharge should be cultured for N gonorrhoeae and Chlamydia. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.76 ■ **Acute Deep Perianal Lacerations and Bruising.** An acute rectal laceration is visible at 8-o'clock position with bruising on the right side of the perianal area. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.78 ■ **Condyloma Accuminata.** Several small warts are visible in the perineum in this young child. (Photo contributor: Hope K. Haefner, MD.)



FIGURE 15.79 ■ **Perianal Condyloma Accuminata.** Several perianal warts are visible. Warts can be transmitted sexually and nonsexually. Workup should include an evaluation (examination, forensic interview) for sexual abuse and testing for other STIs. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.80 ■ **Perianal Condyloma Accuminata.** Extensive perianal warts are visible. The diagnosis of genital or anal warts does not automatically mean that they were sexually transmitted (see above). (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.81 ■ **Perianal Condyloma Lata (Secondary Syphilis).** Perianal condyloma lata are visible around the rectum. This should not be confused with condyloma accuminata. (Photo contributor: Cincinnati Children's Hospital Medical Center.)

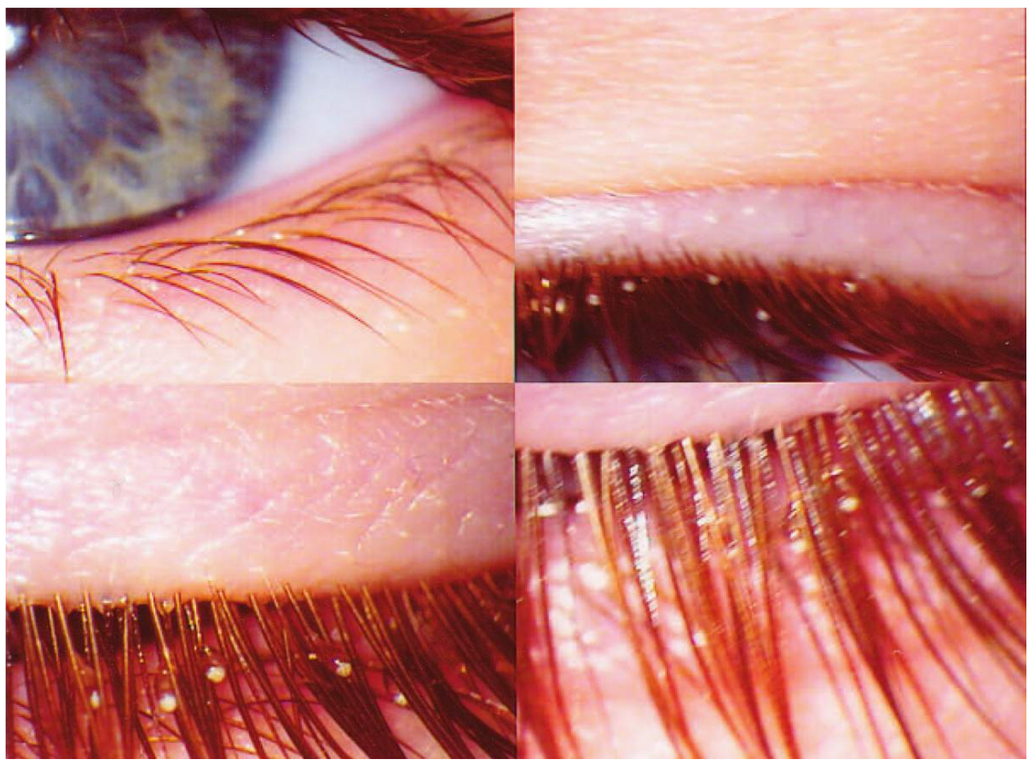


FIGURE 15.82 ■ **Nits.** Nits (the larval form of the louse) from Pthirus pubis are seen firmly adherent to the eyelashes in this child. Sexual abuse should be strongly considered; these are not from head hair. (Photo contributor: Robert A. Shapiro, MD.)

Clinical Summary

Genital injury can result from a fall (straddle) onto an object. A straddle injury usually causes damage to the external genitalia with unilateral bruising and swelling of labia and periurethral tissues. Occasional penetrating trauma to the hymen with internal injury can be seen. The history and the type of object involved are important historical elements. Always obtain a detailed history from the child (if developmentally appropriate) separately from the accompanying parent/adult. Factors that increase concern of possible sexual abuse include no history of injury, injury in a nonambulatory child, vaginal, or hymenal injury without history of penetrating trauma, history inconsistent with physical findings, and/or additional nongenital trauma.

Management and Disposition

Check for urethral injury. Treatment is usually symptomatic. Sitz baths and antibacterial ointment promote healing and minimize discomfort. If the child has difficulty voiding, encouraged voiding in a bath with a few inches of warm water to reduce dysuria. If severe injuries occur, surgical repair may be necessary with consultation with pediatric gynecology or surgery.

Pearls

1. Straddle injuries usually present with a clear mechanism of injury and a physical examination that supports the history.
2. Straddle injuries do not typically involve the hymen or internal vaginal mucosa.





FIGURE 15.83 ■ **Straddle Injury.** This young child fell on to a pool edge sustaining a hematoma to the left labia majora (**A**, **B**). The remainder of her examination, including her hymen, was normal. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.84 ■ **Straddle Injury.** Bruising to the left labia majora and clitoral hood following a fall onto a bicycle bar. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.85 ■ **Straddle Injury.** Ecchymosis, swelling, and contusion of the perineum in a 3-year-old female child who tripped and fell on a large plastic toy. (Photo contributor: James Mensching, MD.)

MIMICS OF TRAUMA: TOILET SEAT INJURIES

Clinical Summary

Acute bruising to the glans and corona of the penis can occur if the toilet seat falls onto the penis during voiding, trapping the penis between the seat and toilet bowl. This injury is not uncommon in boys of about 3 years of age whose height and development set the stage for this injury.

Management and Disposition

No specific treatment is needed unless the child is unable to void. Consult urology if the child cannot void. Consider sexual abuse in any child with a genital injury.



FIGURE 15.86 ■ **Toilet Seat Injuries.** This toddler presented with a straightforward history of the toilet bowel falling onto his penis while voiding. He was able to void without difficulty. (Photo contributor: Kevin J. Knoop, MD, MS.)

Pearl

1. Genital injuries are suspicious of sexual abuse if no appropriate history of accidental trauma is given.



FIGURE 15.87 ■ **Toilet Seat Injuries.** The toilet seat fell onto this toddler's penis and caused bruising to the penis. Discharge is appropriate if the child can void without difficulty. (Photo contributor: Charles J. Schubert.)

Clinical Summary

Urethral prolapse is a condition that only occurs in females, usually in school-aged children. Patients will present with painless bleeding and, on exam, will have what appears as swelling, but is actually the prolapsed portion of the urethra. Urethral prolapse is commonly mistaken for vaginal injury. The etiology is unknown but estrogen deficiency is thought to be a contributing factor. Other risk factors include increased intra-abdominal pressure that results from coughing or constipation and anatomic defects. Urethral prolapse is more common in African American girls.



FIGURE 15.88 ■ **Urethral Prolapse.** A round reddish-purple donut-shaped mass is seen in this child's introitus. Careful examination reveals that the mass originates from the urethra. The hymen is normal. (Photo contributor: Cincinnati Children's Hospital Medical Center.)

Management and Disposition

Medical treatment includes sitz bath and topical estrogen cream. A referral to urology should also be made, as well as reviewing with the family indications for emergent return including increased pain or bleeding, which are signs of strangulation. Surgical repair may be needed when conservative treatment fails or when the prolapse is necrotic.

Pearls

1. Urethral prolapse often presents with painless bleeding in school-aged girls.
2. An examination using good labial traction (see section "The child sexual abuse examination") will demonstrate the prolapse originating from the urethra.



FIGURE 15.89 ■ **Urethral Prolapse.** A round donut-shaped mass consistent with urethral prolapse is seen in this school-aged child. (Photo contributor: Cincinnati Children's Hospital Medical Center.)

MIMICS OF ERYTHEMA/BRUISING: LICHEN SCLEROSIS

Clinical Summary

Lichen sclerosis atrophicus (LSA) is found often in older women but occasionally in prepubertal girls. Findings include thin hypopigmented (as white or yellow plaques) and easily friable skin in the genital and anal areas that can bleed even without trauma; itching and pain can also occur. The hemorrhagic form of LSA includes subepithelial hemorrhagic lesions to the affected skin, which can be mistaken for trauma. The exact cause is unknown but thought to involve a genetic susceptibility, autoimmune dysfunction, and state of hypoestrogenism.

Management and Disposition

Treatment consists of high potency corticosteroids and referral to dermatology or pediatric gynecology.

Pearls

1. The hemorrhagic form of LSA can be mistaken for trauma.
2. Surrounding hypopigmented skin can be a clue to the diagnosis



FIGURE 15.90 ■ **Lichen Sclerosis.** This image shows both the hypopigmentation (white plaque area) and the multiple hemorrhagic areas characteristic of lichen sclerosis. (Photo contributor: Kathi L. Makoroff, MD.)



FIGURE 15.91 ■ **Lichen Sclerosis.** This close-up image shows the hypopigmentation (white plaque area) and the differentiation between it and the surrounding normal skin. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.92 ■ **Lichen Sclerosis.** Hemorrhagic lichen sclerosis atrophicus. The perineum surrounding the vagina and anus is hypopigmented in an hourglass configuration. The hemorrhagic lesions are not traumatic in etiology. (Photo contributor: Cincinnati Children's Hospital Medical Center.)

MIMICS OF ERYTHEMA/BRUISING: LABIAL ADHESIONS

Clinical Summary

Adhesions of the labia minora occur in young girls and may persist until puberty. A thin translucent line is seen where the labia meet. Involvement is often limited to the posterior portion of the labia, but some children have more extensive adhesions that completely obscure the introitus.

Management and Disposition

Estrogen cream (Premarin) can be prescribed and applied gently over the adhesions twice daily for 2 to 4 weeks. Recurrence is not uncommon.

Pearls

1. Adhesions may be congenital or acquired.
2. It is postulated that vulvar irritation from sexual abuse may cause labial adhesions but supporting evidence is lacking.
3. Adhesions distort the vaginal anatomy and make identification of the hymen difficult.
4. Adhesions may be mistaken for scars.

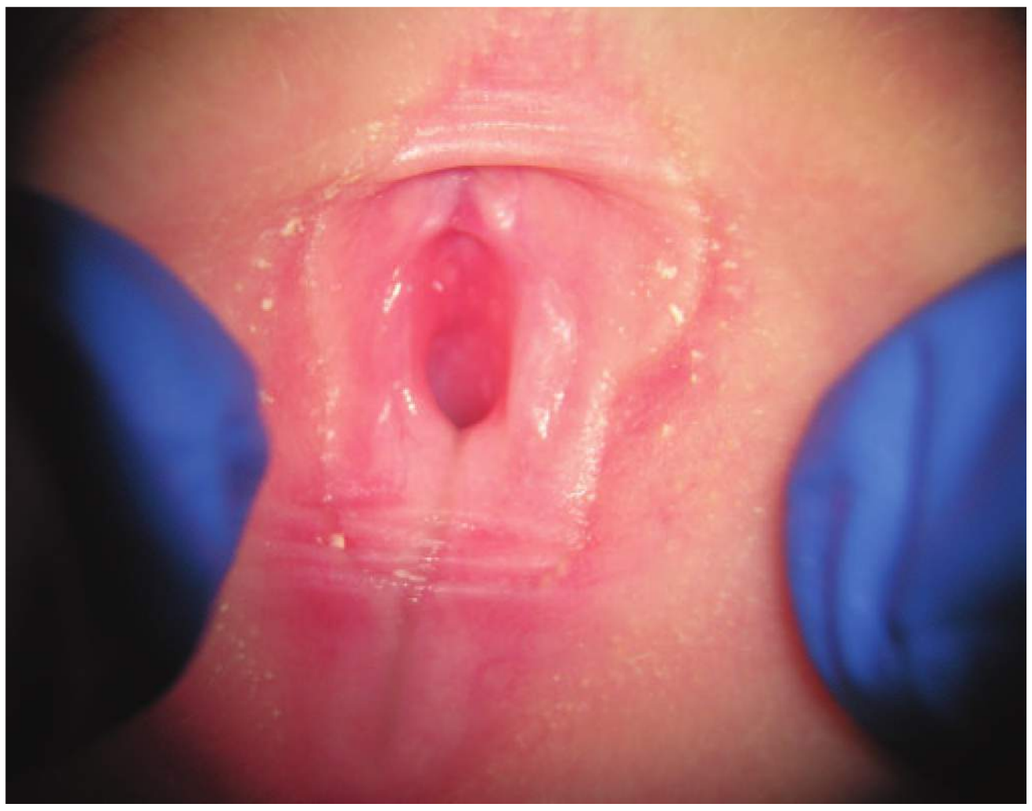


FIGURE 15.93 ■ **Labial Adhesions.** Labial adhesions obscure the hymen in this prepubertal girl. (Photo contributor: Cincinnati Children's Hospital Medical Center.)



FIGURE 15.94 ■ **Labial Adhesions.** Labial adhesions obscure the hymen in this prepubertal girl. (Photo contributor: Larry B. Mellick, MD.)

MIMICS OF ERYTHEMA/BRUISING: PERIANAL STREPTOCOCCAL DISEASE

Clinical Summary

Perianal streptococcal dermatitis/disease is an infectious disease caused by group A β-hemolytic streptococci. Signs and symptoms include perianal as well as vulvovaginal or penile erythematous rash. Itching, pain, anal fissures, blood-streaked stools, rectal bleeding, anal discharge, and vaginal discharge can also be seen. Systematic symptoms (fever, pharyngitis) are absent but can precede the onset of the rash.

Management and Disposition

A bacterial culture for group A streptococci of the perianal and/or vulvovaginal or penile regions should be obtained by rubbing a dry swab over the affected areas. Treatment is with oral penicillin or amoxicillin. Erythromycin is used in patients who are penicillin allergic. Treatment should be 14 to 21 days.

Pearls

1. This can be confused for sexual abuse because of the overlap in clinical presentation: redness, pain, and/or bleeding without a recognized etiology.



FIGURE 15.95 ■ **Perianal Streptococcal Infection.** Intense erythema around the anus consistent with perianal streptococcal infection. (Photo contributor: Raymond C. Baker, MD.)



FIGURE 15.96 ■ **Perianal Streptococcal Infection.** Intense erythema around the anus consistent with perianal streptococcal infection. (Photo contributor: Kevin J. Knoop, MD, MS.)

Clinical Summary

Venous pooling refers to the purple discoloration of the perianal tissues caused by collection of venous blood in the external hemorrhoidal plexus. Venous blood pools in this plexus when a patient is immobile, causing the veins to become distended and visible. It can often be eliminated by getting the patient up and mobilized and then reexamining the patient immediately in the prone or lateral decubitus position.

Management and Disposition

No treatment, other than proper diagnosis and reassurance, is needed.

Pearls

1. The plexus can be visible as a circle of discoloration around the anus or as a linear discoloration.



FIGURE 15.97 ■ **Venous Pooling**. Circumferential venous pooling is noted on this examination. Surrounding venous pooling may be mistaken for bruising. There are no other findings. (Photo contributor: Kathi L. Makoroff, MD.)

Chapter 16

ENVIRONMENTAL CONDITIONS

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Multiple Lightning Strikes. (Photo contributor: Lawrence B. Stack, MD.)

The authors acknowledge the special contributions of Peter Hackett, MD, The Institute for Altitude Medicine Telluride, CO; Edward Otten, MD, University of Cincinnati, Cincinnati, OH; James O'Malley, MD, Providence Alaska Medical Center, Anchorage, Alaska; Murray Hamlet, DVM, and Kathy McCue, MD, Alaska Native Medical Center, Anchorage, Alaska; Sheryl Olson, RN, Manitou Springs, CO; Luanne Freer, MD, Yellowstone National Park, The Nashville Zoo, Nashville, TN; and the Nova Scotia Museum of Natural History, Halifax, Nova Scotia, Canada. The authors thank Joseph C. Schmidt, MD, Lawrence B. Stack, MD, and Alan B. Storrow, MD, for their contributions to prior editions.

HIGH-ALTITUDE RETINAL HEMORRHAGE

Clinical Summary

Retinal hemorrhages are common above 5200 m (17,000 ft) and are not always associated with acute mountain sickness (AMS). High-altitude retinal hemorrhages (HARH) are rarely symptomatic, but if found over the macula, these hemorrhages may cause temporary blindness. The diagnosis can be established by ophthalmoscopy. Without visualization of the lesion, the differential diagnosis of unilaterally decreased vision or blindness at high altitude includes migraine equivalent, cerebrovascular accident, and dry eye (often unilateral, due to strong winds), as well as all conditions found at sea level.

Management and Disposition

HARH generally resolve spontaneously after descent to lower altitudes. No treatment is necessary for asymptomatic HARH. Patients with HARH associated with a decrease in vision should be referred to an ophthalmologist for follow-up.

Pearls

1. Patients with blurred vision and unilateral mydriasis at the high altitude should be asked about use of medications, including transdermal scopolamine patches.
2. As with almost all altitude-related problems, descent is the primary treatment. This is not emergent unless associated with severe altitude illness or progressive visual loss.
3. Although most symptomatic HARH resolve completely in 2 to 8 weeks, cases of permanent paracentral scotomata have been reported.

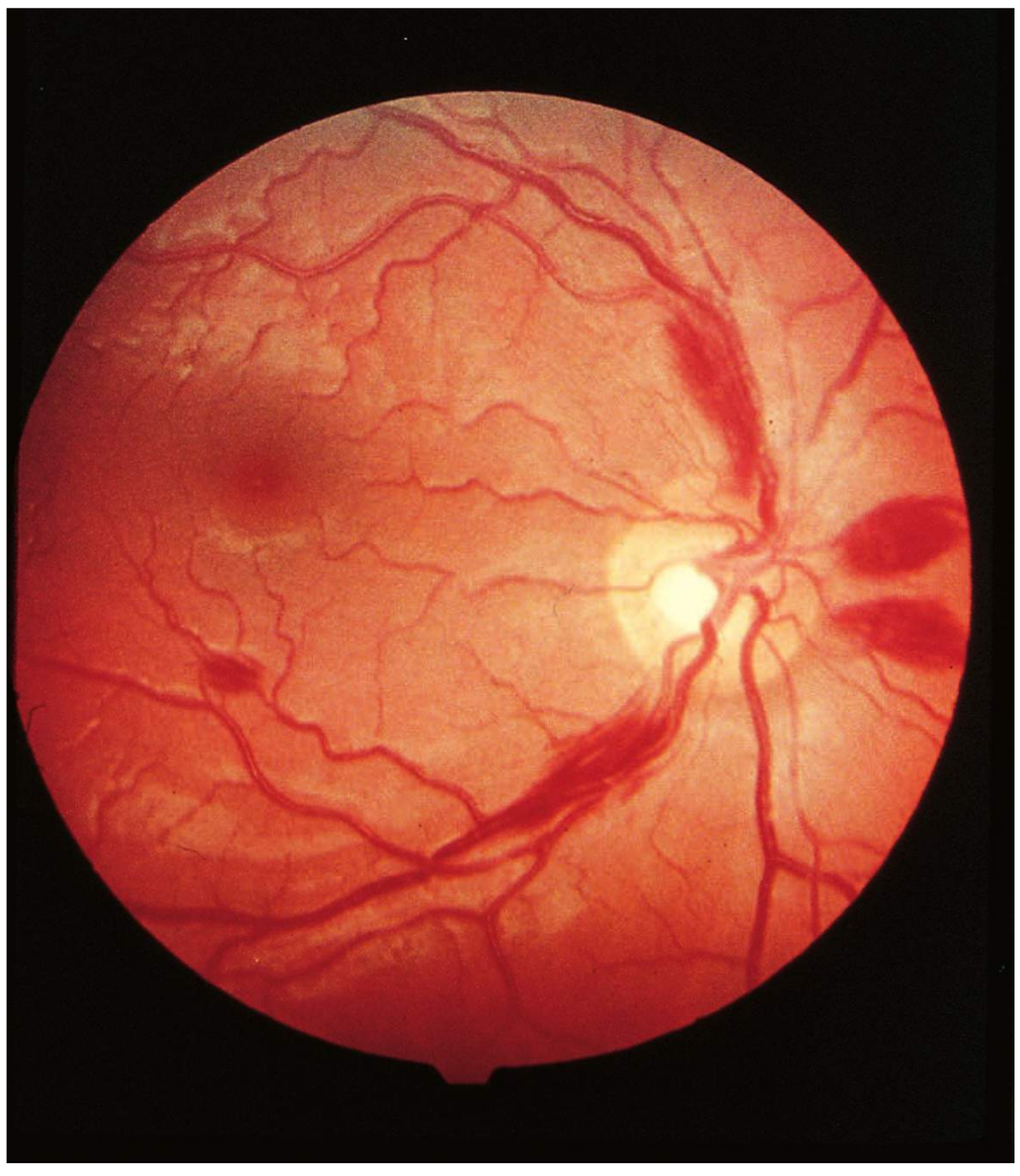


FIGURE 16.1 ■ **High-Altitude Retinal Hemorrhage.** Fundoscopic appearance of high-altitude retinal hemorrhage. (Photo contributor: Peter Hackett, MD.)

Clinical Summary

High-altitude pulmonary edema (HAPE) is a form of noncardiogenic pulmonary edema, generally beginning within the first 2 to 4 days after ascent above 2500 m (8200 ft). The earliest symptoms are fatigue, weakness, dyspnea on exertion, and decreased exercise performance. Symptoms of Acute mountain sickness (AMS) such as headache, anorexia, and lassitude may also be present, but HAPE may develop without AMS. First symptoms usually include persistent dry cough and dyspnea followed by tachycardia, tachypnea, and cyanosis at rest. Patients suffering from HAPE often experience sudden onset of symptoms upon awakening after the second night at altitude. Eventually the victim develops dyspnea at rest and orthopnea with audible crackles in the chest. Pink frothy sputum is a grave sign. Patients may experience concurrent mental status changes and ataxia due to hypoxemia or associated high-altitude cerebral edema (HACE).

Management and Disposition

Mild cases (oxygen saturation in the 90s on low-flow oxygen) at moderate altitudes (below 3500 m, 11,500 ft) may be treated at altitude with bed rest and oxygen. If supplemental oxygen and a reliable person are available, the patient may be discharged with oxygen therapy and bed rest at home or in lodgings. Patients with more severe cases should descend immediately. These patients may require admission to a hospital at a lower altitude and, in extreme cases, intubation and mechanical ventilation. Nifedipine is of benefit but is not a substitute for descent. Some experts now use PDE-5 inhibitors such as sildenafil or tadalafil instead of nifedipine. Hyperbaric therapy, especially with a portable hyperbaric chamber (Gamow bag), has an efficacy equal to that of supplemental oxygen and is mainly helpful in prehospital settings where oxygen availability is limited.

Pearls

1. Crackles may be unilateral or bilateral but usually start in the right middle lobe and are heard first in the right axilla.
2. HAPE limited to the left lung in association with a small right hemothorax without pulmonary markings is pathognomonic for unilateral absent pulmonary artery syndrome. These patients develop HAPE at relatively low altitudes, sometimes below 2500 m.
3. Patients treated for HAPE may resume normal activities once they are asymptomatic and may ascend further during the same trip.

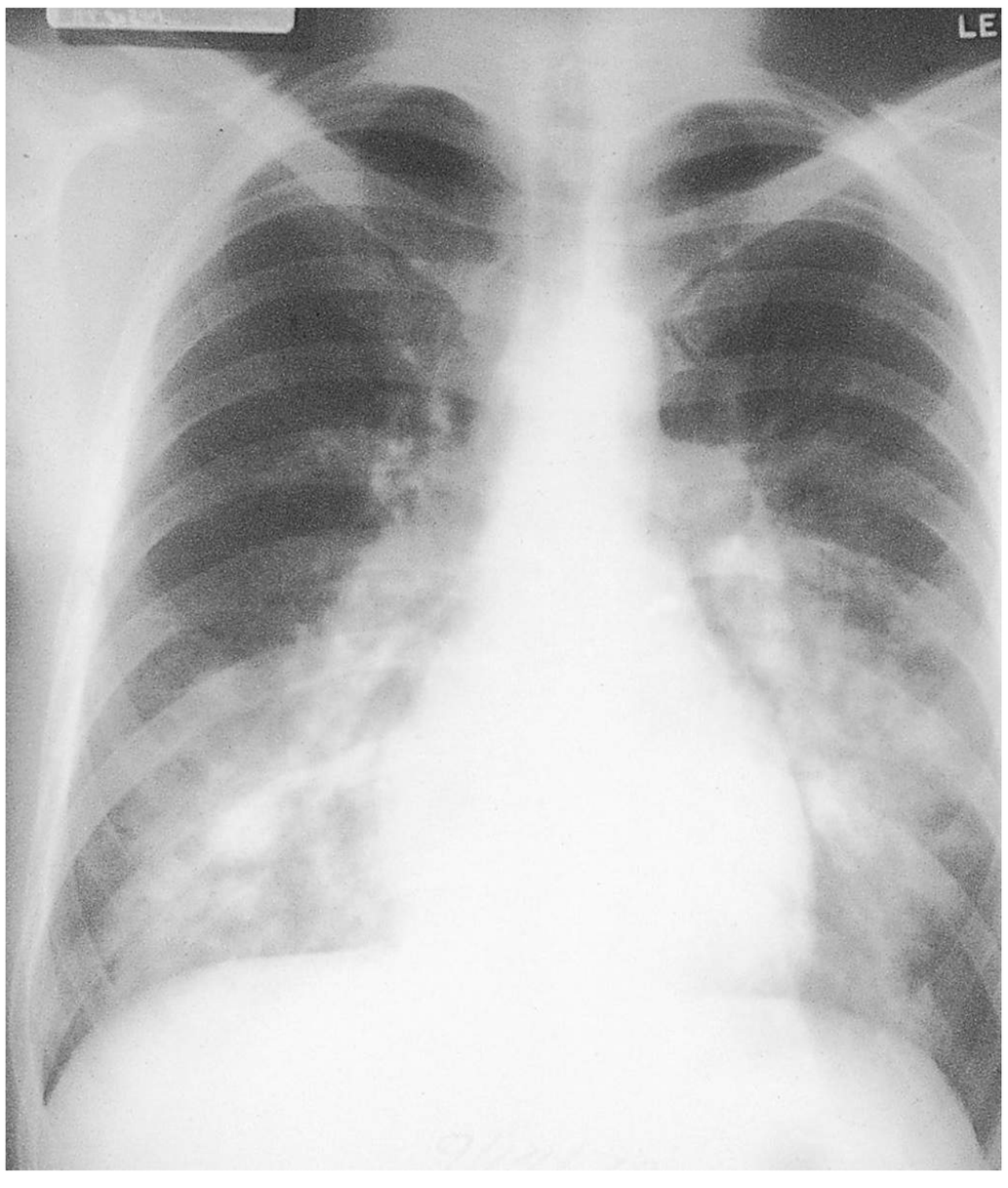


FIGURE 16.2 ■ **High-Altitude Pulmonary Edema.** Chest x-ray in patient with HAPE. Note normal heart size with bilateral "patchy" pulmonary infiltrates. (Photo contributor: Peter Hackett, MD.)



FIGURE 16.3 ■ **"Gamow Bag."** Portable hyperbaric chamber (Gamow bag). A HAPE patient is being treated at 4300 m at Pheriche, Nepal. Due to orthopnea, the patient was unable to tolerate lying flat, so the bag was propped up immediately after inflation. (Photo contributor: Ken Zafren, MD.)

HIGH-ALTITUDE CEREBRAL EDEMA

Clinical Summary

Acute mountain sickness (AMS) is a symptom complex which usually begins 12 to 24 hours after ascent to high altitude and consists of headache and one or more other symptoms, including gastrointestinal symptoms, fatigue and/or weakness, dizziness and/or lightheadedness, and difficulty sleeping. High altitude cerebral edema (HACE) is a severe form of AMS, clinically defined by the presence of acute truncal ataxia, altered mental status, or both. Usually this occurs as a progression from AMS to HACE, but HACE may occur without antecedent AMS. If not effectively treated, HACE may progress to coma or death. Focal neurologic findings other than truncal ataxia are rare and should suggest another diagnosis, such as acute stroke or venous sinus thrombosis.

Management and Disposition

Treatment of HACE consists of immediate descent or evacuation, oxygen, and high-dose dexamethasone. Simulated descent using a portable hyperbaric bag (Gamow bag) may be more effective than oxygen alone and may be used in place of oxygen in field settings. Actual descent may be complicated by the inability of the patient to walk unassisted or at all. Patients with HACE may not ascend during the same trip and probably should not reascend to altitude for several months.

Pearls

1. The first sign of HACE is usually truncal ataxia. This should be tested using tandem gait (heel-to-toe walking).
2. HACE may occur without symptoms of AMS.
3. HACE is often associated with some degree of HAPE.



FIGURE 16.4 ■ **HACE-Related Ataxia.** Ataxia due to HACE in the Nepal Himalayas. The patient (middle) developed HAPE and severe truncal ataxia at 5600 m (18,400 ft). The patient required full ambulatory assistance. Descent was started immediately. With descent, the patient's breathing improved, but he remained too ataxic to walk. (Photo contributor: Ken Zafren, MD.)

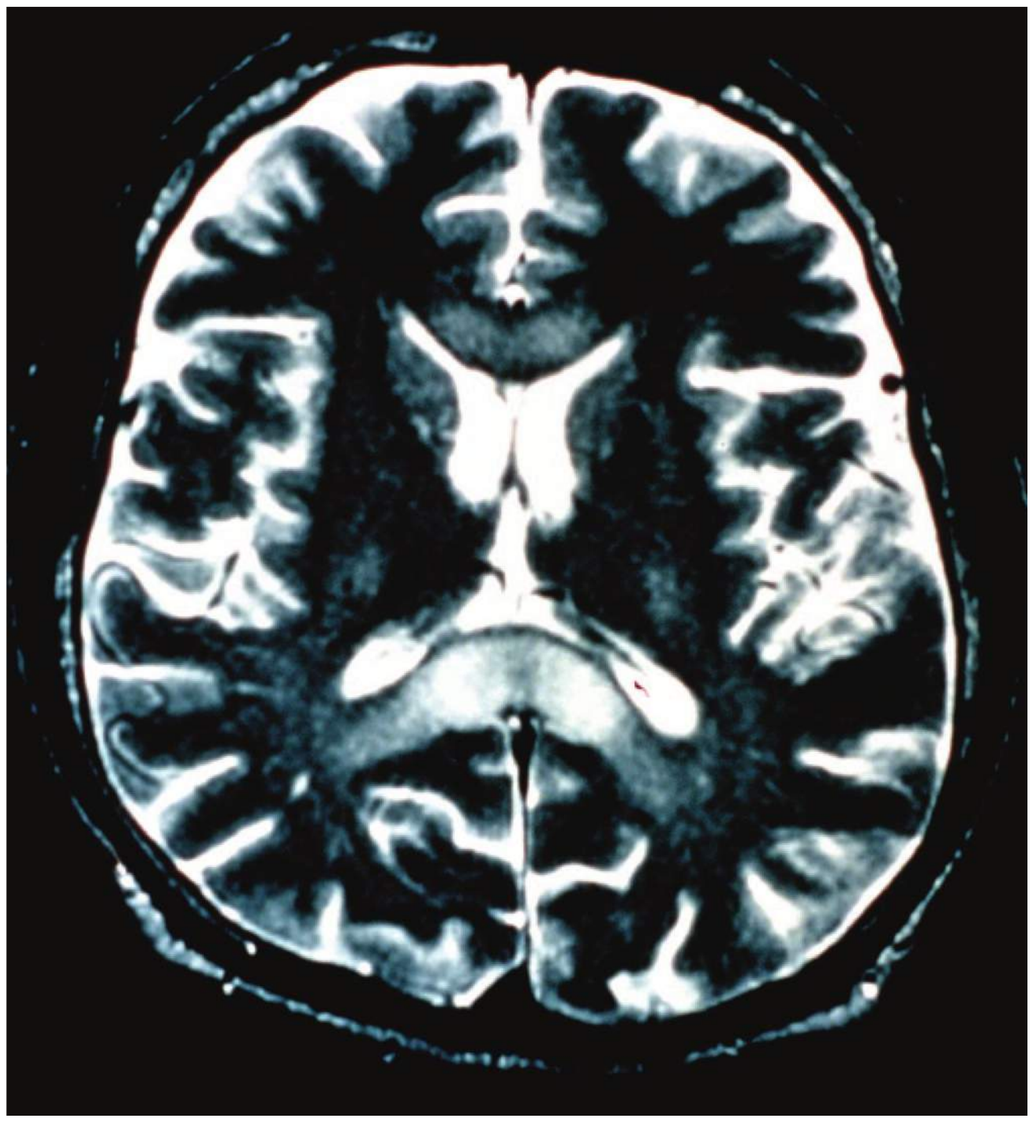


FIGURE 16.5 ■ **MRI Brain—HACE.** MRI of a patient with high-altitude cerebral edema. Note the increased white matter signal, especially in the splenium of the corpus callosum, in this T2-weighted image. (Photo contributor: Ken Zafren, MD.)

Clinical Summary

Accidental hypothermia is an unintentional decline in core temperature below 35°C (95°F). Presentation may be obvious or subtle, especially in urban settings. Symptoms vary from vague complaints to altered levels of consciousness. Physical findings include progressive abnormalities of every organ system. Following initial tachycardia, there is progressive bradycardia (50% decrease in heart rate at 28°C [82.4°F]) with decline in blood pressure and cardiac output. ECG intervals are prolonged, beginning with the PR interval followed by the QRS interval and finally the QT interval. A J wave (Osborn wave; hypothermic "hump") may be seen, but is neither pathognomonic nor prognostic. The J wave is present at the junction of the QRS complex and the ST segment. J waves may also be associated with central nervous system lesions, focal cardiac ischemia, young age, and sepsis. In mildly hypothermic patients, invisible preshivering muscle tone may obscure P waves.

Management and Disposition

Core temperature measurement is best made with an esophageal probe inserted into the lower third of the esophagus. Rectal temperature is less accurate without the use of a special low-reading thermometer. Gentle handling and appropriate warming methods are the mainstays of emergency department (ED) treatment. Cardiovascular instability often complicates rewarming; Advanced Cardiac Life Support (ACLS) guidelines for hypothermia provide guidance. If not obvious, a precipitating cause should be sought (eg, hypothyroidism, hypoglycemia, sepsis), as should associated pathology. Most patients require admission for observation or to treat associated injuries or comorbidities.

Pearls

1. The most common problem with the misdiagnosis of hypothermia in the ED stems from incomplete data on vital signs.
2. Accurate core temperatures, preferably by esophageal probe, and continuous cardiac monitoring are crucial to appropriate management.
3. Atrial arrhythmias are generally benign and should not be treated. They resolve with rewarming.
4. Most cardiovascular drugs are inactive during hypothermia and should be given only once the body temperature is above 35°C (95°F).

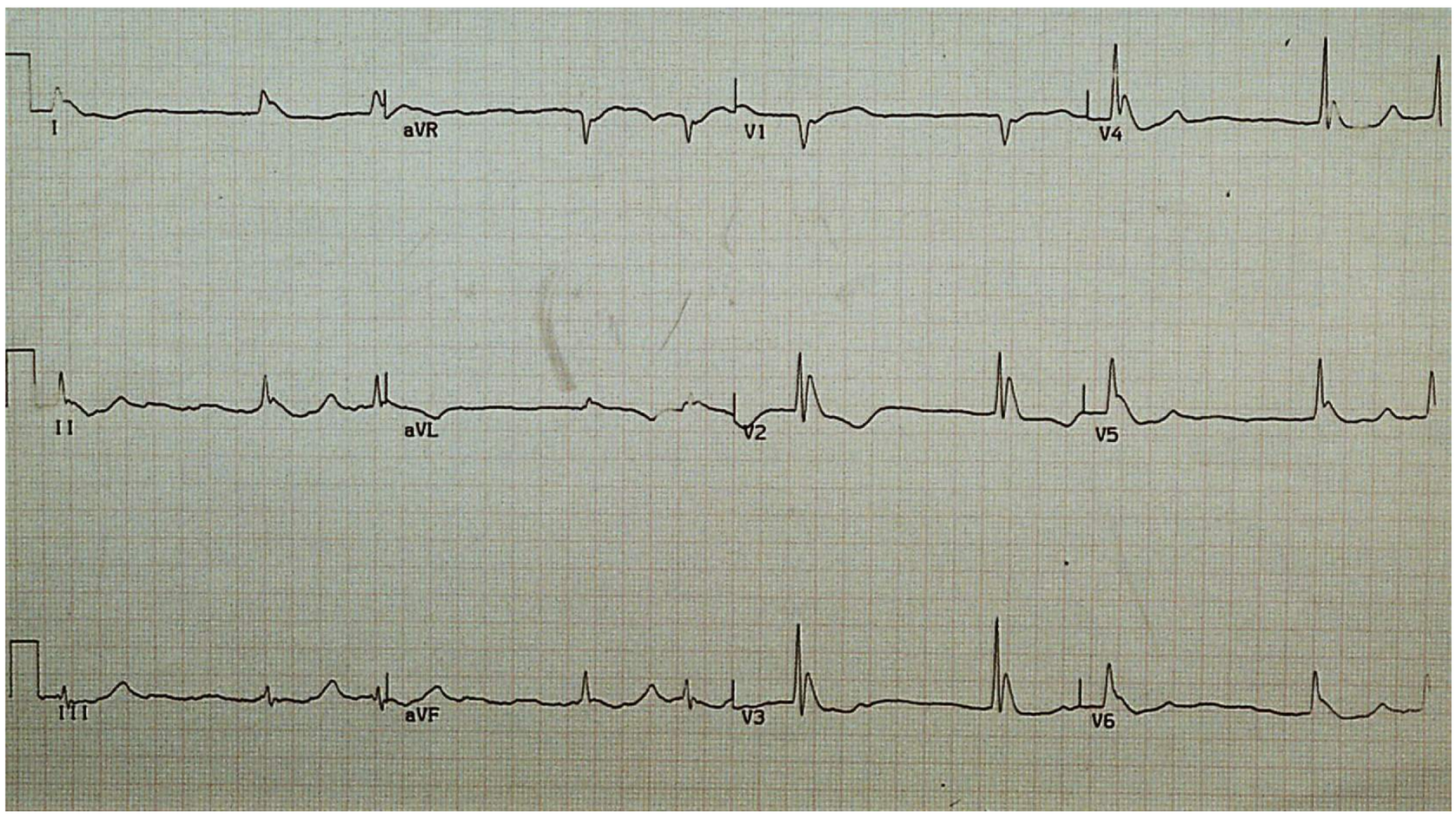


FIGURE 16.6 ■ **J Waves.** J waves in a hypothermic patient with core temperature (rectal probe) of 25.5°C. J waves may be seen at any temperature below 32.2°C, most frequently in leads II and V_6. Below a core temperature of 25°C, they are most commonly found in the precordial leads (especially V_3 and V_4) and their size increases. J waves are usually upright in aVL, aVF, and the left precordial leads (see also Fig. 23.44A "Hypothermia with Osborne Waves ("J" Waves) Present"). (Photo contributor: Alan B. Storrow, MD.)

Clinical Summary

Frostbite is tissue freezing resulting from heat loss sufficient to cause ice crystal formation in superficial or deep tissue. Frostbite usually affects the extremities, nose, or ears (and the scrotum and penis in joggers). A sensation of numbness with accompanying sensory loss is the most common initial complaint. Often, by the time the patient arrives in the ED, the frozen tissue has thawed. The initial appearance of the overlying skin may be deceptively benign. Frozen tissue may appear mottled blue, violaceous, yellowish-white, or waxy. Following rapid rewarming, there is early hyperemia even in severe cases.

Favorable signs include return of normal sensation, color, and warmth. Edema should appear within 3 hours of thawing; lack of edema is an unfavorable sign. Vesicles and bullae appear in 6 to 24 hours. Early formation of large clear blebs that extend to the tips of affected digits is a good indicator. Small dark blebs that do not extend to the tips indicate damage to subdermal plexi and are a poor prognostic sign. When seen early or after rewarming occurs, frostbite may be indistinguishable from nonfreezing cold injury such as immersion foot. Mixed injuries are common.

Management and Disposition

If other injuries are ruled out by history and physical examination, rewarm frostbitten areas in warm water bath (37°C-39°C [98.6°F-102.2°F]). If associated with severe hypothermia, active core rewarming should precede frostbite rewarming. If swelling occurs, surgical consultation is advisable along with compartment pressure measurement to determine the need for fasciotomy. Admit all patients with associated hypothermia or in whom swelling occurs. Superficial frostbite (minimal skin changes and erythema) may be treated by home care with nursing instructions. Deep superficial frostbite (clear, fluid-filled blebs, swelling, pain) may be treated by home care in a reliable patient. Deep frostbite (proximal hemorrhagic blebs, no swelling, no pulses) mandates hospital admission.



FIGURE 16.7 ■ **Thawed Frostbite.** Typical appearance of frostbite soon after rewarming. Deep frostbite was caused by wearing mountaineering boots that were too tight in extreme cold at high altitude. Note the deceptively benign appearance of this devastating injury, which ultimately resulted in bilateral below-the-knee amputations. (Photo contributor: Ken Zafren, MD.)



FIGURE 16.8 ■ **Deep Frostbite.** Deep frostbite at Everest Base Camp in Nepal, located at an altitude of 5360 m (17,585 ft), 3 days after exposure at 6400 m (21,000 ft). Dusky appearance and lack of distal blistering on the great toe are poor prognostic signs. The great toe eventually required partial amputation. (Photo contributor: Chris Imray, MD.)

Pearls

1. Early transfer of the patient to a center experienced in the care of frostbite injuries (even if far away) should be considered. On the other hand, transfer of the patient to a major medical center that does not generally manage frostbite is seldom in the patient's best interest.
2. Treatment of clear versus hemorrhagic blisters is controversial; one approach is to debride clear blisters and use topical aloe vera while leaving hemorrhagic blisters intact.



FIGURE 16.9 ■ **Frostbite Blebs.** Intact proximal blebs, both clear and hemorrhagic, indicate deep frostbite and a poor prognosis. (Photo contributor: Scott W. Zackowski, MD.)



FIGURE 16.11 ■ **Late Frostbite.** Late appearance of frostbite with demarcation starting to occur. Early surgery should be avoided in favor of autoamputation unless infection supervenes. (Photo contributor: James O'Malley, MD.)



FIGURE 16.10 ■ **Frostbite-Ruptured Blebs.** Frostbite injury to the hand with ruptured blebs. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 16.12 ■ **Late Frostbite.** Late appearance of deep frostbite with clear demarcation. (Photo contributor: Wael Alrifai, MD.)

Clinical Summary

Pernio, also known as perniosis or chilblains, is the result of nonfreezing cold exposure in susceptible individuals. Pernio appears within 24 hours of cold exposure, most frequently on the face, ears, hands, feet, and pretibial areas. A large range of lesions may be seen, with localized edema, erythema, cyanosis, plaques, and blue nodules occasionally progressing to more severe lesions including vesicles, bullae, and ulcerations. The lesions persist for up to 2 weeks and may become chronic. They are typically very pruritic and associated with burning paresthesias. Following rewarming, pernio often takes the form of blue nodules, which are quite tender. In the setting of recent cold exposure, pernio might be confused with the more severe syndrome of trench foot. If the history of cold exposure is not elicited, the differential diagnosis is potentially very broad.

Management and Disposition

Management is supportive. The skin should be warmed, washed, and dried. Affected extremities can be dressed in soft, dry, sterile dressings and elevated. Nifedipine (20-60 mg daily) may be helpful in chronic cases.

Pearls

1. Healing may be followed by hyperpigmentation.
2. Recurrences are possible following milder cold exposure.
3. Chilblains may be more frequent in young women, especially in association with Raynaud phenomenon, and may also be associated with underlying dermatologic or vascular disease.



FIGURE 16.14 ■ **Pernio.** Pernio of the hand with localized erythema and painful nodules. (Photo contributor: Alison Jarman, MD.)



FIGURE 16.13 ■ **Pernio.** Pernio or chilblains with localized erythema, cyanosis, and nodules. (Photo contributor: Ken Zafren, MD.)

IMMERSION INJURY (TRENCH FOOT)

Clinical Summary

Immersion injury is a peripheral nonfreezing cold injury resulting from exposure to water, usually at temperatures just above freezing. However, the condition can occur during prolonged exposure to any cool wet environment. Dependency and immobility predispose to immersion injury. The degree of injury depends on exposure time and temperature. The first symptoms usually appear within hours; tissue loss may occur after many days of exposure. Prior to rewarming, the distal extremities are numb and swollen. The skin is first red, then changes to pale, mottled, or black. Cramping of the calves may occur. Immersion injury is distinct from tropical immersion foot or warm-water immersion foot as seen in the Vietnam War. Tropical immersion foot was typically seen after 3 to 7 days of exposure to water at 22°C to 32°C. Warm-water immersion foot was seen after 1 to 3 days at 15°C to 32°C. These syndromes were characterized by burning in the feet, pain on walking, pitting edema, and erythema, with wrinkling and hyperhydration of the skin. They resolved completely after rest and removal from the wet environment.

Management and Disposition

Hypovolemia, hypothermia, and associated injuries are the rule and should be treated first. General treatment of immersion foot (or hand) is similar to that for rewarmed frostbite. Swelling may produce compartment syndrome and require fasciotomy. Most patients require admission to the hospital.



FIGURE 16.15 ■ **Immersion Injury.** Immersion injury to hands (unusual location) several hours after rewarming. The patient spent 18 hours bailing out a boat in waters just above freezing in Alaska. (Photo contributor: Ken Zafren, MD.)

Pearls

1. Doppler ultrasound may be useful to identify peripheral pulses, as pulses are often difficult to palpate in affected extremities.
2. Mixed injuries (frostbite and immersion) are common.



FIGURE 16.16 ■ **Immersion Foot.** Early appearance of immersion foot in a mentally ill homeless patient. (Photo contributor: Ken Zafren, MD.)



FIGURE 16.17 ■ **Severe Trench Foot.** This unfortunate homeless patient suffered prolonged exposure to a cold wet environment with resulting severe trench foot. (Photo contributor: Alan B. Storrow, MD.)

ULTRAVIOLET RADIATION EXPOSURE

Clinical Summary

Ultraviolet (UV) radiation causes both acute and chronic skin changes. Sunburn is a partial-thickness burn, which may become a full-thickness injury if infected. "Sun poisoning" is a severe systemic reaction to UV radiation. Patients may complain of nausea, vomiting, headache, fever, chills, and prostration. Excessive UV radiation may cause injury to the cornea and conjunctiva, termed UV keratitis (photokeratitis, snow blindness). This painful condition may occur in skiers, welders, or tanning salon patrons who do not wear proper eye protection.

Photosensitivity reactions (photodermatoses) are of several types. Phototoxic reactions are an abnormal response to UV radiation caused by a substance that is ingested (eg, prescription or over-the-counter medications) or applied to the skin; there is a direct relation between the amount of UV exposure and severity. Photoallergic reactions are clinically similar to contact dermatitis and, like phototoxic reactions, may be precipitated by ingested or applied drugs. Unlike phototoxic reactions, photoallergies may be precipitated by a small amount of light. Phytophotodermatitis is precipitated by skin contact with certain plants followed by exposure to UV radiation.

Phototoxicity should be suspected in any patient with severe or exaggerated sunburn. Photoallergy is easily misdiagnosed as allergic eczema or contact dermatitis, especially since onset is often delayed up to 2 days after exposure. Phytophotodermatitis may mimic severe sunburn or contact dermatitis, especially rhus dermatitis. Endogenous photosensitizers (endogenous photodermatoses) include solar urticaria, porphyria cutanea tarda, polymorphous light eruption, and systemic lupus erythrematosus. These may be provoked by visible light as well as by UV radiation.

Management and Disposition

Treatment of sunburn and sun poisoning involves standard burn therapy and supportive care. Sunburn is usually a self-limited problem. Cool compresses and nonsteroidal anti-inflammatory drugs may be beneficial. UV keratitis is treated with mydriatic-cycloplegic eye drops to decrease pain; initial examination is made easier with topical anesthetics. Severe cases may require antibiotic ointment, narcotic analgesics, and bilateral eye patches for 12 to 24 hours, though the use of eye patches remains controversial. These patients require 24- to 48-hour follow-up; ophthalmology referral is indicated to rule out retinal damage.

Treatment of photosensitivity reaction has two components: treatment of the sunburn and recognition of the sensitizing agent or endogenous medical condition. Topical steroids and oral analgesics and antipruritics may be helpful. Systemic steroids may be required. Patients with severe reactions should be referred to a dermatologist for possible photo patch testing.

Pearls

1. Para-aminobenzoic acid (PABA) in sunscreens may be a photosensitizer and can cause a photoallergic reaction.
2. The unique properties of individual skin types produce marked differences in response to UV radiation.
3. Victims of UV keratitis typically present 2 to 12 hours after exposure. As such, patients may present during the night with severe unexplained bilateral eye pain.



FIGURE 16.18 ■ **Sunburn.** Sunburn is characterized by erythema, edema, warmth, tenderness, and blisters. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 16.19 ■ **Healing UV Exposure.** This patient suffered significant UV exposure with resultant sunburn in the Himalayas. Darkly pigmented skin provides limited protection in high-altitude subtropical areas with very high amounts of UV exposure. (Photo contributor: Luanne Freer, MD.)



FIGURE 16.21 ■ **Phytophotodermatitis.** This reaction may require aggressive systemic steroid therapy. The case illustrated is a mild one caused by exposure to limes and UVA. A clue to the diagnosis is the patchy distribution with linear edges. More severe reactions resemble rhus dermatitis. (Photo contributor: Lee Kaplan, MD.)

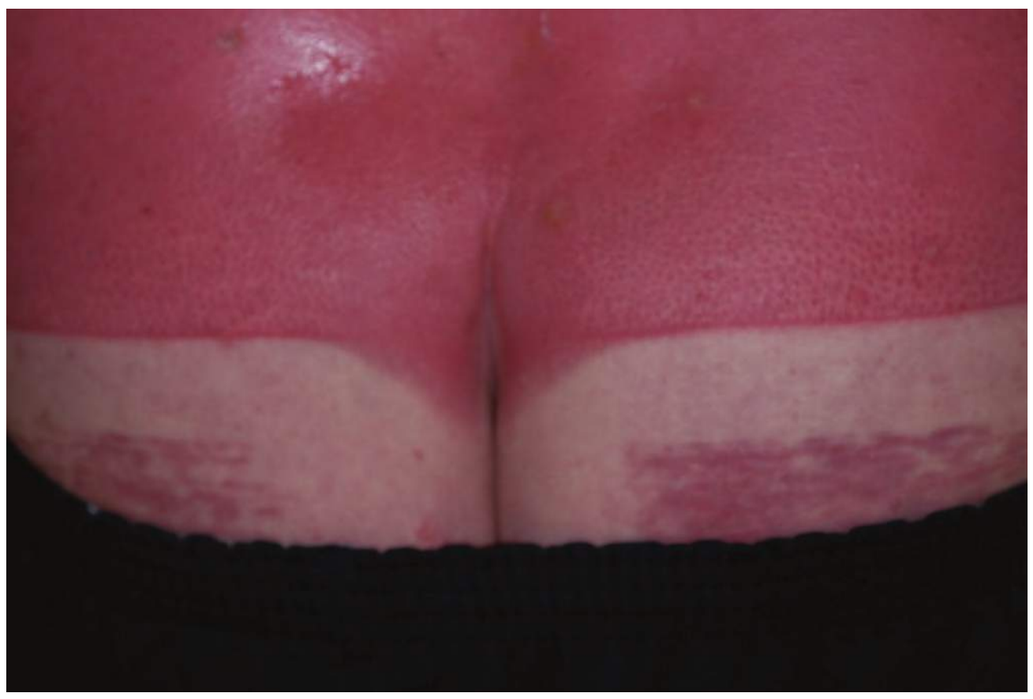


FIGURE 16.20 ■ **Tanning Bed Burn.** This patient suffered a severe diffuse partial thickness burn from prolonged UV exposure in a tanning bed. Note the sharp demarcation on the buttocks at the point where the patient was partially protected by his pants. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 16.22 ■ **Phytophotodermatitis—Cow Parsnip.** Severe phytophotodermatitis caused by exposure to cow parsnip (Heracleum lanatum) in Alaska. Another name for cow parsnip is "pushkie," so this is also known locally as "pushkie burn." (Photo contributor: Kathy McCue, MD.)



FIGURE 16.23 ■ **Phytophotodermatitis—Lime.** Phytophotodermatitis caused by exposure to limes and UV radiation in Jamaica. The patient had been exposed while riding shirtless on a horse through a lime orchard. (Photo contributor: Stephan Russ, MD.)

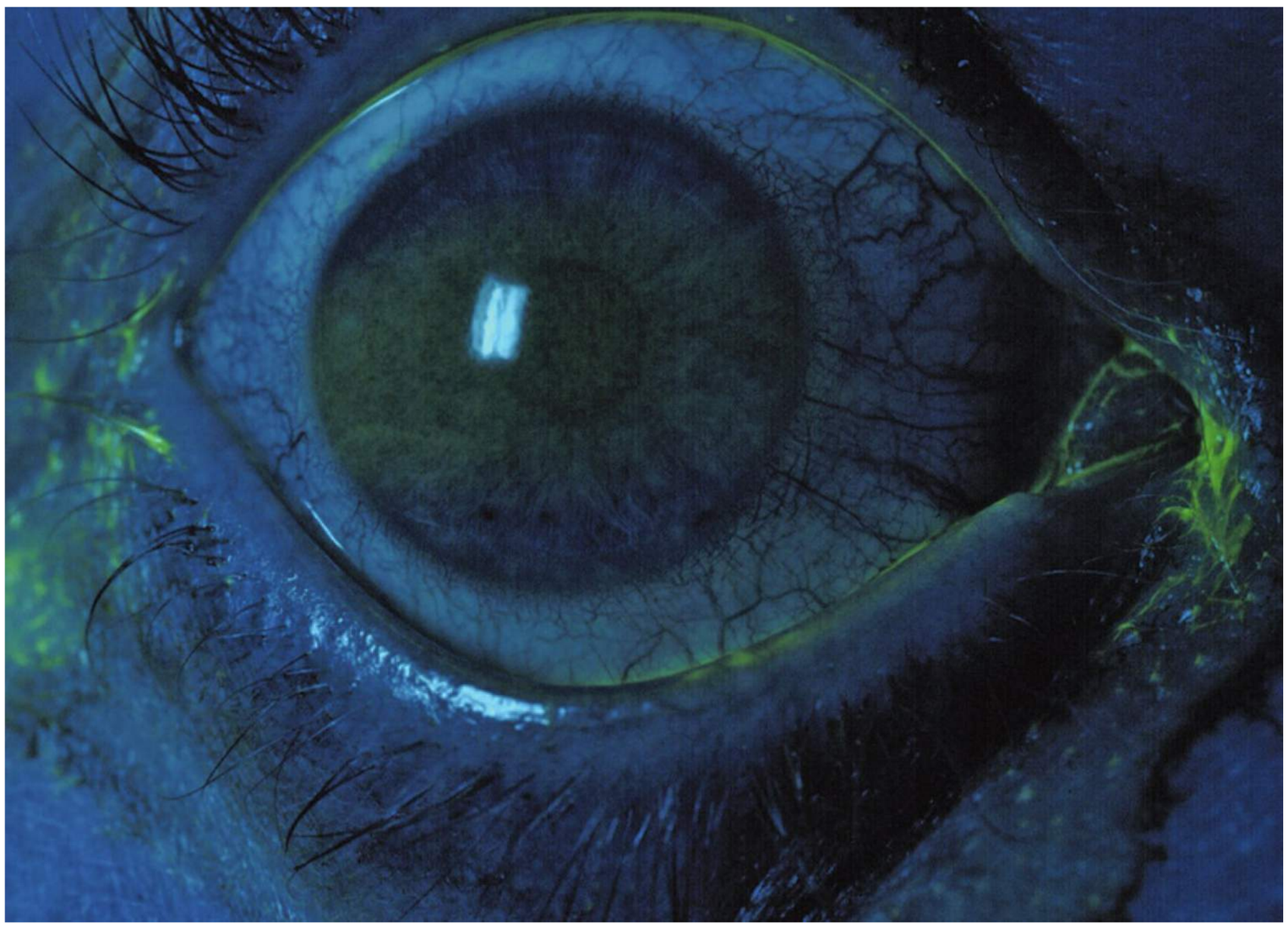


FIGURE 16.24 ■ **Ultraviolet Keratitis.** This patient presented with severe eye pain at 3 AM after a day of arc welding without proper eye protection. Notice the absence of fluorescein uptake where the eyelids were protective. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Lightning produces injury from high voltage, heat production, and explosive shock waves. Direct injuries include cardiorespiratory arrest, cardiac arrhythmias, and neurologic abnormalities such as seizures, deafness, confusion, amnesia, blindness, and paralysis. The patient may suffer contusions from the shock wave or from opisthotonic muscle contractions. Chest pain and muscle aches are common. One or both tympanic membranes (TMs) rupture in more than 50% of victims. Cataracts are usually a delayed occurrence. Hematologic abnormalities including disseminated intravascular coagulation (DIC) have been described. Fetal demise may occur.

Burns may result from vaporization of sweat or moist clothing, heating of clothing and metal objects such as climbing equipment, belt buckles or bra wiring, and direct effects of the strike. Linear burns and punctate burns are thermal burns. Feathering burns, also known as ferning or Lichtenberg figures, are not actually burns but may represent superficial bruising. They are pathognomonic of lightning injury.

Diagnosis of lightning injury is straightforward when there is a thunderstorm, when there are witnesses to the strike, or when there are typical physical findings. Lightning on relatively sunny days (without loud thunder) striking a lone victim may produce a confusing picture. The scattering of clothing and belongings may mimic an assault. Side flashes from metal objects and wiring may produce indoor victims during storms.



FIGURE 16.25 ■ **Linear Lightning Burns.** Linear burns from lightning are due to thermal effects. (Photo contributor: William Barsan, MD.)



FIGURE 16.26 ■ **Linear Lightning Burns.** Linear lightning burns along the leg and foot of a strike victim. (Photo contributor: Sheryl Olson, RN, BSN, CCRN.)



FIGURE 16.27 ■ **Punctate Lightning Burns.** Punctate burns due to lightning are partial or full-thickness thermal burns that range from a few millimeters to a centimeter in diameter. They are multiple and closely spaced. (Photo contributor: Arthur Kahn, MD.)

Management and Disposition

History and physical examination to rule out associated injuries and standard ED care and workup for critical patients—including cardiac enzymes, urinalysis, and ECG—are necessary. All patients, even those apparently well, require admission for observation since their condition may change over several hours following the lightning strike.

Pearls

1. The amount of damage to the exterior of the body does not predict the amount of internal injury.
2. Since lightning most commonly produces cardiac standstill by means of massive direct current countershock, prompt, spontaneous return of normal heart rhythm (by virtue of cardiac automaticity) is the rule. However, respiratory arrest is often more prolonged. In a triage situation, the normal rules do not apply, since victims breathing spontaneously are already recovering. The rule in lightning strikes is to resuscitate the "dead." Ventilatory support is often all that is required.
3. Keraunoparalysis (lightning paralysis) is a condition that is specific to lightning injury and is caused by extreme vasoconstriction, usually in the legs. It resolves spontaneously within a few hours.
4. Psychological sequelae following lightning injuries are underreported and may include memory loss, difficulty with concentration, and depression.

FIGURE 16.29 ■ **Feathering from Underwire Bra.** Feathering (ferning) burns are pathognomonic of lightning injury. These are not true burns but may represent superficial bruising. (Photo contributor: Sheryl Olson, RN, BSN, CCRN.)

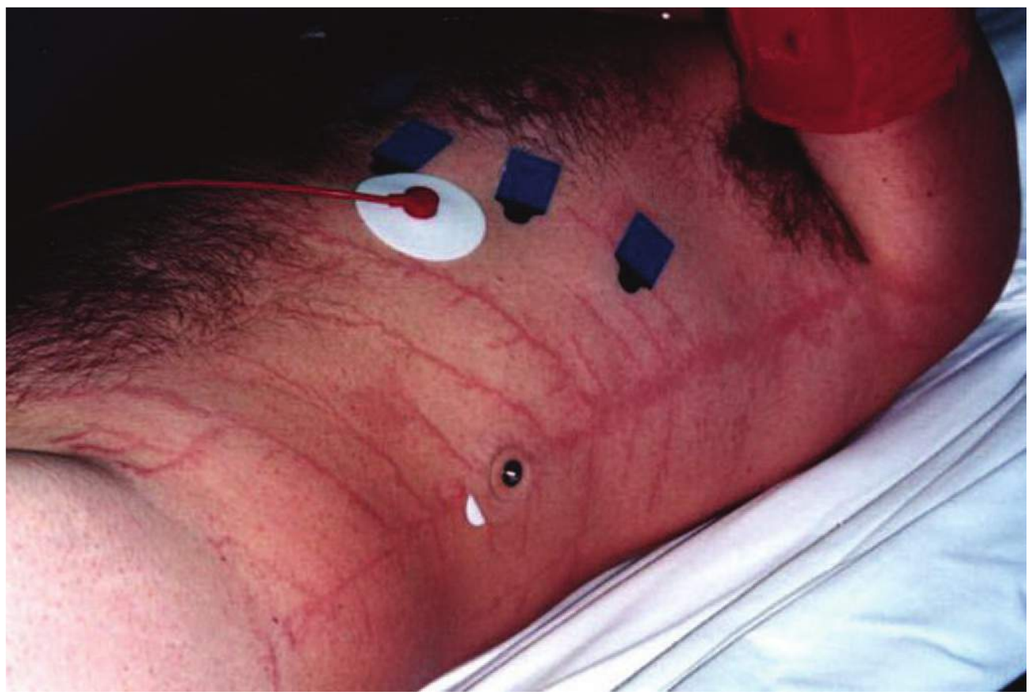


FIGURE 16.28 ■ **Feathering.** Feathering (ferning) on the left side of a lightning strike victim. (Photo contributor: Sheryl Olson, RN, BSN, CCRN.)

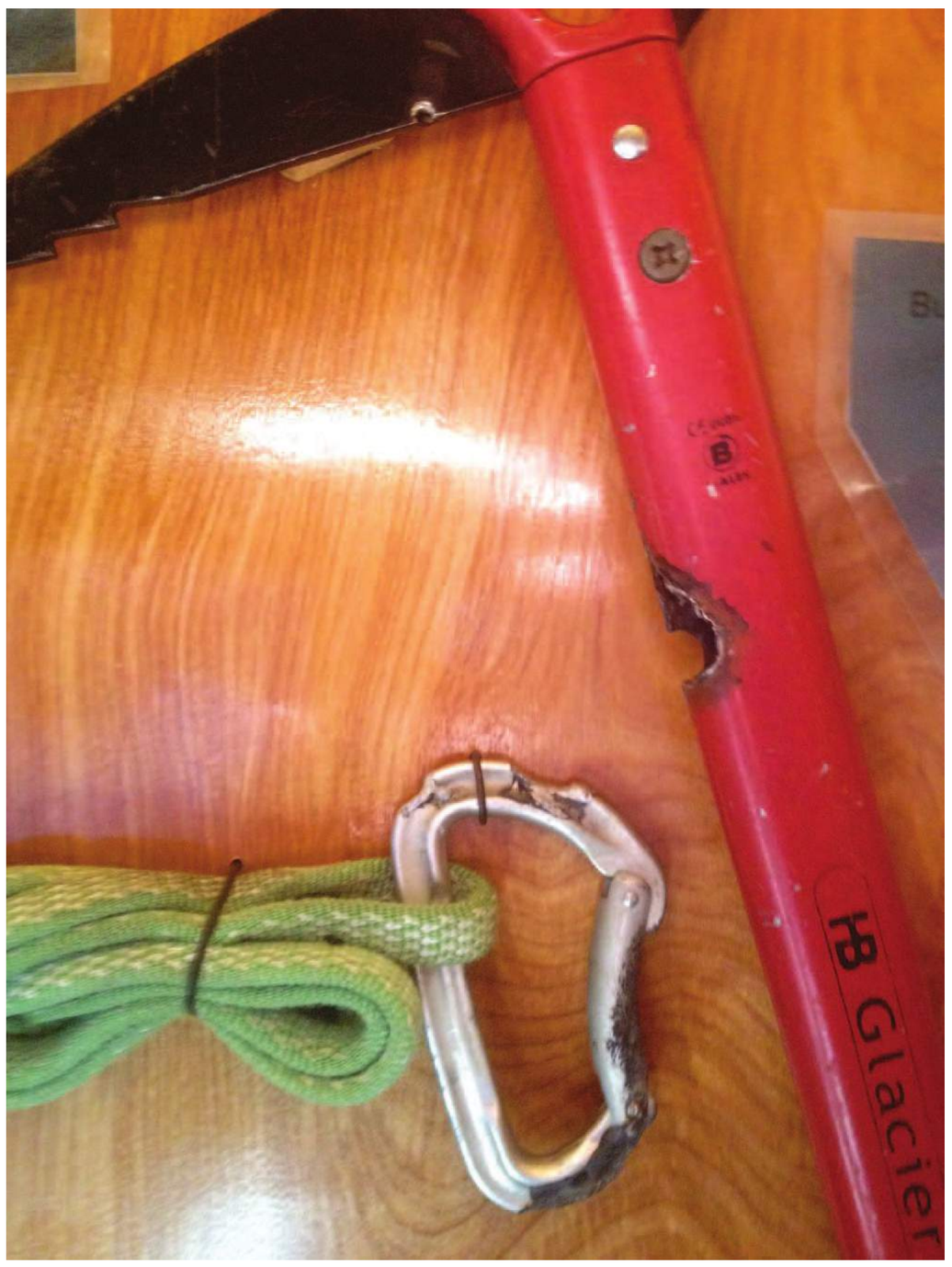


FIGURE 16.30 ■ **Lightning Damage.** Lightning can instantly heat metal objects, causing significant burns to skin in contact with the equipment. (Photo contributor: Nicholas Kanaan, MD.)

Clinical Summary

Both domestic and wild animals are known to attack humans. Injuries are caused by combinations of penetrating and blunt trauma. Penetrating injuries can be inflicted by teeth, claws, and horns, while severe blunt injuries may result from the victim being knocked over, trampled or otherwise crushed, being thrown into the air, or dragged. Injuries may involve massive tissue injury or avulsion often associated with neurovascular damage and long bone fractures.

Management and Disposition

Evaluation and treatment is the same as for any other multiple trauma victim with a high-energy mechanism. After attention to the ABCs, which may be complicated by injuries of the face, neck, or chest, victims should be evaluated for less obvious blunt traumatic injuries. Open fractures and injuries to internal organs may not be clinically apparent initially as with any multitrauma victim.

Pearls

1. Wounds often require operative debridement and repair by appropriate specialists. Consider tetanus and rabies prophylaxis along with antibiotic treatment for high-risk wounds.
2. Bite wounds can penetrate the skull or joint. Displaced teeth or claws may remain imbedded in wounds. Evaluation should include x-rays, ultrasound, and/or CT scanning along with appropriate surgical consultations.

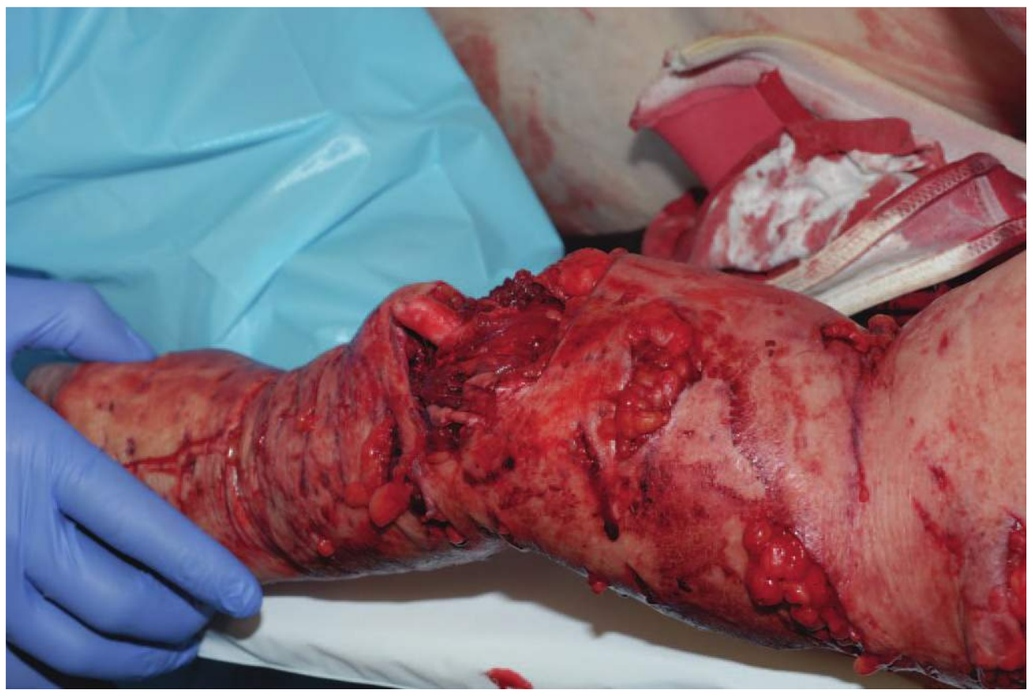


FIGURE 16.32 ■ **Pit Bull Mauling.** The left arm of a pit bull mauling victim. Note the open fracture of the radius. The patient sustained multiple severe defensive wounds to both arms resulting in extensive soft tissue and neurovascular injuries. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 16.31 ■ **Cougar Mauling.** This patient sustained large wounds as a result of a cougar attack. Here, the weight and force of this large animal has resulted in severe shearing injuries. Penetrating injuries from large animals may be deeper than they appear. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 16.33 ■ **Bear Attack—Open Scapula Fracture.** Although grizzly claws are not very sharp, they can exert considerable force. This innocuous appearing injury was complicated by an open scapula fracture. (Photo contributor: Luanne Freer, MD.)

CORAL SNAKE ENVENOMATION

Clinical Summary

The most important species of coral snakes (Elapidae family) found in the United States are the eastern coral snake (Micrurus fulvius) and the Texas coral snake (Micrurus tener). Coral snakes have small mouths, and bites are usually limited to fingers, toes, or folds of skin. Due to a less efficient venom apparatus than their Crotalid colleagues, coral snakes generally need to hold on or chew to inflict a significant envenomation. The bite typically produces minimal local inflammation and pain. Paresthesias and muscle fasciculations are common. Systemic symptoms resulting from the powerful neurotoxic effects of the venom can include tremors, drowsiness, euphoria, marked salivation, and respiratory distress. Cranial nerve involvement, manifested by slurred speech and diplopia, may be followed by bulbar paralysis with dysphagia and dyspnea. Death may result from respiratory and cardiac arrest. Onset of severe symptoms may be delayed up to 12 hours, but may also be rapidly progressive.

Management and Disposition

In contrast to pit viper envenomation, prehospital application of a constrictive bandage may be of benefit in limiting the spread of neurotoxic coral snake venom. Severe systemic symptoms following envenomation by Elapidae may be delayed and cannot be accurately predicted by local wound reactions. It is therefore recommended that four to six vials of antivenom be administered for all suspected envenomations by eastern or Texas coral snakes. Treatment of western coral snake (Micruroides euryxanthus) bites is purely supportive. Tetanus prophylaxis should be addressed.

Pearls

1. Treatment with antivenom should be initiated early in cases of eastern coral snake bites, since symptoms are often delayed and severe.
2. The adage "red on yellow, kill a fellow; red on black, venom lack" applies to all coral snakes found in the United States but does not hold true in other parts of the world.
3. As many as 60% of North American coral snake bites do not result in envenomation of the victim.



FIGURE 16.35 ■ **Coral Snake (Close-Up).** A close-up view of the striking and distinctive markings of the highly venomous coral snake. (Photo contributor: Mike Cardwell, MS.)



FIGURE 16.34 ■ **Coral Snake.** United States eastern coral snake with typical coloring and red-on-yellow bands. (Photo contributor: Mike Cardwell, MS.)



FIGURE 16.36 ■ **Nonvenomous Milk Snake.** As opposed to the red-on-yellow rings seen in the venomous US coral snake, these red-on-black rings indicate a nonvenomous snake. Unfortunately, this applies only to animals native to the United States. (Photo contributor: Sean P. Bush, MD.)

Clinical Summary

The pit vipers (Crotalidae family) indigenous to the United States include rattlesnake species, cottonmouths, and copperheads. Physical characteristics of pit vipers include a triangular head, heat-sensing pits, elliptical pupils, and a single row of subcaudal ventral scales. Pit viper venom is complex and produces hematologic, cardiovascular, and neuromuscular effects. Clinically, pit viper envenomations are divided into four categories. Snakebites without envenomation are characterized only by the direct tissue damage caused by the strike. Minimal envenomations consist of fang marks and local swelling but no systemic symptoms. Moderate envenomations include progression of tissue changes beyond the immediate location of the bite and/or systemic symptoms and mild changes in coagulation parameters. Severe envenomations include marked local and progressive swelling and significant systemic symptoms and coagulopathy (eg, subcutaneous ecchymosis and/or signs of bleeding).

Management and Disposition

Prehospital management of pit viper bites should include immobilization and rapid transport without delay. Lymphatic constriction bands and extractor devices should not be used. These devices may worsen focal complications of envenomation. Tourniquets and local incision are ineffective and generally do more harm than good. Electric shock, cryotherapy, and mouth suction are not recommended. ED management includes resuscitation, establishing a physiologic baseline, and determining the need for antivenom. CroFab (Savage Labs), a recombinant antivenom effective in neutralizing venom toxins, is the treatment of choice. Indications for antivenom administration include any progression of swelling, erythema, or ecchymosis beyond the immediate bite area, presence of any systemic signs of envenomation, or any coagulation abnormalities and/or bleeding complications associated with envenomation. The dose of antivenom increases with the severity of envenomation. The package insert details the current recommendations for antivenom administration and allergy testing, but in general treatment is initiated with 4 to 6 vials of CroFab intravenously with further dosing regimen based on clinical course. Compartment syndrome is a possible complication of envenomation, and should initially be treated with antivenom. Fasciotomy should be performed only if compartment pressures are greater than 30 mm Hg in spite of antivenom treatment. Patients who do not develop evidence of envenomation after 8 hours of observation may be safely discharged home with close follow-up. Tetanus prophylaxis should be addressed and given when necessary.

Pearls

1. Up to half of all pit viper bites may be "dry" (without any injection of venom).
2. Intramuscular epinephrine (0.3 mg 1:1000) given prior to administration of horse serum antivenin may reduce the potential allergic response.



FIGURE 16.37 ■ **Eastern Diamondback Rattlesnake.** The eastern diamondback is the largest US rattlesnake and has a characteristic diamond-shaped pattern on its dorsal aspect. Note the triangular head, which is characteristic of pit vipers. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 16.38 ■ **Western Diamondback Rattlesnake.** The western diamondback causes the most fatalities from snakebites in Mexico and the second most in the United States behind the eastern diamondback. (Photo contributor: Mike Cardwell, MS.)



FIGURE 16.39 ■ **Mojave Green Rattlesnake.** The Mojave green is found in the southwestern United States and Mexico. There are two subspecies, Types A and B, with Type A thought to have the most toxic venom of all North American snakes. Unlike most rattlesnake venoms, Mojave Type A venom contains a potent neurotoxin. (Photo contributor: Mike Cardwell, MS.)



FIGURE 16.40 ■ **Red Diamond Rattlesnake.** The elliptical pupils and heat-sensing pits in this red diamond rattlesnake are characteristic of pit vipers. (Photo contributor: Sean P. Bush, MD.)



FIGURE 16.41 ■ **Cottonmouth.** The cottonmouth is a semiaquatic venomous pit viper that may crawl or swim with its head raised at an angle of 45 degrees. When disturbed, it may open its mouth wide to reveal a white lining. (Photo contributor: Stephen J. Knoop.)



FIGURE 16.42 ■ **Copperhead Snake.** The copperhead is frequently encountered in wooded mountains, abandoned buildings, and damp, grassy areas. It is able to climb low bushes and trees in search of food. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 16.43 ■ **Rattlesnake Envenomation, Minutes Old.** Timber rattlesnake envenomation on the leg of an unsuspecting victim. The fang marks are 4 cm apart. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 16.44 ■ **Rattlesnake Envenomation, Day 1.** This rattlesnake bite shows local swelling, some edema beyond the initial bite site, and a hemorrhagic bleb at 6 hours. (Photo contributor: Sean P. Bush, MD.)



FIGURE 16.45 ■ **Progression of Rattlesnake Envenomation, 7 Weeks.** Seven weeks later, the patient shown in Fig. 16.44 has progressed to tissue loss, eschar formation, and mild changes in coagulation parameters. (Photo contributor: Sean P. Bush, MD.)



FIGURE 16.47 ■ **Severe Rattlesnake Envenomation.** This patient sustained a rattlesnake bite to his hand and presented with marked and progressive swelling, subcutaneous ecchymosis, and a significant coagulopathy. (Photo contributor: Sean P. Bush, MD.)

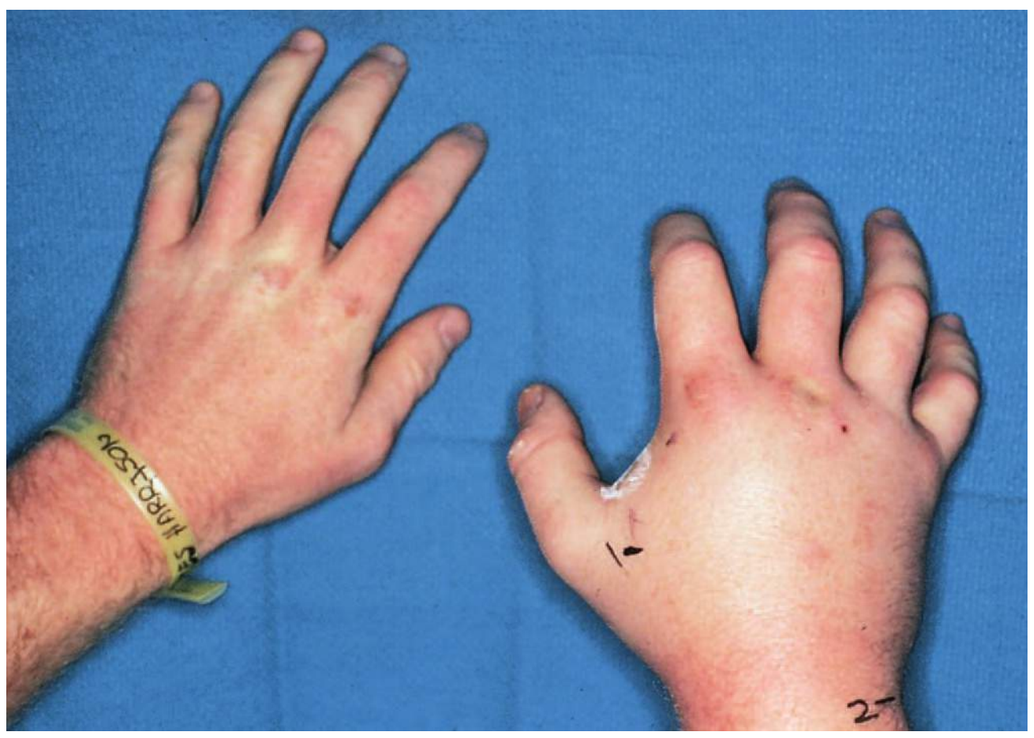


FIGURE 16.46 ■ **Moderate Crotalid Envenomation.** This patient was bitten by a rattlesnake on the dorsal aspect of the right hand and presented with edema extending to the wrist as well as nausea and vomiting. (Photo contributor: Edward J. Otten, MD.)



FIGURE 16.48 ■ **Nonvenomous Snake.** In contrast to the elliptical pupils and triangular head characteristic of pit vipers, this nonvenomous rat snake has a rounded head and circular pupils. (Photo contributor: Sean P. Bush, MD.)

Clinical Summary

The epidemiology of snakebites in tropical regions differs considerably from that seen in more temperate climates. In general, the absolute number of venomous snakes is higher in the tropics and snakes are often located in areas of high population density. The prevalence of bites is also higher due to differences in agricultural and hunting practices, frequent flooding, lack of adequate footwear in many locations, and housing that allows access of snakes into living areas. The annual mortality of snakebite in India possibly exceeds 20,000 and has been stated to be the fifth most common cause of death in Burma (Myanmar). In some native populations in South America, up to 20% of adult deaths are from snakebite. Places where snakebites are common are often in remote locations where medical care may not be immediately available. Signs and symptoms will depend upon the type of envenomation and the amount of toxin injected. Local pain, swelling, and blistering are seen with many snakebites. Clotting disturbances, frank hemorrhage, and shock are seen with many viper bites, while neurotoxicity can be seen with elapid and sea snake bites.

Management and Disposition

Patients should be reassured and kept as immobile as possible. Any involved extremity should be splinted. Tourniquets are generally discouraged, but may be useful in some neurotoxic snakebites. Many modalities such as suction, incision, pumping apparatus, cryotherapy, and electrical shocks have been advocated, but are of no proven benefit and may be harmful. Specific antivenin is used as indicated, but is often either not available or prohibitively expensive.



FIGURE 16.50 ■ **Fer-de-Lance Bite.** This patient was bitten on the index finger by a Fer-de-Lance snake while clearing bush in rural Peru. Note the ecchymosis proximal to the bite. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)



FIGURE 16.49 ■ **Fer-de-Lance (Bothrops jararaca).** Fer-de-Lance, French for "lance head," refers to a number of venomous pit vipers of the genus Bothrops in Central and South America (B atrox, B asper, B jararaca, B lanceolatus). This family of snakes is responsible for more deaths than any other New World snake. (Photo contributor: Cybele Sabino Lisboa, BSc.)



FIGURE 16.51 ■ **Fer-de-Lance Bite (Late).** Local tissue destruction at the site of a Bothrops atrox bite in a Guyanese teenager. (Photo contributor: Ian Jones, MD.)

Pearls

1. There are approximately 3000 species of snakes in the world, with 600 being venomous, and over 200 considered to be medically important. Venomous snakes can be found at altitudes as high as 4000 m in equatorial tropical regions.
2. Neurotoxicity is seen in bites of elapids such as kraits, coral snakes, mambas, and cobras, but is not a feature of the African spitting cobra. The most common cause of death in neurotoxic envenomation is respiratory paralysis.
3. Snake venom ophthalmia is the syndrome caused by the spitting cobras. Severe pain, swelling, and corneal ulceration are seen, and blindness can ensue as a secondary complication.
4. A simple bedside 20-minute blood clotting test can be used to determine the presence of significant coagulopathy in areas without laboratory coagulation capability.
5. The WHO has an online database with all antivenom products currently under production.



FIGURE 16.52 ■ **Old World Viper Bite.** Local hemorrhagic manifestations at the site of a snake bite in a Syrian man. Old World vipers lack the heat sensing pits of the pit vipers. Bites can cause significant hemorrhagic, coagulopathy, and local tissue complications. (Photo contributor: Seth W. Wright, MD.)

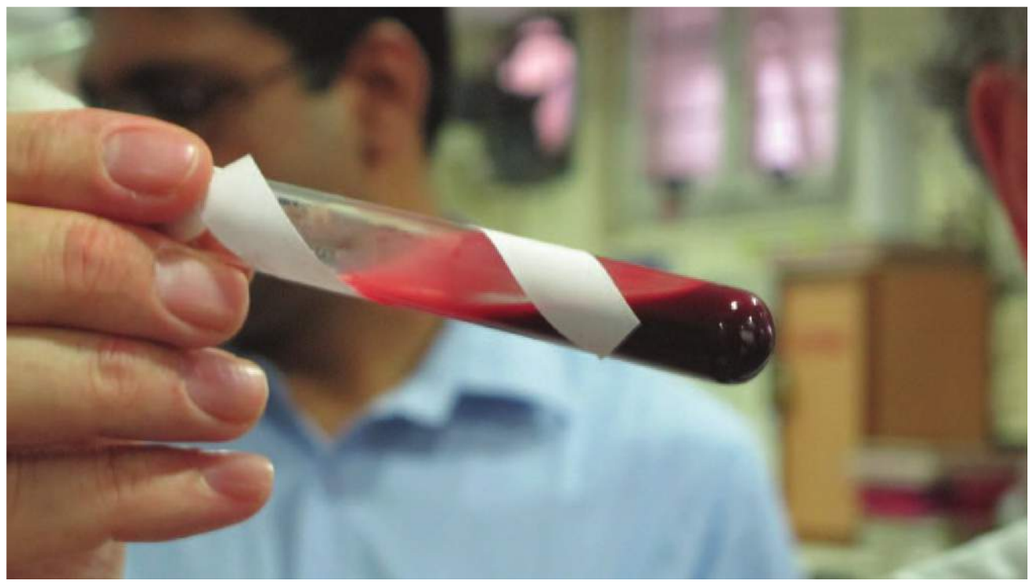


FIGURE 16.53 ■ **Clotting Test.** Positive results of a 20-minute whole blood clotting test are seen with the blood from a man bitten by a Russell's viper. Lack of clotting at 20 minutes correlates closely with other clotting studies. (Photo contributor: Christian Medical College, Vellore, India.)

GILA MONSTER ENVENOMATION

Clinical Summary

The Gila monster (Heloderma suspectum) is a venomous lizard found in the southwestern United States and northwestern Mexico. The only other venomous lizard in the Americas is the Mexican beaded lizard, found from Mexico to Guatemala. Both species are unlikely to bite unless provoked. They secrete venom into their saliva and increase saliva production when they are agitated. When biting their victim, the Gila monster chews rather than injecting, and is known to hang on vigorously and often must be forced to release its powerful grasp. Teeth may break off and contaminate wounds. Like snake venoms, Gila monster and beaded lizard venoms are complex mixtures of proteolytic enzymes and vasoactive substances. The bite may cause pain with local edema and proximal radiation. Tachycardia and hypotension may occur, along with anaphylactic reactions. Systemic complaints may include generalized weakness, nausea and vomiting, diaphoresis, and paresthesias.

Management and Disposition

If not already separated from the victim in the prehospital phase, the lizard must be removed. Putting the lizard under running hot water or prying the jaws apart using an appropriate tool are two options. Caregivers should take precautions against being bitten themselves and should also take care to insure the victim is not bitten again in the removal process. Hypotension can be treated with volume resuscitation and anaphylaxis should be treated with epinephrine. There is no antivenom. Wound care should include copious irrigation and debridement as needed, and imaging should be employed to rule out retained teeth. Tetanus prophylaxis is indicated, but prophylactic antibiotics are generally unnecessary. If the victim has no systemic symptoms several hours after observation, discharge with return precautions is appropriate. Patients with systemic symptoms should be admitted.

Pearls

1. Gila monsters and Mexican beaded lizards have very powerful jaws. Forcible removal is often necessary and must be carefully undertaken to avoid further bites.
2. Patients with systemic symptoms should be admitted, as progression to anaphylactic reactions is possible.



FIGURE 16.55 ■ **Gila Monster Bite.** Gila monster bites may be extremely painful. They usually do not cause severe tissue damage. Retained teeth are a hazard. (Photo contributor: John Sakles, MD.)



FIGURE 16.54 ■ **Reticulate Gila Monster.** The Gila monster (Heloderma suspectum) is one of two venomous lizards found in the Americas. Its range is the southwestern United States and northwestern Mexico. (Photo contributor: Jeff Servoss, US Fish and Wildlife Service.)

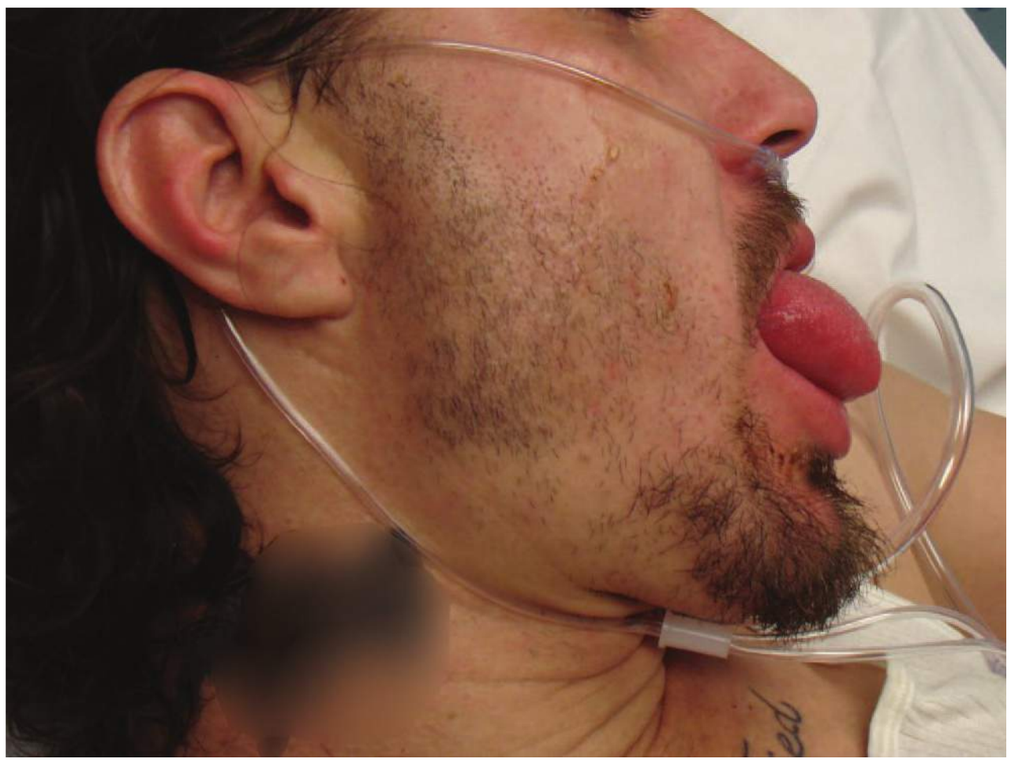


FIGURE 16.56 ■ **Gila Monster Bite, Tongue Swelling.** Gila monster bites may cause anaphylactic reactions. (Photo contributor: John Sakles, MD.)

BLACK WIDOW SPIDER ENVENOMATION

Clinical Summary

The black widow spider (Latrodectus mactans) is the prototype for the genus Latrodectus, several members of which cause human disease. The black widow spider is not particularly aggressive but will defend her web, which is often found in woodpiles, basements, and garages. Most envenomations occur between April and October, with bites most commonly located on the hand and forearm. The clinical presentation of severe and sustained muscle spasm is produced by a neurotoxic protein, which causes the release of acetylcholine and norepinephrine at the presynaptic neuromuscular junction. The initial bite may be mild to moderately painful, but is often missed. Within approximately 1 hour, local erythema and muscle cramping begin, followed by generalized cramping involving large muscle groups such as the thighs, shoulders, abdomen, and back. Associated clinical features can include fasciculations, weakness, fever, salivation, vomiting, diaphoresis, localized sweating at the envenomation site, and a characteristic pattern of facial swelling called Latrodectus facies. Rare cases of seizures, uncontrolled hypertension, and respiratory arrest have occurred.

Management and Disposition

Treatment of the local wound should include cleansing and tetanus prophylaxis. Severe pain and spasm may require intravenous benzodiazepines and opiates. Calcium gluconate infusion has long been recommended to reduce symptoms, although evidence for its efficacy is controversial. Antivenom exists, but carries the same risk as all horse serum products. Antivenom should be considered only in cases of respiratory arrest, seizures, uncontrolled hypertension, and pregnancy.

Pearls

1. Of the five Latrodectus species indigenous to the United States, only three are black and only one has the orange-red hourglass marking.
2. Envenomations by L mactans can mimic an acute abdomen and should be considered in the differential diagnosis of patients presenting with severe acute abdominal pain.



FIGURE 16.58 ■ **Black Widow Spider Bite.** The bite of the black widow spider is clinically subtle. Local reaction is usually trivial, as in this confirmed bite with a small patch of mild erythema. (Photo contributor: Gerald O'Malley, DO.)



FIGURE 16.57 ■ **Black Widow Spider (With Offspring).** Latrodectus mactans, with characteristic hourglass marking on its abdomen. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 16.59 ■ **Black Widow Facies.** A pattern of facial swelling, known as Latrodectus facies, may occur several hours after envenomation. (Photo contributor: Gerald O'Malley, DO.)

BROWN RECLUSE SPIDER ENVENOMATION

Clinical Summary

The brown recluse spider (Loxosceles reclusa) is the prototypical member of the genus Loxosceles, which as a group can produce necrotic arachnidism following envenomation. These small spiders (approximately 1 cm in body length and 3 cm in leg length) have a worldwide distribution and are identified by fiddle-shaped markings on their anterodorsal cephalothorax. Initial envenomation may be painful, although patients often report no recollection of being bitten. Initial stinging gives way to aching and pruritus. The wound then may become edematous, with an erythematous halo surrounding a violaceous center. The erythematous margin often spreads in a pattern influenced by gravity, leaving the necrotic center near the superior aspect of the lesion. Bullae may erupt, and—over a period of 2 to 5 weeks—the eschar sloughs, leaving a deep, poorly healing ulcer. In approximately 10% of cases, systemic symptoms (loxoscelism) are present. Systemic features of brown recluse envenomation may include fever, nausea, vomiting, headache, morbilliform rash, arthralgias, and, in severe cases, hemolytic anemia, coagulopathy, renal failure, and even death. Children are at higher risk of systemic disease.

Management and Disposition

Most cutaneous lesions secondary to brown recluse spider bites can be managed with cold compresses, elevation, loose immobilization, and attention to tetanus immunization. Severe lesions may require reconstructive plastic surgery several weeks after wound stabilization. The use of dapsone to prevent lesion progression is controversial. Any systemic reaction with evidence of hemolysis, hemoglobinuria, or coagulopathy should prompt admission. Hyperbaric oxygen (HBO) therapy and antivenom have been suggested as possible adjuncts, but no clear consensus of preferred treatment has been established.



FIGURE 16.60 ■ **Brown Recluse Spider.** Brown recluse spider with characteristic "fiddle" marking on the anterodorsal aspect of the cephalothorax. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 16.61 ■ **Fiddle Back Marking.** A close-up look at the characteristic fiddle back marking of the brown recluse spider. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 16.62 ■ **Early Brown Recluse Spider Bite.** Early brown recluse spider bite (approximately 8 hours) with a violaceous center surrounded by faint-spreading erythema. Note the "red, white, and blue" appearance. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. The asymmetric spread of erythema, due to the local effects of gravity on the toxin, may help to distinguish a brown recluse spider bite from other arthropod envenomations.
2. If dapsone therapy is to be administered, screening for glucose-6-phosphate dehydrogenase (G6PD) deficiency should be considered.
3. Urinalysis may be helpful in identifying hemolysis early in the clinical course of system reactions.



FIGURE 16.63 ■ **Later Recluse Spider Bite (24 hours).** Brown recluse spider bite at approximately 24 hours. Note asymmetric spread of erythema and early central ulcer formation. (Photo contributor: Edward Eitzen, MD, MPH.)



FIGURE 16.64 ■ **Recluse Necrosis (Weeks).** Within about 2 to 5 weeks, significant brown recluse envenomations may produce a deep, poorly healing ulcer with necrosis. (Photo contributor: Kevin J. Knoop, MD.)

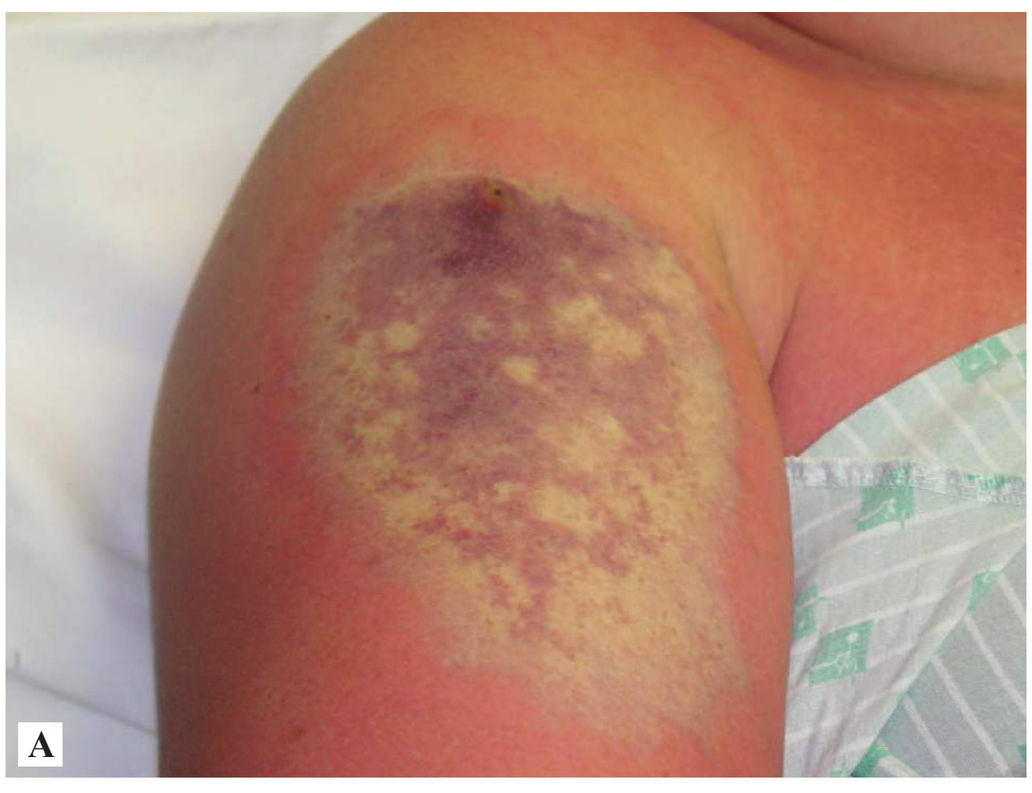


FIGURE 16.65A ■ **Brown Recluse Spider Bite.** Red, white, and blue appearance with significant asymmetric spread of erythema inferiorly down the arm beyond the envenomation site due to effect of gravity. (Photo contributor: Shannon B. Snyder, MD.)



FIGURE 16.65B ■ **Systemic Loxoscelism.** This patient suffered fever, headache, and a diffuse erythematous rash as a result of systemic loxoscelism. Note the brown recluse envenomation on the patient's right arm, seen also in Fig.16.65A. (Photo contributor: Shannon B. Snyder, MD.)

Clinical Summary

The most significant morbidity and mortality from scorpion stings is from the Buthidae family, characterized by a triangular central sternal plate. This family includes the venomous Androctonus genus in northern Africa, Leiurus in the Middle East, Tityus in South America, and Centruroides in North America.

Centruroides are found primarily in the southwestern United States and northern Mexico and are characterized by a variable subaculear tooth beneath the stinger. They may be striped, and are yellow to brown in color. These scorpions tend to hide in crevices, woodpiles, bedding, clothing, and shoes. Envenomation produces a fairly mild local reaction of pain, swelling, burning, and ecchymosis.

Centruroides exilicauda (the bark scorpion) envenomation can lead to progressive symptoms and, very rarely, death. The venom of C exilicauda initially produces local paresthesias and pain (grade 1), which may be accentuated by tapping the involved area. More severe envenomations may produce remote paresthesias (grade II) and either somatic or autonomic nervous system dysfunction (grade III). Systemic symptoms may include tachycardia, nausea, wandering eye

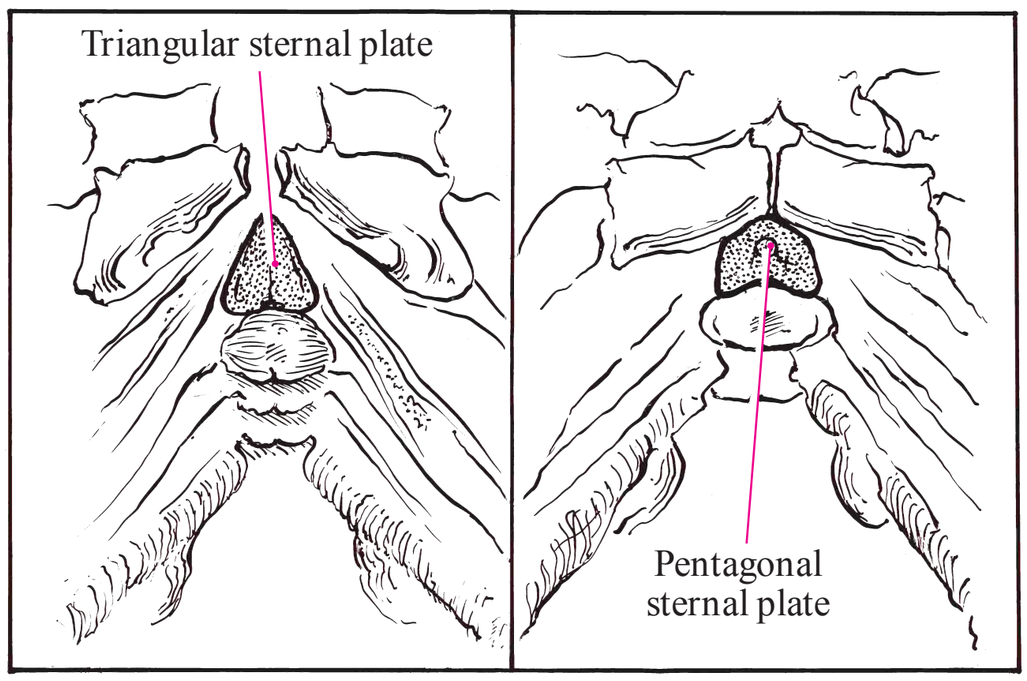


FIGURE 16.66 ■ **Buthidae Sternal Plate.** The Buthidae family is associated with the most significant envenomations and is characterized by triangular sternal plates (left). Members of the other scorpion families have pentagonal sternal plates (right).



FIGURE 16.68 ■ Centruroides exilicauda. Members of this species are yellow to brown and usually less than 5 cm long. Below the stinger is the telson, within which are two glands containing venom. (Photo contributor: Sean P. Bush, MD.)



FIGURE 16.67 ■ **Buthidae Sternal Plate.** The triangular appearance of the sternal plate is well seen in this scorpion, a member of the Buthidae family. (Photo contributor: Sean P. Bush, MD.)

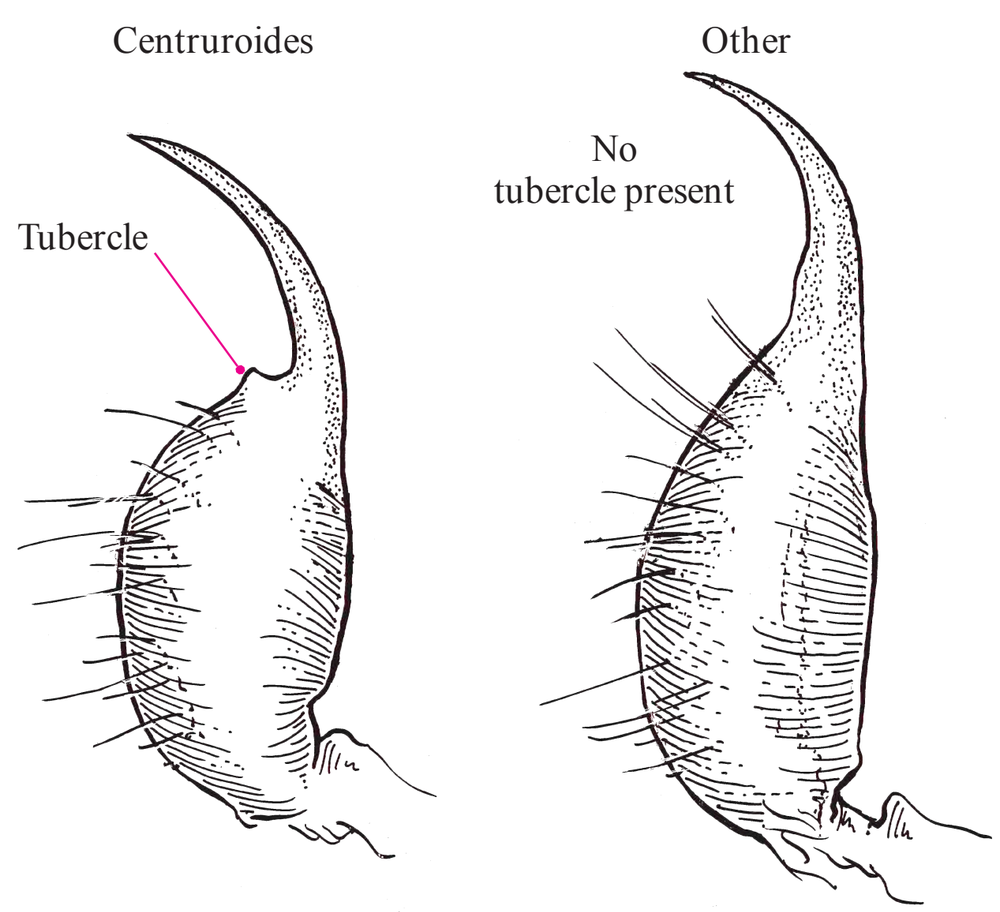


FIGURE 16.69 ■ **Subaculear Tooth.** The barb noted at the base of the stinger is variably present in Centruroides (left) and absent in other species (right).

movements, blurred vision, difficulty breathing, trouble swallowing, restlessness, and involuntary shaking. Both somatic and autonomic dysfunction may be present (grade IV). Systemic reactions tend to be more severe in younger patients and may result in death, usually from respiratory arrest.

Management and Disposition

Treatment depends on the severity of envenomation. Grade I or II envenomations are treated with supportive care (ice, oral analgesia) and tetanus immunization. Envenomations that progress to grade III or IV must be treated aggressively and may require paralysis and intubation for severe spasms. Centuroides antivenom (Anascorp) is available, but is horse-serum based and carries a risk of hypersensitivity reactions. Pain and paresthesias may persist for up to 2 weeks; most systemic symptoms improve within 9 to 30 hours without antivenom treatment and usually peak at about 5 hours.

Pearls

1. Exercise caution when treating pain of C exilicauda envenomations with opioids, as synergistic respiratory depressive effects between venom and opiates may occur.
2. If the scorpion is brought in, it should be examined for the presence of a triangular plate and subaculear tooth.
3. Almost all scorpions, including C exilicauda, fluoresce with intense brightness under cobalt light.



FIGURE 16.70 ■ Centruroides limbatus. Subaculear tooth. A variable subaculear tooth is characteristic of Centruroides. This is an example of a large subaculear tooth on the telson from C limbatus. Centruroides exilicauda typically has a smaller, sometimes subtle "tooth." (Photo contributor: Sean P. Bush, MD.)



FIGURE 16.71 ■ **Scorpion Sting.** Most scorpion envenomations are mild and produce local pain, swelling, paresthesias, and mild ecchymosis. (Photo contributor: Stephen W. Corbett, MD.)

Clinical Summary

Ticks are blood-sucking parasites of people and animals. Ticks cause illness by acting as vectors for pathogens, or by secreting toxins or venoms. Ticks carry more types of infectious pathogens than any other arthropods except mosquitoes. The most important of these include Borrelia (responsible for Lyme disease and relapsing fever), Rickettsia (eg, Rocky Mountain spotted fever [RMSF]), Ehrlichia (ehrlichiosis), viral pathogens (eg, Colorado tick fever), and babesiosis. Rashes are prominent in Lyme disease, RMSF, and southern tick associated rash illness (STARI), sometimes present in relapsing fever, uncommon in Colorado tick fever, and absent in babesiosis.

Clinically important ticks in North America include Ixodes dammini, the deer tick (Lyme disease and babesiosis), Dermacentor andersoni, the wood tick (RMSF and Colorado tick fever), Dermacentor variabilis, the dog tick (RMSF, ehrlichiosis), and Amblyomma americanum, the lone star tick (a very widespread tick implicated in the transmission of Lyme disease outside of the range of I dammini as well as STARI and ehrlichiosis). More than 40 species of ticks can cause tick paralysis. In North America, the most common cause is D andersoni, but A americanum and Ixodes species have also been associated with tick paralysis.

Tick paralysis develops 5 to 6 days after an adult female tick attaches. Over the next 24 to 48 hours, an ascending, symmetric, flaccid paralysis develops. Alternative presentations include ataxia and associated cerebellar findings without muscle weakness or isolated facial paralysis. Resolution of the paralysis after removal of the tick establishes the diagnosis.

Management and Disposition

If still embedded, the tick should be removed promptly by grasping it as close to the skin surface as possible, using blunt curved forceps or tweezers. The tick should be pulled out with slow, gentle traction, taking care not to crush or squeeze the body, which may result in injection of contaminated tick fluids. Other methods of tick removal—such as application of fingernail polish, isopropyl alcohol, or a hot match head—have not been proven to effect detachment and may induce regurgitation of tick contents into the wound.

Patients with tick paralysis may require supportive care, including mechanical ventilation. Patients with tick-borne illnesses may require admission for supportive care or intensive antibiotic treatment, but when clinically appropriate may be treated as an outpatient with appropriate antibiotic therapy.

Pearls

1. Prevention of tick bites includes the use of protective clothing containing N, N-diethylmetatoluamide (DEET).
2. A clear history of a tick bite is present in less than one-third of Lyme disease cases.
3. Unusual neurologic presentations, particularly bilateral peripheral seventh-nerve palsies, should prompt consideration of Lyme disease.
4. Patients with paralysis in endemic areas should be thoroughly searched for embedded ticks.



FIGURE 16.72 ■ **Deer Tick.** Ixodes dammini, the deer tick, is a vector of Lyme disease and babesiosis. (Photo contributor: Centers for Disease Control and Prevention, Atlanta, GA.)



FIGURE 16.73 ■ **Wood Tick.** Dermacentor andersoni, the wood tick, is a vector of Rocky Mountain spotted fever and Colorado tick fever. (Photo contributor: Centers for Disease Control and Prevention, Atlanta, GA.)



FIGURE 16.74 ■ **Lone Star Tick.** Amblyomma americanum, the lone star tick, has been implicated as a vector in STARI. (Photo contributor: Sherman Minton, MD.)



FIGURE 16.76 ■ **American Dog Tick.** Dermacentor variabilis, the dog tick, is a vector of Rocky Mountain spotted fever and ehrlichiosis. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 16.75 ■ **Imbedded Tick.** This lone star tick was found imbedded into the patient's shoulder. It was easily removed intact with tweezers. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 16.77 ■ **Erythema Migrans Rash.** This erythema migrans rash was located at the site of a tick bite in a patient who developed southern tick associated rash illness (STARI). Erythema migrans rash is also associated with Lyme disease. (Photo contributor: Shannon B. Snyder, MD.)

HYMENOPTERA ENVENOMATION

Clinical Summary

The order Hymenoptera includes wasps, hornets, yellow jackets, bees, and ants. Envenomation usually results in local pain, mild erythema, swelling, and pruritus. Severe systemic or toxic reactions may occur from one or multiple stings, manifesting as gastrointestinal symptoms, headache, pyrexia, muscle spasms, or seizures. Anaphylaxis may occur within minutes from a single sting, and may cause death from airway obstruction and/or cardiovascular collapse. A serum sickness–type reaction may occur 7 to 14 days after envenomation.

Solenopsis invicta was imported from South America and is the most prominent fire ant in the United States. These ants are primarily found in the South and build mound nests in open grass settings, commonly in urban yards. Disturbing the nests may result in severe swarming attacks, a common occurrence in the unsuspecting barefoot victim. Bites are painful and produce sterile pustules that crust over in a few days.

Management and Disposition

Anaphylaxis is treated with conventional therapy with careful attention to airway management. Local reactions may be treated with ice packs, steroid cream, and oral antihistamines. Opiates may be needed for severe pain.

Pearls

1. Honeybee stings are usually apparent since the stinger apparatus, including barb and venom sac, is often detached and present on the patient's skin. These should be removed by scraping them off, as grasping with tweezers may result in the release of more venom.
2. "Brazilian killer" or "Africanized" bees are present primarily in the southwestern United States. Their venom is not known to be more toxic than that of other bees, but their aggressiveness, tendency to swarm in large numbers, and ability to travel long distances make them potentially more dangerous to humans.
3. Immediately remove any rings when envenomations are located on the hands or feet as marked local swelling can occur, putting the distal digits at risk for ischemia.



FIGURE 16.79 ■ **Paper Wasp Nest.** A typical paper wasp nest in a roof corner. Disturbance of a nest may result in swarming attacks. (Photo contributor: Clay B. Smith, MD.)



FIGURE 16.78 ■ **Paper Wasp.** Paper wasps are found throughout the world and often establish nests close to or within human dwellings. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 16.80 ■ **Fire Ant Mound.** This typical fire ant mound is a raised area of dirt in an urban yard. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 16.81 ■ **Fire Ant Bites.** These fire ant bites on the anterior knee occurred after this patient knelt on a mound. These bites are 3 days old; the initial sterile pustules have begun to crust over. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 16.82 ■ **Honeybee Envenomation.** Many honeybee stingers (barbs and venom sacs) are seen on this patient's cheek and ear and along the hairline. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 16.83 ■ **Honeybee Stingers.** The barbs and attached venom sacs (stinger apparatus) after removal from the patient. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 16.84 ■ **Imbedded Honeybee Stinger.** Note the venom sac, still pulsating and attached to the honeybee stinger imbedded in the victim. (Photo contributor: Lawrence B. Stack, MD.)

CATERPILLAR, MITE, AND CENTIPEDE ENVENOMATION

Clinical Summary

Caterpillar venom apparatus typically consists of barbed spines arranged in clumps or scattered about the dorsal surface of the insect. These are purely defensive in nature. Patients who are stung commonly have intentionally handled the insect or have had accidental skin contact while gardening. Envenomated patients typically present with acute pain followed by focal erythema and swelling. Caterpillars with a less sophisticated venom apparatus or low-potency venom may cause simple focal pruritus or urticaria, though some caterpillars are capable of producing a very painful sting requiring aggressive pharmacological pain control. Systemic symptoms are rare. The puss caterpillar, or wooly slug (Megalopyge opercularis) is perhaps the most well-known and important venomous caterpillar in the United States. Wooly slug caterpillars have a widespread distribution, appear hairy and flat, and may reach a length of 4 cm.

Chiggers are the larvae of trombiculid mites and may inflict multiple intensely pruritic bites on their victims. They are parasitic only as larvae and infest humans by crawling onto them and latching on. Proper clothing precautions and repellents are usually effective in reducing unpleasant chigger infestations.

Centipedes are venomous arthropods that have one pair of legs per body segment. The first segment contains hollow curved "fangs" (really modified legs) bearing venom glands at the bases, which are capable of penetrating human skin. Centipedes generally use venom to kill prey, but, when provoked, may envenomate humans and produce local intense burning pain, erythema, and swelling. Systemic reactions may occur but are uncommon.

Management and Disposition

Treatment of caterpillar, mite, and centipede envenomations is purely supportive and consists of pain control, antihistamines, topical antipruritic creams, and basic wound care.

Pearls

1. Attached caterpillar spines may be removed easily with adhesive tape.
2. Infiltration with local anesthetics may be useful in markedly painful centipede envenomations or for the removal of retained "fang" fragments.
3. The caudal appendages of centipedes are not associated with a venom apparatus.

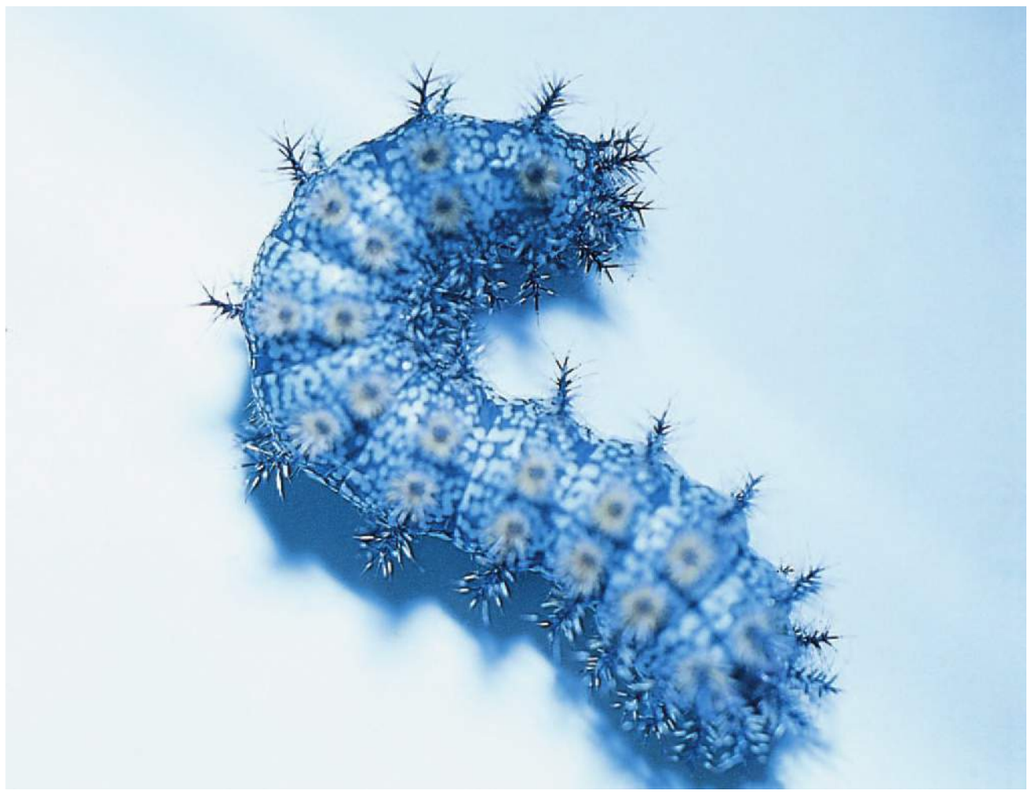


FIGURE 16.85 ■ **Caterpillar with Barbed Spines.** Typical garden caterpillar with barbed spines arranged in clumps. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 16.86 ■ **Caterpillar Sting.** Appearance of a caterpillar sting at 2 hours. The patient presented with moderate pain and severe itching. Note how the erythema follows the pattern of the caterpillar. (Photo contributor: Alan B. Storrow, MD.)

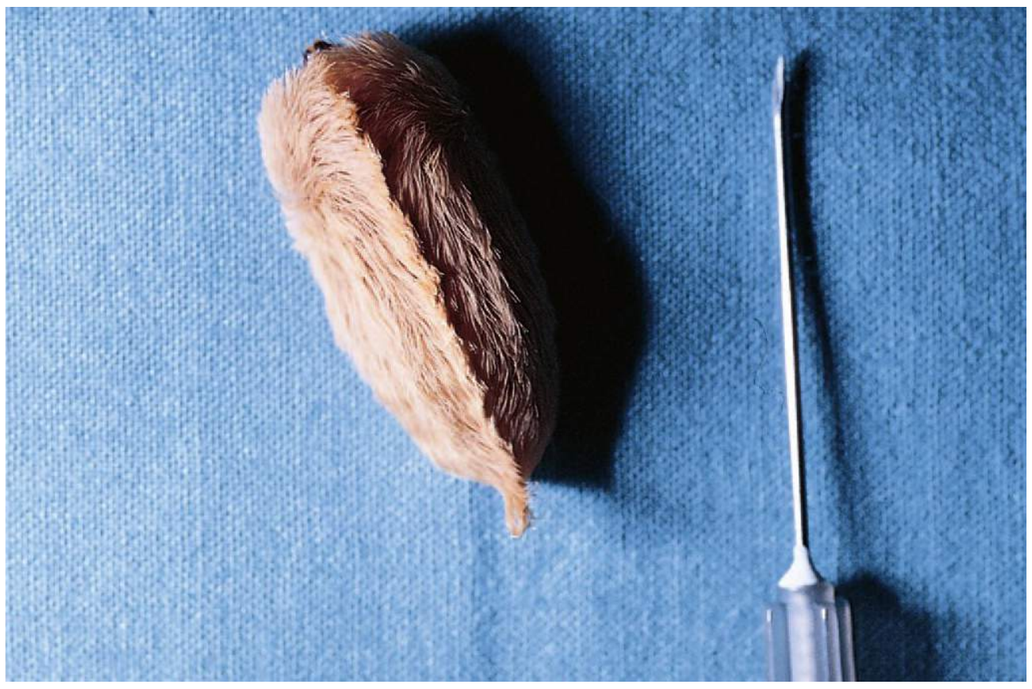


FIGURE 16.87 ■ **Puss Caterpillar.** The "puss caterpillar" or "woolly slug" is likely the most important venomous caterpillar in the United States. The hairy appearance and small hair tail is characteristic. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 16.88 ■ **Centipede.** Note the curved "fangs" (actually modified legs) on the first segment of this centipede from Texas. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 16.89 ■ **Chigger Bites.** Chigger bites on a leg appear as puncta surrounded by erythema. (Photo contributor: Kevin J. Knoop, MD, MS.)

MIDDLE EAR SQUEEZE

Clinical Summary

A middle ear squeeze (barotitis media) results from a decrease in pressure within the middle ear as an individual descends through water or is exposed to an increase in atmospheric pressure that can be seen in descending aircraft or while driving in mountainous terrain. According to Boyle's law, as pressure increases, volume must decrease proportionately. At a depth of approximately 4 ft, the pressure difference is great enough to collapse the eustachian tube and cause obstruction. If attempts to equalize the pressure (eg, Valsalva or Frenzel maneuver) fail, ascent is necessary or injury may ensue. If a diver continues to descend, hemorrhage and edema occur within the middle ear and rupture of the TM may occur. The influx of water into the middle ear may cause extreme vertigo and lead to a diving disaster.

Barotitis media may present with pain only (grade 0), TM erythema (grade 1), erythema and mild TM hemorrhage (grade 2), gross TM hemorrhage (grade 3), free middle ear blood (grade 4), or free blood with TM perforation (grade 5).

Management and Disposition

Treatment includes decongestants and appropriate analgesia. Antihistamines may be of use for allergy-related eustachian tube dysfunction. Antibiotics are recommended for preexisting infections or for TM rupture. Most cases resolve spontaneously within hours to days. The patient should not resume diving until the condition has resolved or the TM is completely healed.

Pearls

1. Barotitis media is the most common medical problem associated with diving.
2. Associated barotraumatic injuries should be considered when the diagnosis of barotitis media is made.



FIGURE 16.90 ■ **Barotitis Media.** Tympanic membrane erythema and mild hemorrhage consistent with barotitis media. (Photo contributor: Richard A. Chole, MD, PhD.)

Clinical Summary

Mask squeeze results when a diver fails to maintain the balance between the air pressure within his or her mask and the external water pressure during descent. If a diver descends without equalizing pressure by exhaling through the nose, significant negative air pressure will exist inside the mask. The net result may include rupture of capillary beds, leading to conjunctival hemorrhage and skin ecchymosis.

Management and Disposition

Treatment consists of ascent and essentially supportive care. A history of recent eye surgery or use of anticoagulant medication should be sought. A thorough eye examination should be performed and ophthalmologic consultation considered if warranted.

Pearls

1. Diver education and proper diving technique minimize the risk of mask squeeze.
2. Special consideration should be given to patients with anticoagulant use or recent keratotomy, as corneal incisions heal relatively slowly.



FIGURE 16.91 ■ **Mask Squeeze.** Mask squeeze in a diver who descended to 45 FSW without exhaling into his mask. (Photo contributor: Kenneth W. Kizer, MD; reprinted with permission from Auerbach PS (ed). Wilderness Medicine: Management of Wilderness and Environmental Emergencies. 3rd ed. St. Louis, MO: Mosby-Year Book; 1995. Copyright © Elsevier.)

STINGRAY ENVENOMATION

Clinical Summary

Stingrays are found throughout the oceans of the world. Stingrays are not typically aggressive and the majority of envenomations are defensive in nature. Injuries typically involve the lower extremity if the animal is stepped upon or upper extremity if the animal is handled. Fatal injuries have been reported from chest trauma, which may result in perforation of the myocardium. Stingray envenomation occurs when a reflexive and forceful forward thrust of the caudal spine or spines of the animal impacts the victim, producing a puncture wound or laceration. The force of injection causes the integumentary sheath covering the spine to be driven into the wound, fragmenting and potentially releasing venom, mucus, pieces of the sheath, and spine fragments deep within the wound. Envenomation typically produces immediate and intense pain, edema, and bleeding. The initially dusky or cyanotic wound may progress to erythema, with rapid fat and muscle hemorrhage. Systemic symptoms may include nausea, vomiting, diarrhea, diaphoresis, muscle cramps, fasciculations, weakness, headache, vertigo, paralysis, seizures, hypotension, and syncope.

Management and Disposition

The wound should be irrigated immediately and primary exploration accomplished to remove any visible debris. Pain relief should be initiated early, and opiates may be needed. Stingray venom is made up of heat-labile polypeptides that may be inactivated by immersion in hot water (43°C-46°C [110°F-115°F]) for 30 to 90 minutes. After soaking, wounds should be formally explored, debrided, and dressed for delayed primary closure or primary closure with drainage.

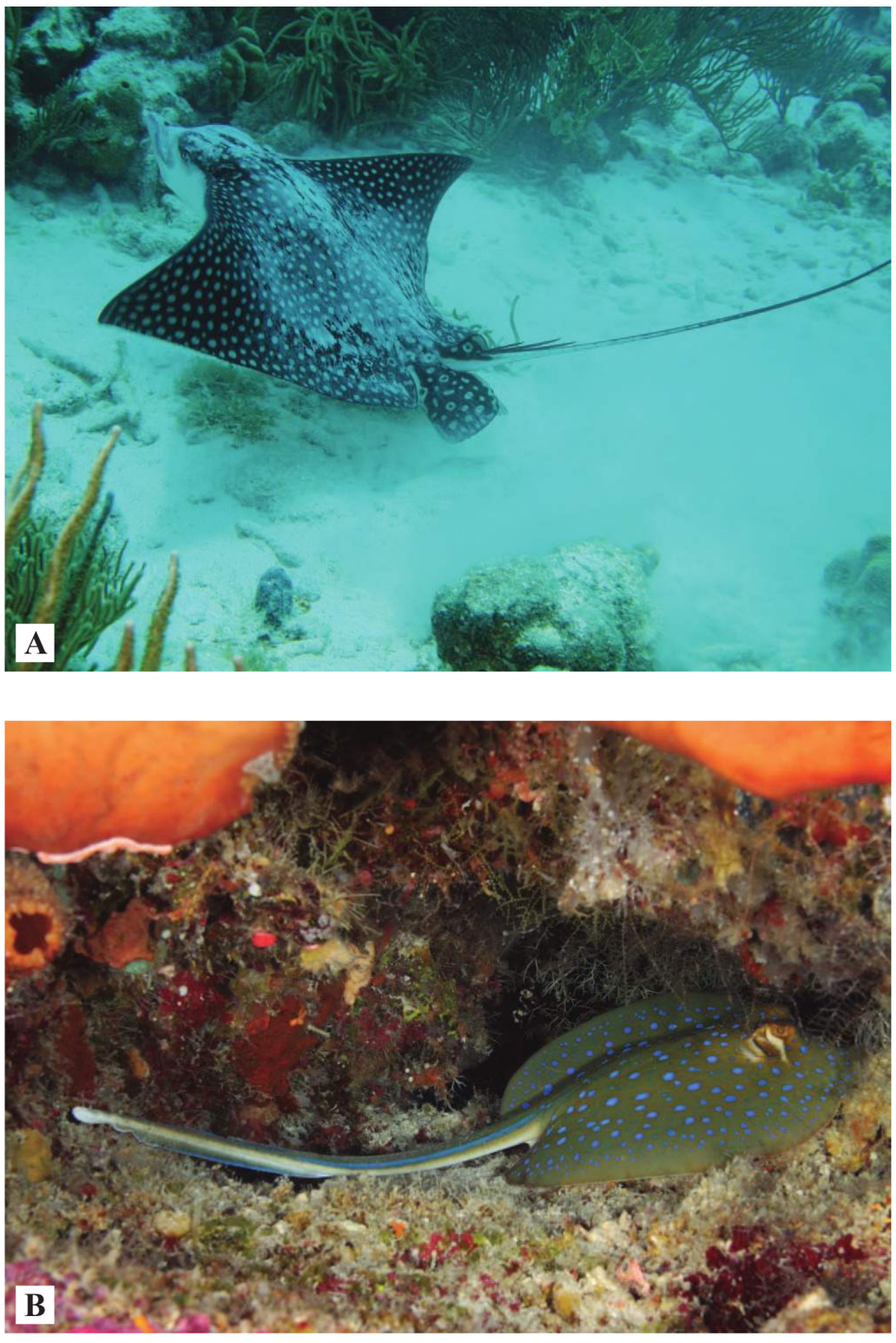


FIGURE 16.92 ■ **Stingrays.** (**A**) Spotted eagle stingray. This graceful stingray was photographed in waters off the coast of Bonaire. Note the three venomous spines at the base of the tail. (Courtesy of Lynne Bentsen, RN.); (**B**) Blue spotted stingray. This stingray was photographed in waters off Indonesia. Stingrays often dwell on the ocean floor and may burrow into the sand, leading to envenomation by accidentally stepping on the animal. (Photo contributor: Ian D. Jones, MD); (**C**) Southern stingray. This stingray was photographed in the Caribbean. The coloring of the stingray tends to blend with the ocean floor, leading to injury from inadvertent stepping on the stingray. (Photo contributor: Kevin J. Knoop, MD.)

Surgical consultation may be warranted in certain injury locations. A radiograph should be obtained after debridment to rule out retained foreign body. Broad-spectrum antibiotics covering marine organisms are recommended. Patients can usually be discharged home after a 3- to 4-hour observation period if no systemic symptoms arise. Tetanus prophylaxis should be given if indicated.

Pearls

1. Aggressive debridement is of primary importance in managing stingray wounds, as retained foreign bodies are a common problem.
2. Bacteria cultured from marine envenomations are extremely diverse. Antibiotics chosen should include coverage of staphylococci, streptococci, and Vibrio species.

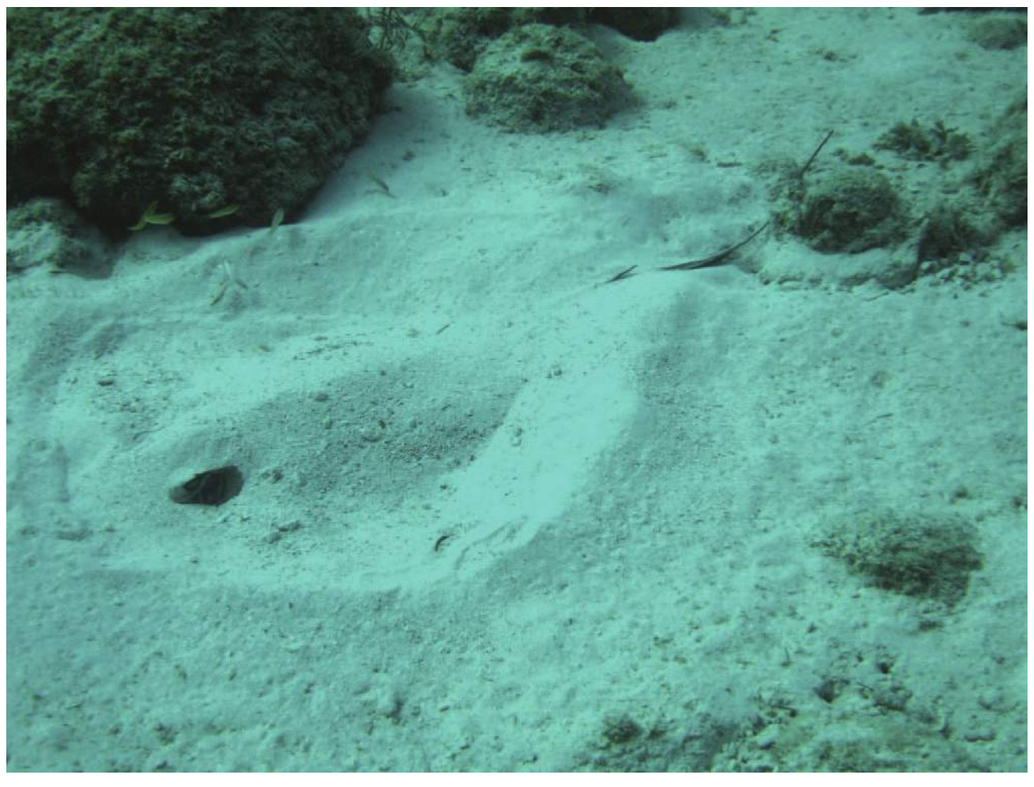


FIGURE 16.93 ■ **Buried Stingray.** Stingrays like to burrow into the sand on the ocean floor, making them very difficult to see and easy to inadvertently step on. (Photo contributor: Keven Reed, OD.)



FIGURE 16.94 ■ **Stingray Barb in Forearm.** This envenomation occurred after the fisherman inadvertently caught a stingray and was envenonmated taking the animal off the hook. The barb is imbedded in the patients forearm. (Photo contributor: John Meade, MD.)



FIGURE 16.95 ■ **Stingray Envenomation.** Puncture wound from stingray envenomation in a lower extremity. (Photo contributor: Daniel L. Savitt, MD.)

SEA URCHIN ENVENOMATION

Clinical Summary

Sea urchins belong to the phylum Echinodermata and are nonaggressive, slow-moving creatures. Envenomation usually occurs after accidental contact with the organism. Long, brittle, venom-filled spines or specialized jaw-like appendages (pedicellariae) are responsible for the injury. Other echinoderms, notably the crown of thorns starfish, may also cause injury via similar mechanisms. The spines frequently break and pedicellariae can remain attached and active for several hours. They may advance into muscle or joint spaces and cause infection or injury from the venom itself. The usual presentation is burning pain progressing to localized muscle aches. Erythema and edema may be present. Multiple envenomations may produce systemic symptoms including nausea, vomiting, abdominal pain, paresthesias, numbness, paralysis, hypotension, syncope, or respiratory distress. While the envenomation causes a reaction that may be quite painful, deaths, though reported, are exceedingly rare.

Management and Disposition

Following envenomation, the affected area should be submersed in hot water (43°C-46°C [110°F-115°F]) for 30 to 90 minutes. Pedicellariae may be removed by applying shaving cream and gently scraping with a razor. Obvious embedded spines should be removed. An x-ray should be performed to rule out retained foreign body. In certain cases, CT, ultrasound, or MRI may be considered. Hand wounds often require surgical debridement. Retained spines often dissolve spontaneously; however, granulomas may form, producing locally destructive inflammation. Antibiotics may be useful in certain cases, and tetanus prophylaxis should be addressed.



FIGURE 16.96 ■ **Sea Urchin Spine on X-Ray.** This x-ray is of a patient who was reaching into a rock crevasse in the ocean and experienced sudden sharp pain. Note the retained sea urchin spine in the fifth digit adjacent to the proximal phalanx. (Photo contributor: Marion Berg, MD.)

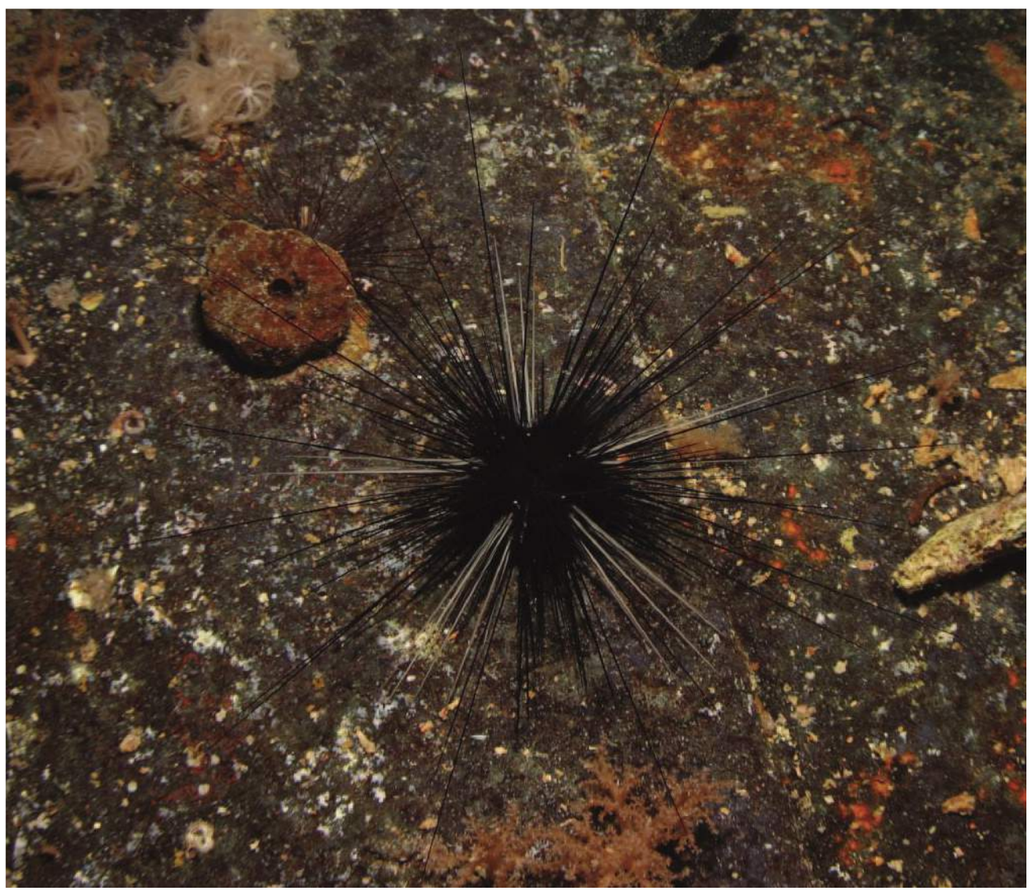


FIGURE 16.97 ■ **Long-Spined Sea Urchin.** The majority of urchins are nonvenomous and only cause injury from puncture wounds. Their spines may break off in the victim, resulting in a high likelihood of infection. (Photo contributor: Ian D. Jones, MD.)



FIGURE 16.98 ■ **Sea Urchin Envenomation.** Note the multiple puncture wounds on the foot of a patient who accidentally stepped on a sea urchin in shallow water. (Photo contributor: Saralyn R. Williams, MD.)

Pearls

1. Sea urchin envenomation involving a joint may produce severe synovitis.
2. Some species of sea urchin contain dye, which may give the false impression of a retained spine.
3. Sea urchins known to be hazardous to humans are generally found in the Indian Ocean, Pacific Ocean, and Red Sea.



FIGURE 16.99 ■ **Fire Urchin.** A beautiful fire urchin, found in the Indo-Pacific. (Photo contributor: Ian D. Jones, MD.)

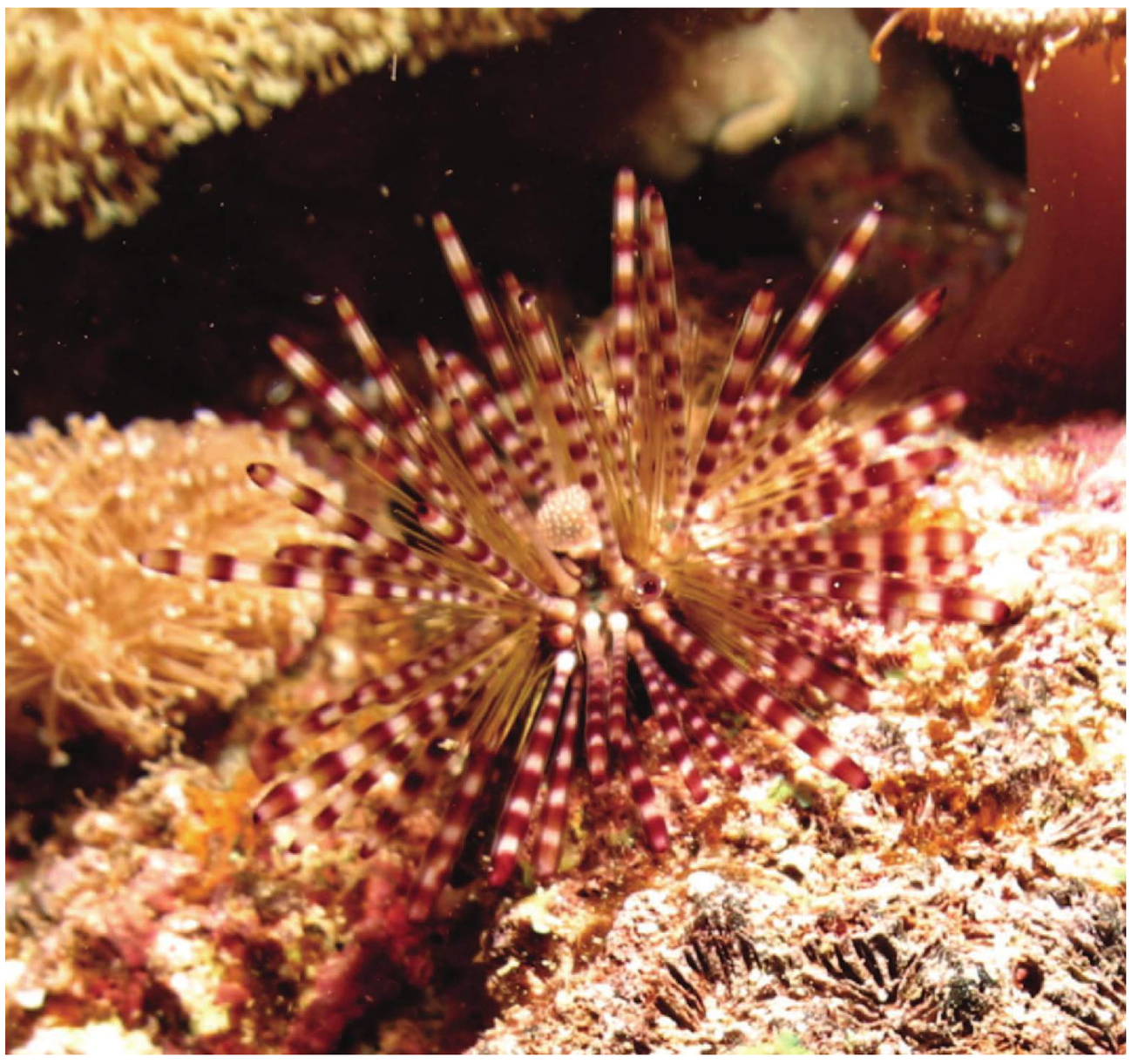


FIGURE 16.100 ■ **Fire Urchin.** Fire urchins are venomous sea urchins. Envenomations from fire urchins, while extremely painful, are seldom if ever fatal. (Photo contributor: Ian D. Jones, MD.)

Clinical Summary

The phylum Coelenterata contains approximately 10,000 different species, of which several hundred are a danger to humans. This diverse group includes hydrozoans (eg, Portuguese man-of-war, stinging hydrozoans, and fire coral), scyphozoans (ie, "true" jellyfish), and anthozoans (ie, soft corals, stony corals, and anemones). They account for more marine envenomations than any other phylum. The important species involved in human injuries have stinging cells called nematocysts. Nematocysts are enclosed in venom sacs and are present in tentacles that hang from air-filled structures. After external contact, the nematocysts are discharged from their sacs, often penetrating the skin, and release their venom. Nematocyst venom is an extremely complex substance containing numerous proteins and enzymes. Clinical presentation following envenomation ranges from the mild dermatitis to cardiovascular and pulmonary collapse. Mild envenomations usually result in a self-limited papular inflammatory eruption associated with burning and limited to areas of contact. Moderate to severe envenomations produce a spectrum of neurologic, cardiovascular, respiratory, and gastrointestinal symptoms. Anaphylactoid reactions—including hypotension, dysrhythmias, bronchospasm, and cardiovascular collapse—may occur, resulting in unexplained drownings.

Management and Disposition

Concurrently with primary resuscitation, nematocyst decontamination should be accomplished beginning with



FIGURE 16.102 ■ **Hydrozoan Envenomation.** This hydrozoan sting resulted from accidental direct contact with the diver's finger while taking an underwater photograph. (Photo contributor: Ian D. Jones, MD.)



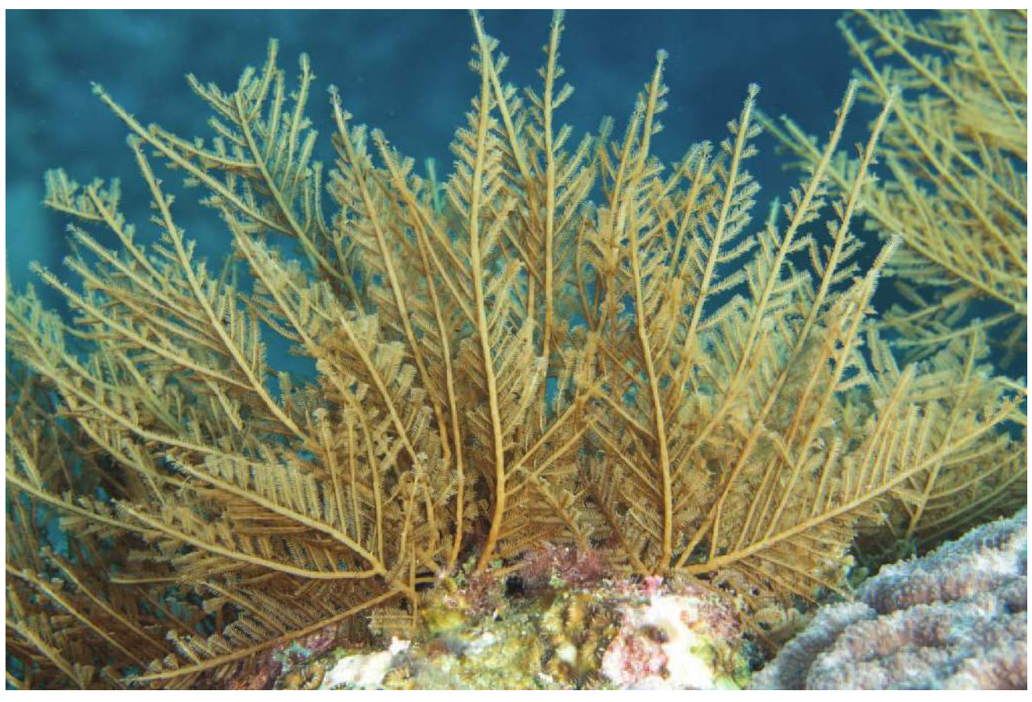


FIGURE 16.101 ■ **Stinging Hydrozoan.** Similar to fire coral, contact with stinging hydrozoan results in local burning pain followed by erythematous papules or urticarial eruptions and blisters. (Photo contributor: Shawn Miller.)



FIGURE 16.103 ■ **Fire Coral.** (**A**) Fire coral. (Photo contributor: Shawn Miller); (**B**) Fire coral envenomation. After contact, fire coral most commonly causes immediate local burning pain, followed by erythematous papules or urticarial eruptions. Pruritus may last for several days. (Photo contributor: Emily R. Stack.)

seawater flushing. The hypotonic nature of fresh water, as well as isopropyl alcohol, may cause additional nematocysts to fire and should be avoided. A 5% solution of acetic acid (vinegar) applied for at least 30 minutes is the most widely accepted mechanism for inactivating nematocysts. It has been suggested to remove tentacles with the application of shaving cream, followed in 5 minutes by a careful scraping with a firm, dull object (eg, tongue blade, credit card). Pruritus may be treated with antihistamines. Pain may be addressed with immersion in hot water or with systemic analgesics. Any victim with systemic symptoms requires at least 6 to 8 hours of observation because rebound phenomena are common.

Pearls

1. The box jellyfish (Chironex fleckeri) is generally considered the most deadly of marine animals and is most predominant in Australian and Southeast Asian waters.
2. The detached tentacles of some species may contain active nematocysts for months, even when fragmented on the beach or floating in water.
3. The Portuguese man-of-war is present in the Floridian Atlantic coast and the Gulf of Mexico. It is known to have a neurotoxin that may cause severe pain. Death, while reported, is extremely rare.



FIGURE 16.104 ■ **Portuguese Man-of-War.** The beautiful Portuguese man-of-war with multiple tentacles dangling in the water. The tentacles, filled with venomous nematocysts, can extend several meters in length. (Photo contributor: Adam Laverty.)

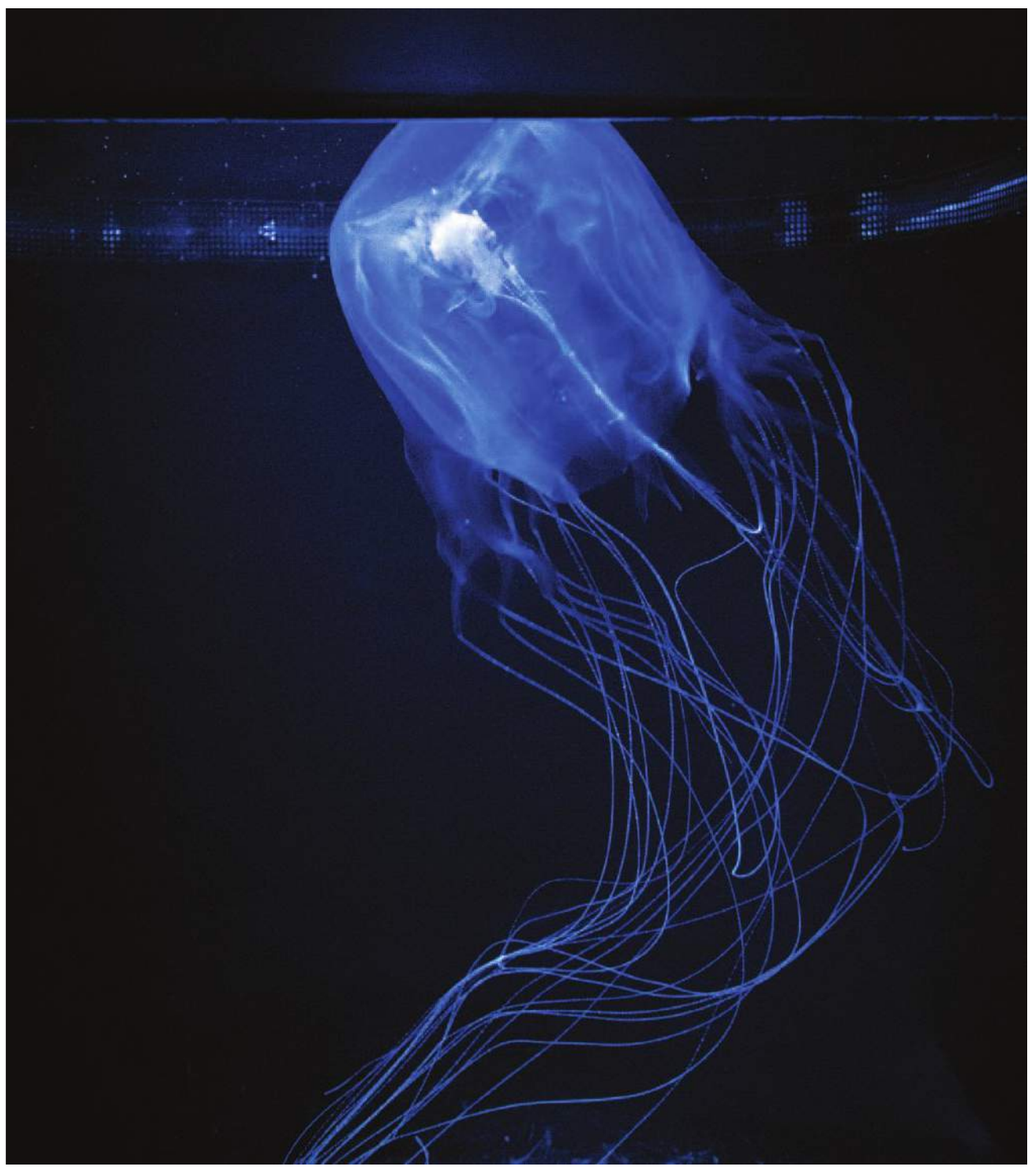


FIGURE 16.105 ■ **Box Jellyfish (Under Blue Light).** The box jellyfish delivers an unbearably painful sting. Envenomations can be life-threatening and require immediate treatment. (Photo contributor: Shawn Miller.)



FIGURE 16.106 ■ **Coelenterate Envenomation.** The sharp angulations and undulations characteristic of jellyfish envenomation. (From Halstead BH. Venomous Marine Animals of the World. Washington, DC: US Government Printing Office; 1965.)

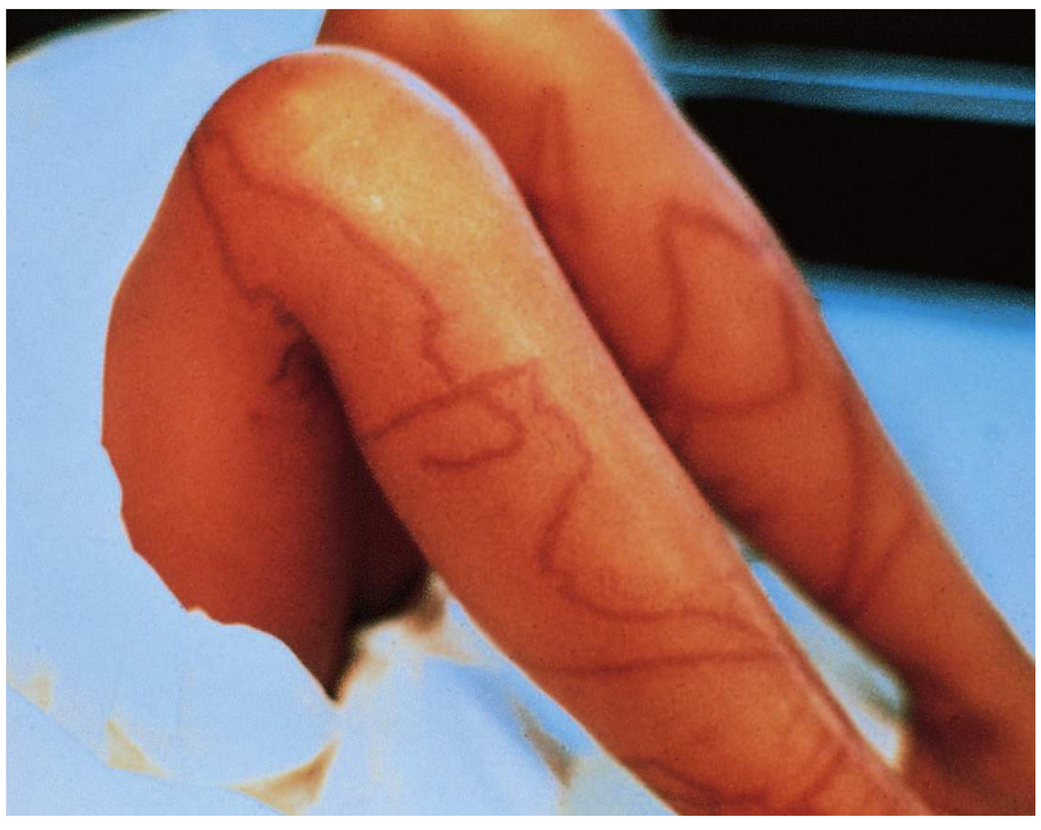


FIGURE 16.107 ■ **Coelenterate Envenomation.** Jellyfish envenomation on the lower extremities. (Photo contributor: Department of Dermatology, Naval Medical Center, Portsmouth, VA.)



FIGURE 16.108 ■ **Box Jellyfish Sting.** This 5-year-old child was stung by a box jellyfish (Chiropsalmus quadrigatus) off the coast of Japan. Severe envenomations may be fatal. (Photo contributor: Keven Reed, OD.)



FIGURE 16.109 ■ **Sea Anemone Envenomation.** Contact with sea anemones results mainly in local skin irritation, initially manifest as pruritus, burning, throbbing, and, sometimes, radiation of pain to other areas. The area involved may reveal blistering, local edema, and violaceous petechial hemorrhages. Skin lesions are confined to the areas of contact. (Photo contributor: Susan Scott.)

Clinical Summary

Marine dermatitis, also known as "sea bather's eruption," is a pruritic condition commonly mislabelled sea lice. Symptoms usually occur a few minutes to 12 hours after exposure. The offending organisms are probably numerous and include the larval form of the thimble jellyfish and the planula form of the sea anemone Edwardsiella lineata. The rash consists of erythematous wheals and papules, which may be extremely pruritic. Systemic manifestations include fever, malaise, headache, conjunctivitis, and urethritis. Unlike cercarial dermatitis, marine dermatitis primarily affects areas of the body covered by caps, fins, and bathing suits.

Cercarial dermatitis, or "swimmer's itch," occurs when humans become accidental hosts of nonhuman schistosomes. This causes an immune response that results in itching, erythema, and mild edema. After 60 minutes, the classic signs are red macules that become pruritic papules 3 to 5 cm in diameter and surrounded by erythema.



FIGURE 16.110 ■ **Marine Dermatitis.** Typical appearance of marine dermatitis. (Photo contributor: Richard A. Clinchy III, PhD.)

Management and Disposition

Marine dermatitis is self-limited, rarely persisting beyond 2 weeks. The dermatitis may be partially prevented by a vigorous soap-and-water scrub after saltwater bathing. Treatment is symptomatic, and calamine lotion with 1% menthol may bring relief. Topical steroids may provide additional relief. In severe cases, oral antihistamines and corticosteroids may be necessary.

Cercarial dermatitis is treated with isopropyl alcohol or calamine lotion. Severe cases may require systemic corticosteroids, whereas bacterial infection may require topical or oral antibiotics.

Pearls

1. Marine dermatitis primarily affects areas covered by caps, fins, and bathing suits.
2. During late spring and summer, incidence increases along the US east coast. In one reported outbreak, 25% of individuals entering the water were affected.



FIGURE 16.111 ■ **Cercarial Dermatitis.** The discrete and highly pruritic papules of cercarial dermatitis commonly occur on exposed body areas. (Photo contributor: David O. Parrish, MD. Reproduced with permission from Parrish DO. Seabather's eruption or diver's dermatitis. JAMA. 1993;270:2300-2301. Copyright © 1993 American Medical Association. All rights reserved.)

SCORPION FISH STING

Clinical Summary

Scorpion fish are colorful venomous marine animals found primarily in tropical waters. Their exotic beautiful appearance has made them increasingly popular among marine aquarists in the United States. Many envenomations have resulted from mishandling. They are well camouflaged in the wild and stings are usually caused by accidentally stepping on them. Scorpion fish are grouped into the genera Pterois (lion fish), Scorpaena (scorpion fish proper), and Synanceja (stone fish), with the severity of envenomation progressing, respectively. All have multiple spines associated with venom glands; envenomation results from skin puncture followed by venom release. Immediately following a sting, the victim experiences intense pain that, if untreated, lasts for hours. The site may become warm, erythematous, and edematous and vesicles may arise. Lion fish stings are painful but relatively mild, usually limited to localized pain and tissue responses. Severe systemic effects are more common with stone fish stings and may produce a constellation of cardiovascular, pulmonary, neurologic, and gastrointestinal sequelae. Death has been reported from stone fish stings.

Management and Disposition

Hot water immersion (43°C-46°C [110°F-115°F]) for 30 to 90 minutes is initiated as soon as possible. Rebound pain is common and is treated with repeated hot water immersion. The wound should be inspected for pieces of spine and sheath. Thorough warm saline irrigation should be performed along with wound exploration. Severe pain is treated with local injection of lidocaine without epinephrine and with narcotic analgesia. Antibiotic prophylaxis should be considered in high-risk wounds and tetanus status addressed.

Pearls

1. Stone fish stings are the most dangerous. Severe systemic reactions may occur. Antivenin (Commonwealth Serum Labs, Australia) is available.
2. Scorpion fish venom is heat-labile. Hot water immersion is effective in treating pain and inactivating venom.
3. Since 2002, Indo-Pacific lionfish have been reported in increasing numbers from New York to the Bahamas.



FIGURE 16.113 ■ **Scorpion Fish.** Note the well-camouflaged scorpion fish, leading to accidental contact and envenomation in unsuspecting divers. (Photo contributor: Kevin J. Knoop, MD.)



FIGURE 16.112 ■ **Lion Fish** (Pterois volitans). Envenomations occur by contact with the erectile spines on the dorsal, pelvic, and anal fins of the fish. (Photo contributor: Kevin J. Knoop, MD.)



FIGURE 16.114 ■ **Stonefish.** The extremely venomous stonefish is spectacularly camouflaged. Envenomation usually results when the unsuspecting victim steps on the fish. (Photo contributor: Ian D. Jones, MD.)

Clinical Summary

Cone snails, also referred to as cone shells or cone fish, are venomous predatory marine gastropod molluscs capable of inflicting a painful and even dangerous sting to humans. They may be found in wide distributions across the world's oceans and seas. Cone snails prey on marine worms and fish, using their venom apparatus to inject the victim with paralytic toxins. The cone snail uses a dart like tooth which fires out from the shell. Many have beautiful patterns on their shell, making them attractive to collect for unsuspecting divers. When disturbed, the snail may deploy its harpoon like tooth and envenomate the handler. Smaller species inflict a sting similar to that of a wasp, but envenomations from larger species may cause intense pain, swelling, paresthesias, and vomiting. Rarely, severe envenomations may progress to muscle paralysis, respiratory failure, and death.

Management and Disposition

Cone snail venom is heat-labile, so initial management consists of hot water immersion similar to the treatment of scorpion fish stings. Rebound pain is common and is treated with repeated hot water immersion. Inspect the wound for foreign material, and perform thorough warm saline irrigation. Severe pain may be treated with local injection of lidocaine without epinephrine and with narcotic analgesia. Consider antibiotic prophylaxis in high-risk wounds and address tetanus status. Intravenous edrophonium has been reported in the treatment of cone snail–associated paralysis.

Pearls

1. Cone snail venom is heat-labile. Hot water immersion is effective in treating pain and inactivating venom.
2. Cone snail venom may have potential therapeutic uses, including the management of severe pain and intractable epilepsy.
3. Although fatal cone snail envenomations are rare, severe stings may cause death secondary to the rapid onset of respiratory paralysis.



FIGURE 16.116 ■ **Geographic Cone Snail.** This cone snail is seen in full view with tentacles, water siphon, and short pharynx (proboscis) visible. The geographic cone snail is highly venomous with a potentially fatal sting. (Photo contributor: Shawn Miller.)



FIGURE 16.115 ■ **Cone Snail.** The beautiful shells of the cone snail make it attractive to collect for divers. Envenomations commonly occur on the hand and fingers of unsuspecting victims due to handling. (Photo contributor: Kevin J. Knoop, MD.)



FIGURE 16.117 ■ **Cone Snail (old).** As cone snails age, the shells may become covered in adherent materials, making them difficult to see and increasing the risk of inadvertent contact. (Photo contributor: Kevin J. Knoop, MD.)

SEA SNAKE ENVENOMATION

Clinical Summary

Sea snakes are members of the elapid family of snakes that have evolved to survive in a variety of ocean habitats. There are over 70 species of sea snakes with the majority found in the tropical portions of the Indian and Pacific Oceans. Sea snakes are not found in the Atlantic or Carribean. Most species of sea snakes live close to the coast although there is a pelagic species, Pelamis platurus, that is found across a large swath of the Pacific Ocean. Reports vary regarding the docile nature of sea snakes, although it is well reported that some species have a much higher propensity to bite. Bites occur when the animal is disturbed or inadvertently handled. There are many reports of bites occurring as fishermen are removing the snake from their nets. Because of the small size of the organism's fangs, bites may be inconspicuous and are often painless. The majority of bites do not result in envenomation.

The most potent toxin in sea snake venoms, similar to other elapids, is a neurotoxin that competes for acetylcholine at the neuromuscular junction. Severe envenomations may ultimately lead to paralysis and respiratory failure. Additional components of the venom are myotoxic and may result in rhabdomyolysis and renal failure.

The symptoms of sea snake envenomation generally occur within 30 minutes of envenomation and may be quite variable. Common symptoms include confusion, headache, myalgias, weakness of the facial muscles, followed by an ascending flaccid paralysis, and ultimately respiratory arrest.

Management and Disposition

Treatment for a sea snake bite includes a combination of supportive care and the immediate administration of polyvalent sea snake antivenom. Supportive care should include intubation if indicated. The patient should also be carefully observed for signs of rhabdomyolysis and hyperkalemia. The patient should be aggressively hydrated to avoid further complications such as renal failure. Consultation with a toxicologist or individual experienced in managing sea snake envenomations should be considered.

Pearls

1. An effective polyvalent antivenom exists and is effective against envenomation from all species of sea snakes.
2. In addition to respiratory support, victims of sea snake envenomation should be observed for the development of rhabdomyolysis and hyperkalemia.



FIGURE 16.118 ■ **Sea Snake.** The sea krait, seen here swimming free in the ocean, is very docile and rarely bites humans unless provoked. (Photo contributor: Ian D. Jones, MD.)



FIGURE 16.119 ■ **Sea Snake.** A close-up of a sea snake found feeding on a reef. (Photo contributor: Kevin J. Knoop, MD.)

Clinical Summary

There are over 750 species of cephalopods found throughout the world's oceans. Cephalopods are members of the phylum mollusca and include cuttlefish, squid, octopuses, and the nautilus. Several species of cephalopods are known or thought to be venomous but only the blue-ringed octopus, genus Hapalochlaena, poses a significant threat to humans.

Four separate species of blue-ringed octopus are known to exist. All are small and none exceed a few inches in size. The animal is found in tide pools and shallow reefs across a wide area of the Indo-Pacific ranging from Japan to Australia. The blue-ringed octopus is generally not aggressive and is relatively nondescript when left in peace. When threatened, however, the animal will flash or pulsate with striking iridescent blue rings. Bites occur when the animal is disturbed or inadvertently handled. It may be found hiding in old bottles or shells in tidal pools and in this way poses a hazard to collectors. Like other cephalopods, the blue-ringed octopus has a beak that can be powerful enough to penetrate a wet suit. Some bites may be relatively painless.

Blue-ringed octopus venom has several identified components. The most powerful component of the venom, tetrodotoxin, is a potent sodium channel blocker that is identical to the toxin found in pufferfish. Once bitten, symptoms may develop in as few as 10 minutes and include perioral paresthesias, facial weakness, and nausea and vomiting. In some patients, especially children, hypotension may be present. As symptoms progress, flaccid paralysis and respiratory arrest may result.

Management and Disposition

Treatment for a blue-ringed octopus bite is entirely supportive, as no antivenin currently exists. The patient almost always has a normal sensorium unless profoundly hypoxic or hypercarbic. Primary management of severe envenomations includes management of hypotension and prolonged ventilatory support until the toxin can be degraded and excreted.

Pearls

1. No antivenin exists for a blue-ringed octopus envenomation.
2. The patient's mental status is usually normal and a victim may be fully conscious despite being paralyzed and apneic.



FIGURE 16.120 ■ **Blue-Ringed Octopus.** The beautiful but highly venomous blue-ringed octopus. (Photo contributor: Russell C. Gilbert, MD.)



FIGURE 16.121 ■ **Blue-Ringed Octopus.** The blue-ringed octopus demonstrates its iridescent blue colorations, seen when the animal is threatened. (Photo contributor: Kevin J. Knoop, MD.)

ERYSIPELOID

Clinical Summary

Erysipeloid, also known as "fish handler's disease," is a bacterial skin infection caused by Erysipelothrix rhusiopathiae. This condition is frequently seen in those who handle raw meat, fish, and shellfish. The offending organism enters the body through a break in the skin and causes a local infection within 2 to 7 days. Lesions are characterized by an edematous central purplish-red area, surrounded first by central clearing and then circumscribed by an advancing raised, erythematous ring. The area is usually pruritic and painful and may be associated with fever, malaise, and regional lymphadenopathy.

Management and Disposition

If left untreated, erysipeloid will usually resolve spontaneously in about 3 weeks. Skin infections may be treated with penicillin VK, cephalexin, or erythromycin. If severe infection occurs, it should be treated with appropriate broad-spectrum antibiotics.

Pearls

1. A history of occupational or recreational exposure to fish or shellfish is the key to diagnosis.
2. E rhusiopathiae is usually resistant to aminoglycoside antibiotics, thus these should be avoided.



FIGURE 16.122 ■ **Erysipeloid Envenomation.** Typical appearance of Erysipelothrix rhusiopathiae skin infection. (Photo contributor: Paul S. Auerbach, MD; reprinted with permission from Auerbach PS (ed). Wilderness Medicine: Management of Wilderness and Environmental Emergencies. 3rd ed. St. Louis, MO: Mosby-Year Book; 1995. Copyright © Elsevier.)

Clinical Summary

Poison ivy, oak, and sumac cause more cases of allergic contact dermatitis in the United States than all other allergens combined. At least 70% of the US population is sensitive to these Toxicodendron species. The allergen urushiol is responsible for Toxicodendron dermatitis, also known as rhus dermatitis. Urushiol is found in many other plants, including in the skin of mangos. Mangos are a common cause of plant dermatitis in subtropical and tropical areas, including Hawaii.

The dermatitis begins as pruritus and redness usually within 2 days of exposure in susceptible persons. The degree of dermatitis depends on the patient's degree of sensitivity, amount of allergen exposure, and the reactivity of the skin at the exposed body location. The dermatitis may vary from erythema alone or may include papules, vesicles, and bullous eruptions. A linear distribution of the cutaneous lesions is strongly suggestive of Toxicodendron dermatitis. This distribution occurs after plant parts have rubbed against the skin or when contaminated fingernails have scratched it.

Management and Disposition

An immediate rinse or shower with warm water and soap may minimize the reaction. If symptoms are limited to erythema and papules and a small surface area, calamine lotion or topical steroid sprays may provide adequate symptomatic relief. Pruritus may be decreased with oral antihistamines (eg, diphenhydramine or cyproheptadine) and oatmeal baths. Vesicles and bullae may benefit from Domeboro compresses (60 minutes three times daily) to help dry these lesions and relieve pruritus. Systemic corticosteroids tapered over 3 weeks are used in severe reactions. Secondary infection should be treated with systemic antibiotics against staphylococcal and streptococcal species.

Pearls

1. Fluid from the vesicles or bullae does not contain any allergen.
2. Removal of the allergen from the skin within 30 minutes of exposure may prevent dermatitis.
3. Deliberate removal of allergen from under the fingernails may prevent spreading.
4. Treatment with systemic steroids for less than 3 weeks may result in rebound exacerbations of the dermatitis.



FIGURE 16.123 ■ **Poison Ivy.** Toxicodendron radicans (poison ivy—shrub or climbing vine). Note that the leaves of poison ivy have three leaflets and the stems are commonly reddish orange. Poison ivy occurs throughout the United States. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 16.124 ■ **Poison Oak.** Toxicodendron diversiloba (poison oak). Like poison ivy, the terminal part of the branch has a cluster of three shiny leaves. It grows as a tree or woody shrub and occurs west of the Rocky Mountains. (Photo contributor: Ken Zafren, MD.)



FIGURE 16.125 ■ **Poison Sumac.** Toxicodendron vernix (poison sumac). Note that the leaves of poison sumac have 7 to 13 leaflets. It grows as a tree or woody coarse shrub. Only one species of poison sumac is found in the United States. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 16.126 ■ **Poison Sumac Dermatitis.** A moderately severe local reaction to poison sumac. Note the vesicles, bullae, and exudates characteristic of a contact dermatitis. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 16.127 ■ **Poison Oak Dermatitis.** Erythematous papules and vesicles and vesicles. This fire-fighter was exposed to urushiol, the allergen of poison oak, ivy, and sumac, in smoke from burning poison oak. (Photo contributor: Ken Zafren, MD.)



FIGURE 16.128 ■ **Poison Ivy.** Erythematous papules and vesicles in a linear distribution consistent with Toxicodendron dermatitis. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 16.129 ■ **Poison Ivy.** A highly pruritic vesicular eruption of Toxicodendron dermatitis on the patient's forearm. Note the linear groupings of vesicles, likely from scratching with contaminated fingernails. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 16.130 ■ **Mango Dermatitis.** This patient was catching mangos as they dropped from a tree in Mexico. The rash progressed rapidly over a 48-hour period. This patient can eat mangos, but is sensitive to the skin of the fruit. (Photo contributor: Melissa Gray, WEMT.)

SPOROTRICHOSIS

Clinical Summary

Sporotrichosis is a fungal skin infection caused by Sporothrix schenckii, an organism primarily found on plants and flowers and in soil; the problem is common among gardeners and florists. It also affects those who handle animals, since the fungus may inhabit animals' claws. Infection arises when contaminated thorns, spines, or claws penetrate the victim's skin. After an average incubation period of 3 weeks, localized infections become apparent. "Fixed" cutaneous infections are localized to the inoculation site and are manifest as 2- to 4-mm papules or nodules. They may ulcerate, become surrounded by raised erythema, and are typically painless. Progression to lymphocutaneous infections occurs in about 70% of cases. Patients present with a nodule at the site of penetration, with appearance of subcutaneous nodules and skip areas along lymphatic tracks later. The lesions may wax and wane over months to years. Patients with cutaneous sporotrichosis typically lack systemic symptoms.

Management and Disposition

Sporotrichosis may be successfully treated with oral potassium iodide for 1 month after clinical manifestations have resolved. Alternative therapy includes oral itraconazole, ketoconazole, or terbinafine, while disseminated infections may require intravenous amphotericin B. Outpatient therapy is appropriate for nondisseminated infections. Tetanus status should be addressed.

Pearls

1. Fungal cultures and tissue biopsy cultures are somewhat useful to confirm the diagnosis.
2. Treatment must be continued for 1 month following clinical resolution to eradicate S schenckii.
3. Although rare, a pulmonary form of sporotrichosis after inhalation exposure has been reported.



FIGURE 16.131 ■ **Fixed Sporotrichosis.** The ulcer and surrounding erythema of fixed cutaneous sporotrichosis could be confused with a brown recluse spider bite. (Photo contributor: Edward J. Otten, MD.)



FIGURE 16.132 ■ **Lymphocutaneous Sporotrichosis.** Lymphatic spread is common in cutaneous sporotrichosis. (Photo contributor: Kevin J. Knoop, MD, MS.)

Chapter 17

TOXICOLOGICAL CONDITIONS

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Jimsonweed. Jimsonweed seed pod with dried seeds. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)

AMPHETAMINE TOXICITY

Clinical Summary

Amphetamines as a class may be abused by ingestion, insufflation ("snorting"), parenteral injection, and smoking. "Ice" refers to a pure preparation of methamphetamine hydrochloride in a large crystalline form. Designer amphetamines include 3,4-methylenedioxymethamphetamine (MDMA, "ecstasy"), paramethoxyamphetamine (PMA), and synthetic cathinones (ingredients in "bath salts"). While the central nervous system (CNS) targets of these compounds are serotonergic and dopaminergic pathways, the clinical presentation is manifested as a sympathomimetic toxidrome.

Although clinically indistinguishable from cocaine toxicity, the duration of effects is appreciably longer. The most common cardiovascular manifestations of toxicity are tachycardia and hypertension, although myocardial ischemia has been reported. Despite the cardiovascular effects, CNS toxicity is the primary reason most amphetamine users present for medical care. Presentations may range from increased anxiety to life-threatening agitated delirium with hyperthermia. Visual and tactile hallucinations and psychoses are common. Poor dentition is common among chronic users ("meth mouth"), and appears multifactorial in nature.

Management and Disposition

Treatment focuses upon the signs and symptoms of toxicity. As with other causes of sympathomimetic toxicity, initial management includes control of the agitation to prevent other complications (eg, rhabdomyolysis). Benzodiazepines are the first-line therapy for agitation; large repeated doses may be required. Severe hyperthermia requires evaporative cooling techniques. Hypertonic sodium may be useful for MDMA-associated cerebral edema and seizures.

Pearls

1. In addition to the medical complications associated with methamphetamine use, the manufacture of methamphetamine is associated with exposure to a number of toxic chemicals and risk for severe burns.
2. The hyperthermia associated with acute amphetamine poisoning may result in end-organ damage similar to patients with heat-stroke-like illness.
3. MDMA may result in syndrome of inappropriate antidiuretic hormone (SIADH) with subsequent hyponatremia and cerebral edema.



FIGURE 17.1 ■ **"Ice" Methamphetamine.** An example of the "ice" form of amphetamines with a pipe. (Photo contributor: US Drug Enforcement Administration.)



FIGURE 17.2 ■ **Ecstasy.** Examples of the candy-like appearance of ecstasy tablets. (Photo contributor: US Drug Enforcement Administration.)



FIGURE 17.3 ■ **Early "Meth Mouth."** "Meth mouth," the extensive and accelerated dental caries associated with chronic methamphetamine abuse. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 17.4 ■ **Advanced "Meth Mouth."** Note the severe dental decay in this chronic methamphetamine abuser. (Photo contributor: Carson Harris, MD.)

NEW DESIGNER DRUGS: "BATH SALTS" AND "SPICE"

Clinical Summary

The cathinones are derivatives of a naturally occurring phenethylamines found in the leaves of the Catha edulis (khat) plant. Some of these compounds include mephedrone, methylone, and methylenedioxypyrovalerone (MDPV). Adverse effects of these compounds include cardiac, psychiatric, and neurologic signs and symptoms, similar to effects seen with other sympathomimetics.

Synthetic cannabinoids have been promoted as "spice" or "incense" products that contain a substance usually labeled as "JWH" followed by a number. The designation reflects the original synthesis by John W. Huffman, an organic chemist at Clemson University. These compounds have full agonist effects at the cannabinoid receptors, in contrast with tetrahydrocannabinol (THC), which demonstrates only partial agonism. While cannabinoids such as marijuana do not typically result in sympathomimetic effects, the synthetic cannabinoids may also cause acute sympathomimetic toxicity including seizures and tachydysrhythmias.



FIGURE 17.5 ■ **Khat Plant**. The leaves of the khat plant (Catha edulis) are chewed for the stimulant effects. The leaves must be fresh in order for cathinone to be present. (Photo contributor: US Drug Enforcement Administration.)



FIGURE 17.6 ■ **Bath Salts.** Examples of typical packaging for bath salts. (Photo contributor: US Drug Enforcement Administration.)

Forensic analysis of these products has demonstrated significant variability in types and amounts of active products over time, both between and within marketed brands. As a result, individuals may experience different clinical effects with different exposures to the same product.

Management and Disposition

Patients under the influence of one of the synthetic compounds should be approached and managed similar to other acute sympathomimetic poisonings.

Pearls

1. The ingredients in "bath salts" are not detected by routine testing for amphetamines as a class.
2. Agitation, psychosis, and seizures are common clinical effects of synthetic cannabinoid use.



FIGURE 17.7 ■ **Synthetic Cannabinoids—"Spice."** Examples of the packaging of synthetic cannabinoids which are labeled "not for human consumption." (Photo contributor: John G. Benitez, MD, MPH.)

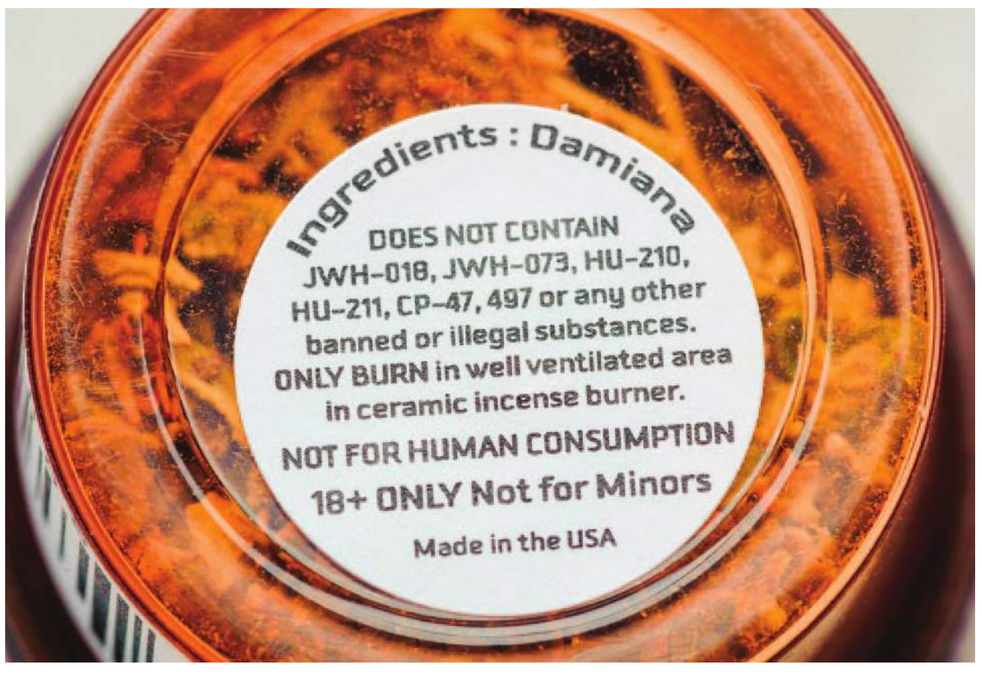


FIGURE 17.8 ■ **Synthetic Cannabinoids Disclaimer.** Close-up of the language used to suggest that the contents do not contain illegal substances. (Photo contributor: John G. Benitez, MD, MPH.)

Clinical Summary

Cocaine is a natural alkaloid derived from the leaves of Erythroxylum coca. Cocaine hydrochloride (powder cocaine) is a crystalline white powder. "Crack," the free-base of cocaine hydrochloride, is an off-white substance named both for its rock-like appearance ("rock") and due to the sound it makes when heated. In contrast to powder cocaine, crack may be smoked as it vaporizes instead of burning.

Cocaine intoxication manifests as a sympathomimetic toxidrome, with tachycardia, hypertension, diaphoresis, mydriasis, delirium, and hyperthermia. Increased muscular activity may result in rhabdomyolysis. Numerous neurological complications have been reported after cocaine use, including subarachnoid hemorrhage, intracerebral hemorrhage, cerebral infarction, and seizures.

Cardiovascular toxicity, including acute myocardial infarction, is well described after cocaine use. Dysrhythmias, including supraventricular tachycardia, atrial fibrillation and flutter, ventricular tachycardia, ventricular fibrillation, and torsades de pointes have been reported. Cocaine is a sodium channel blocker and may cause QRS widening on the electrocardiogram (ECG). Aortic dissection and rupture have been associated with cocaine use.

Pulmonary effects of cocaine use include reactive airway disease exacerbation, pneumothorax, pneumomediastinum, and cardiogenic and noncardiogenic pulmonary edema (NCPE). "Crack-lung" refers to an acute pulmonary syndrome of dyspnea, hypoxia, and diffuse pulmonary alveolar infiltrates.

Management and Disposition

Treatment is primarily supportive, and focuses upon the signs and symptoms of toxicity. No specific antidote exists. Cardiac monitoring is indicated for symptomatic patients. Initial management focuses upon control of agitation and hyperthermia, and prevention of complications (eg, rhabdomyolysis). The use of neuroleptic agents is relatively contraindicated for cocaine-associated psychomotor agitation due to the negative effects of these agents on thermoregulation, seizure threshold, and the potential for dysrhythmias. Benzodiazepines are the first line of therapy for agitation. They also appear beneficial in the management of cocaine-associated chest pain. Sodium bicarbonate administration should be considered for QRS widening in the setting of acute cocaine poisoning.



FIGURE 17.10 ■ **Drug Paraphernalia.** Crack pipe sequestered in the rectum during a patient's arrest, resulting in laceration of the hemorrhoidal venous plexus and massive hemorrhage. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)

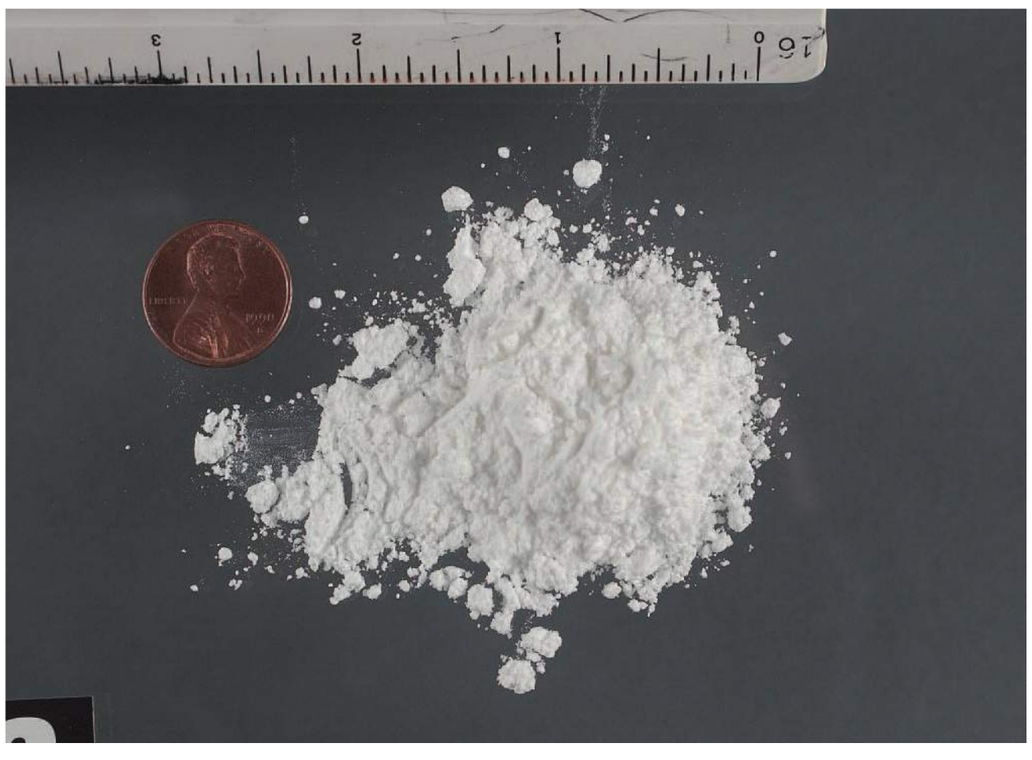


FIGURE 17.9 ■ **Cocaine Powder.** Cocaine powder. (Photo contributor: US Drug Enforcement Administration.)

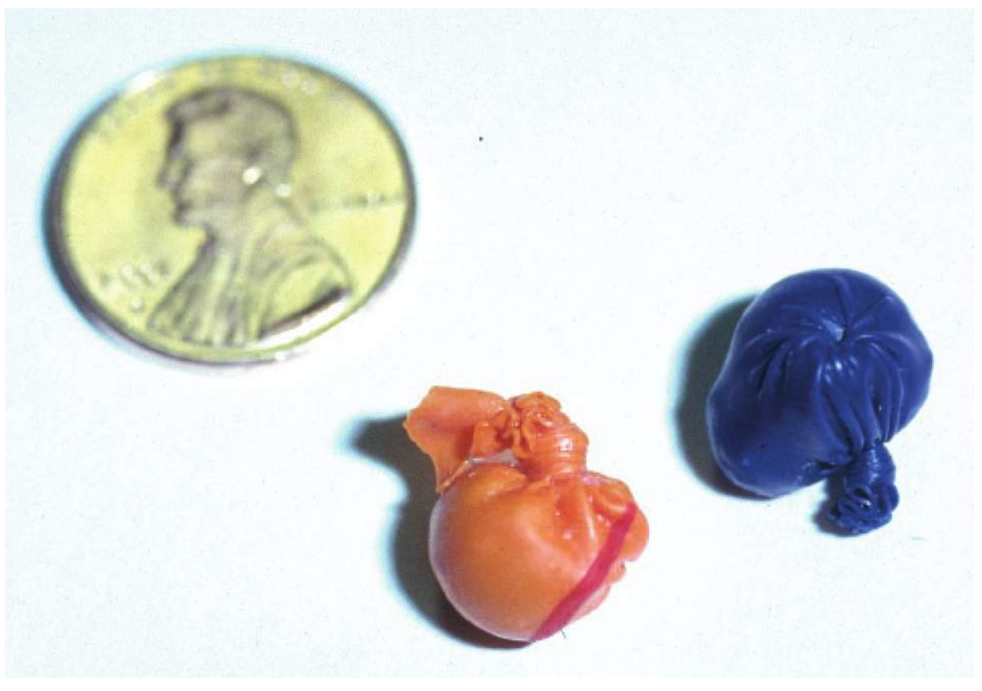


FIGURE 17.11 ■ **Cocaine Body-Packing.** Cocaine-filled balloon packets from the stool of a cocaine "body-packer" (penny used for scale). Radiopaque packets are often visible on KUB radiograph. Severe toxicity may result in the event of a ruptured packet. (Photo contributor: Alan B. Storrow, MD.)

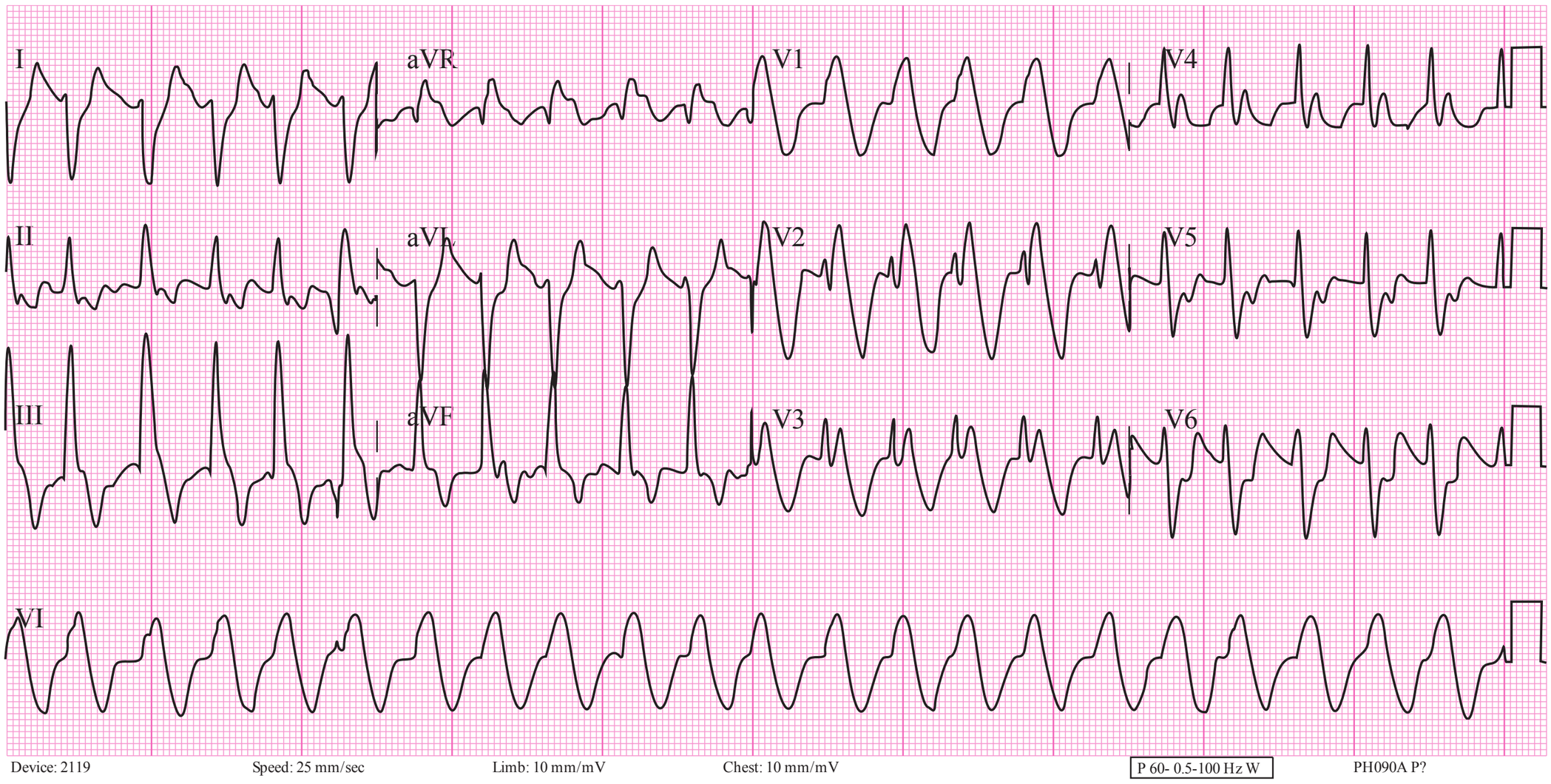


FIGURE 17.12 ■ **Cocaine Cardiotoxicity.** The initial 12-lead EKG of a patient with acute cocaine and cocaethylene poisoning demonstrating with wide complex rhythm from the sodium channel blocking effects. His initial serum pH was 6.8. (Photo contributors: Thomas Babcock, MD and Laurie Lawrence, MD.)

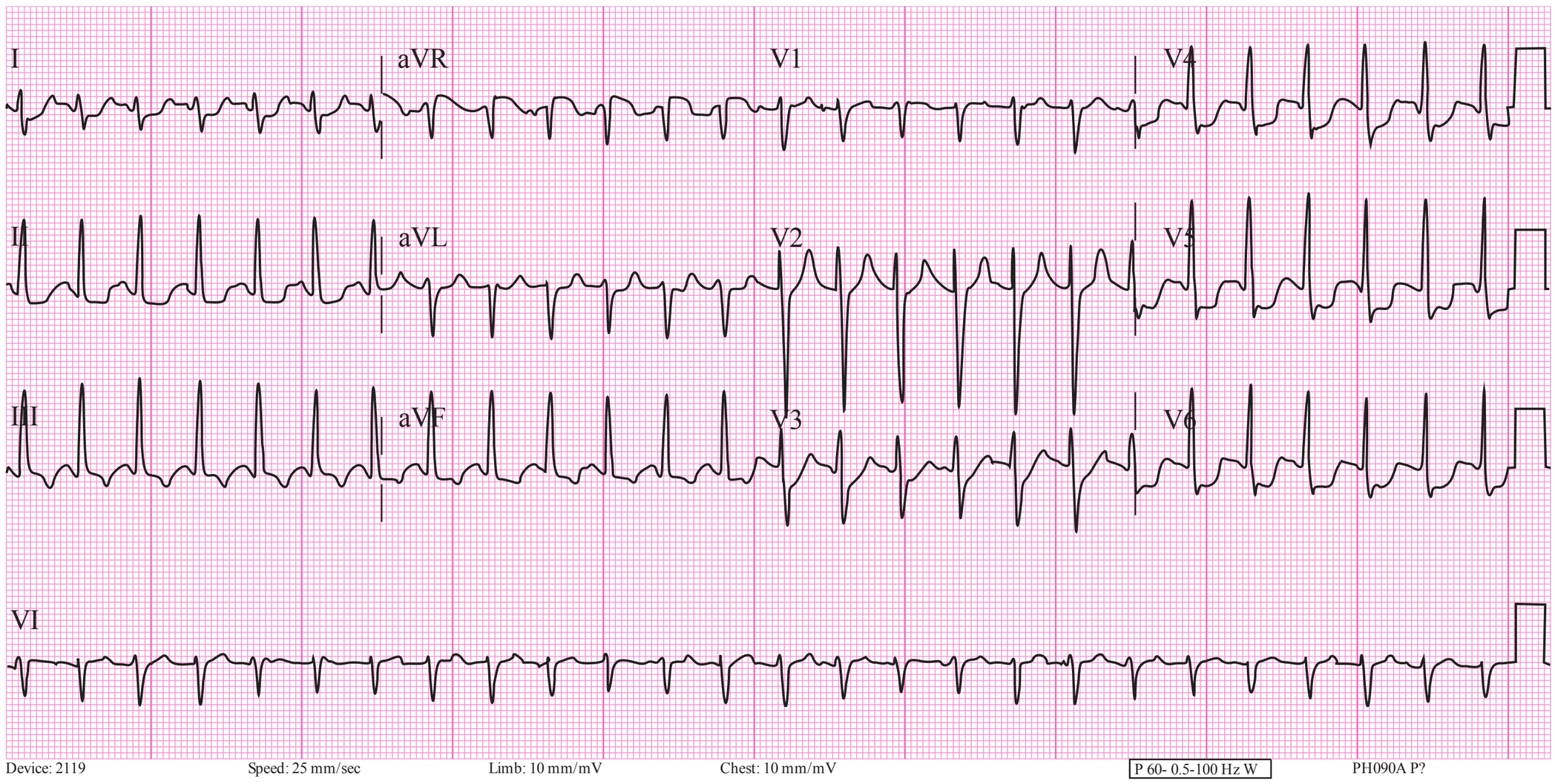


FIGURE 17.13 ■ **Treated Cocaine Cardiotoxicity.** The 12-lead EKG of the same patient in Fig. 17.12, 68 minutes later after aggressive treatment with sodium bicarbonate to a serum pH of 7.26. (Photo contributors: Thomas Babcock, MD and Laurie Lawrence, MD.)

Pearls

1. The use of β-blockers is contraindicated in the management of cocaine-associated chest pain and myocardial ischemia due to the potential for vasospasm and hypertensive crisis ("unopposed α-effect").
2. Although the risk of myocardial infarction is greatest immediately after use, myocardial ischemia may occur up to 6 weeks after last use.
3. Cocaethylene may form in vivo after the use of cocaine and ethanol. Cocaethylene is more cardiotoxic than cocaine and has a longer half-life.
4. The rupture of a cocaine packet in a body-packer may result in fatal toxicity. Emergent surgical intervention may be considered for immediate removal of the packets.



FIGURE 17.14 ■ **Cocaine-Induced Rhabdomyolysis.** Rhabdomyolysis is a common clinical finding in patients with severe cocaine poisoning. (Photo contributor: Mohamud Daya, MD.)

ANTICHOLINERGIC (ANTIMUSCARINIC) TOXIDROME

Clinical Summary

The anticholinergic toxidrome is well described by the mnemonic: hot as a hare, blind as a bat, mad as a hatter, red as a beet, and dry as a bone. As the etiology reflects central and peripheral muscarinic receptor blockade, it is more accurately termed an antimuscarinic toxidrome. A centrally mediated delirium may occur, which is typically not violent but is associated with mumbling speech and persistent "picking" behaviors. Other manifestations include hyperthermia, mydriasis, dry mucus membranes and axillae, tachycardia, decreased gastrointestinal (GI) motility, and urinary retention.

Many xenobiotics are antimuscarinic. One of the more common is diphenhydramine. Tricyclic antidepressants, (TCAs) phenothiazines, cyclobenzaprine, carbamazepine, atropine, scopolamine, glycopyrrolate, and belladonna alkaloids all have antimuscarinic properties. Plants such as jimson weed contain belladonna alkaloids and may be used recreationally.

Management and Disposition

Initial assessments of the vital signs and the duration of the QRS on ECG are critical. Since many antimuscarinic xenobiotics are also sodium channel blockers, QRS interval should be monitored. Hyperthermia occasionally occurs and is treated with evaporative cooling. Most of these patients require only supportive care, with the administration of benzodiazepines for agitation. A Foley catheter may be needed for treatment of the urinary retention. Occasionally, physostigmine is used as a diagnostic reversal agent for antimuscarinic poisoning, but its risks versus benefits must be considered. The half-life of physostigmine is only about 20 minutes.

Pearls

1. The antihistamine diphenhydramine has sodium channel–blocking properties and may cause significant QRS widening.
2. Physostigmine crosses the blood-brain barrier, so an improvement in antimuscarinic delirium can be elicited with administration of the drug.
3. Glycopyrrolate is an antimuscarinic that does not cross the blood-brain barrier, so delirium does not occur.
4. Many features of anticholinergic toxidrome can mimic a sympathomimetic one—moist axillae on exam may help differentiate the two clinically as the anticholinergic toxidrome prevents sweating, while the sympathomimetic toxidrome promotes it.



FIGURE 17.16 ■ **Anticholinergic Delirium.** Anticholinergic delirium is manifested by agitation, confusion, and a "picking" behavior. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)



FIGURE 17.15 ■ **Anticholinergic Mydriasis.** Mydriasis and flushing are some of the characteristic findings of anticholinergic toxidrome. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)

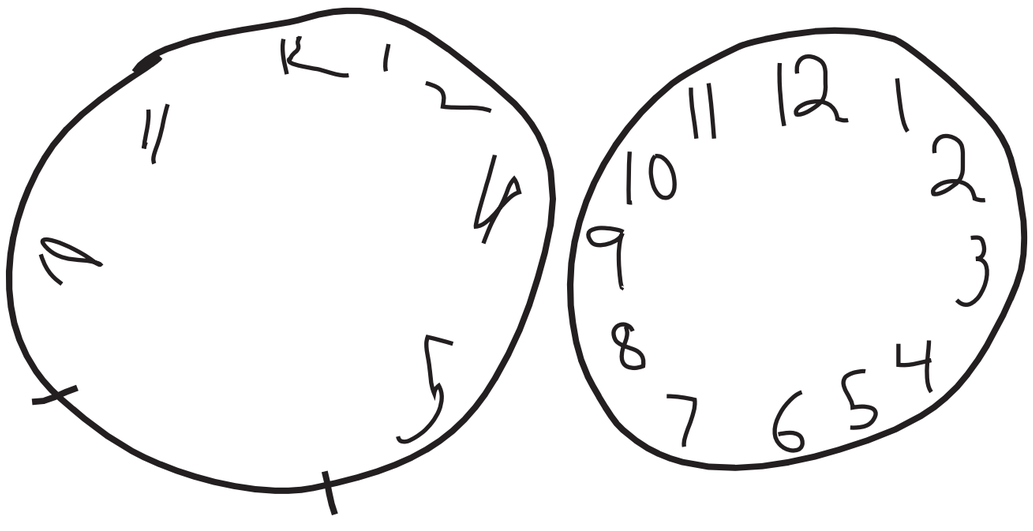


FIGURE 17.17 ■ **Anticholinergic Delirium.** Prior to treatment with physostigmine, a patient suffering acute anticholinergic delirium drew the clock on the left. Following physostigmine administration, the patient drew the clock on the right. (Photo contributor: Division of Medical Toxicology, University of California, San Diego.)

Clinical Summary

Acetylcholine (Ach) is a neurotransmitter of both the central and peripheral nervous systems and is the neurotransmitter for both muscarinic and nicotinic receptors. Inhibition of acetylcholinesterase increases Ach in the synapse. This results in a clinical syndrome producing both muscarinic and nicotinic effects. One mnemonic for the muscarinic effects is "DUMBBEL(L)S": diarrhea, urination, miosis, bronchorrhea, bradycardia, emesis, lacrimation, salivation. Nicotinic effects may include fasciculations, paralysis, and occasionally mydriasis and tachycardia.

The most common cholinergic poisoning occurs after exposure to anticholinesterase insecticides. These include organic phosphorus (OP) compounds and carbamates. OP compounds bind to acetylcholinesterase, and after a period of time, this bond "ages" and becomes permanent. Carbamates are reversible binders of acetylcholinesterase and tend not to cross the blood-brain barrier.

Management and Disposition

The initial management of patients with acute cholinergic poisoning includes decontamination of clothing and skin. The airway should be secured early in the resuscitation as most patients have difficulty oxygenating and ventilating due to bronchorrhea and muscle weakness. Succinylcholine should be avoided as a paralytic agent in cholinergic crisis because succinylcholine requires acetylcholinesterase for its metabolism. Atropine is the initial therapeutic agent and dosing is doubled until the pulmonary secretions are dried. Pralidoxime is administered as early as possible for those patients with acute OP poisoning but would not be of benefit to those patients who have known carbamate poisoning. Seizures should be aggressively treated with benzodiazepines.

Pearls

1. Patients developing central antimuscarinic toxicity during treatment with atropine who still require atropinization for secretion management may be treated with glycopyrrolate. Glycopyrrolate will not treat the central cholinergic effects as it does not cross the blood-brain barrier, but likewise will not cause central antimuscarinic toxicity.
2. The nicotinic clinical findings of cholinergic toxicity can be remembered by the days of the week: MTWtHF: mydriasis, tachycardia, weakness, hypertension, and fasciculations.
3. Nerve agents such as sarin, tabun, and soman are OP compounds and are readily absorbed through inhalational and dermal routes.
4. The pharmaceutical agents physostigmine, neostigmine, and pyridostigmine are carbamates; of these, only physostigmine crosses the blood-brain barrier.



FIGURE 17.18 ■ **Cholinergic Miosis.** Significant miosis seen in a patient with acute cholinergic poisoning. The patient had ingested a pesticide. (Photo contributor: Shannon Langston, MD.)



FIGURE 17.19 ■ **Cholinergic Toxidrome.** This patient is manifesting profound muscarinic effects from acute cholinergic poisoning, with intractable vomiting and diarrhea as prominent clinical features. (Photo contributor: Shannon Langston, MD.)



FIGURE 17.20 ■ **Atropine Therapy.** This picture demonstrates the use of several vials of atropine that were required to dry the secretions of a patient with severe acute cholinergic toxicity. Multiple repeat atropine doses are commonly needed to treat cholinergic poisoning. (Photo contributor: Shannon Langston, MD.)

Clinical Summary

Opium is derived from the poppy plant, Papaver somniferum. Opiates are naturally occurring drugs derived from opium and include morphine, codeine, and paregoric. The term opioid refers to drugs with opium-like activity. Heroin is a bitter-tasting white powder; however, most street-grade heroin varies in color from white to dark brown. Mexican "black tar" heroin may be sticky like roofing tar or hard like coal, and appears dark brown to black in color. Diversion and abuse of legal narcotic agents is a common practice that is resulting in increased number of opioid deaths. For example, time-released preparations can be abused by chewing the tablets, snorting crushed tablets, or dissolving and parenterally administering the tablets, bypassing the sustained release mechanism. Significant mucosal necrosis may occur from the repeated snorting of crushed opioid tablets.

The classic opioid toxidrome is a clinical triad of coma, respiratory depression, and miosis. However, opioid-related CNS depression can range from mild sedation to coma. Normal or dilated pupils may occur after overdose of meperidine or pentazocine, or in the setting of CNS hypoxia. Death is typically due to respiratory depression. NCPE is associated with the use of certain opioids, including heroin, methadone, and morphine.

Management and Disposition

Care of these patients focuses upon airway management and antidotal therapy. Whole-bowel irrigation has been advocated after ingestion of sustained release formulations, or in the setting of body-packing and body-stuffing. In the latter, abdominal x-rays are indicated to look for evidence of foreign bodies. Chest radiographs are indicated for signs and symptoms of NCPE. Rhabdomyolysis and cerebral hypoxia may occur after prolonged periods of respiratory and CNS depression. Naloxone is the antidote of choice for significant opioid toxicity. In administering naloxone, care should be taken not to precipitate acute opioid withdrawal. Naloxone drips are indicated with longer acting opioids to avoid rebound respiratory depression from unopposed effects when the initial naloxone dose wears off.



FIGURE 17.22 ■ **Black Tar Heroin.** Black tar heroin has a different appearance and texture than the South American and Asian heroin. Because it has a "gummier" texture, it is usually injected or smoked. Black tar heroin is associated with wound botulism. (Photo contributor: US Drug Enforcement Administration.)



FIGURE 17.21 ■ **Asian Heroin.** Asian heroin tends to be available in a powder form. (Photo contributor: US Drug Enforcement Administration.)

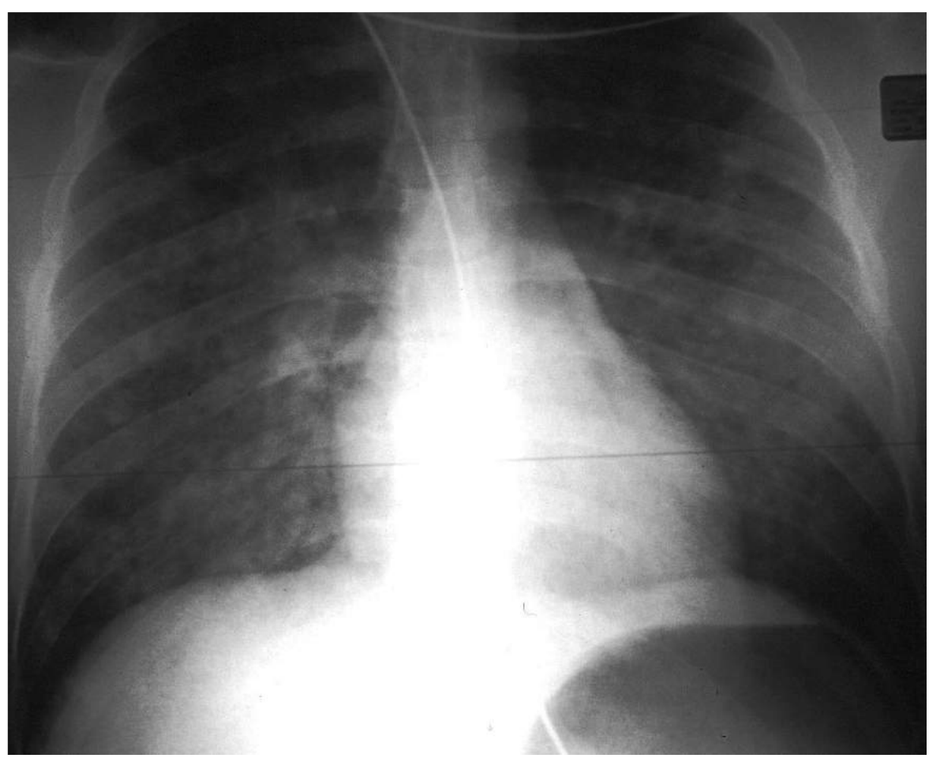


FIGURE 17.23 ■ **Heroin-Related Noncardiogenic Pulmonary Edema.** Noncardiogenic pulmonary edema may occur in the setting of opioid poisoning. The radiograph demonstrates the bilateral airspace opacities and the normal-sized cardiac silhouette. (Photo contributor: Division of Medical Toxicology, University of California, San Diego.)



FIGURE 17.24 ■ **Opioid-Induced Noncardiogenic Pulmonary Edema.** Copious frothy expectorant is flowing from the endotracheal tube in this patient with severe opioid-induced noncardiogenic pulmonary edema. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)

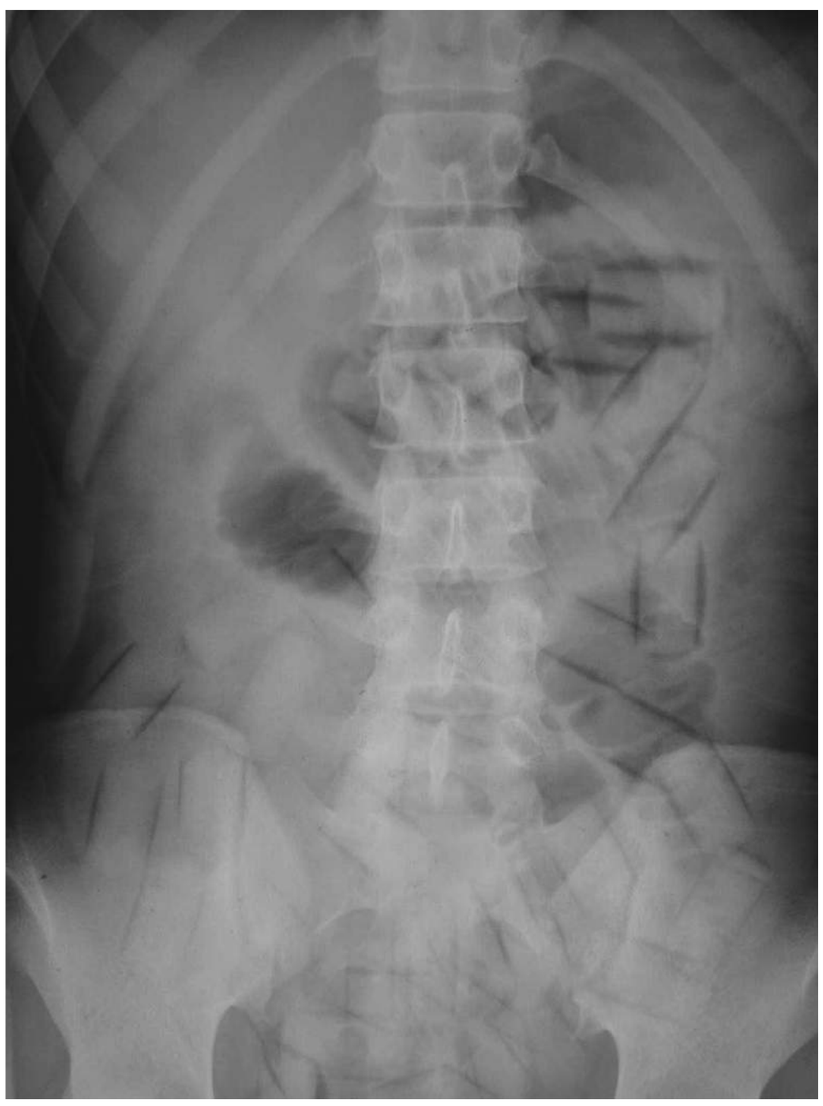


FIGURE 17.25 ■ **Heroin Body-Packing.** KUB radiograph of a "packer" demonstrating the presence of radiopaque foreign bodies. Rupture of a packet may result in severe opioid toxicity. (Photo contributor: Jason Chu, MD.)

Pearls

1. The presence of adulterants such as scopolamine or clenbuterol may mask or alter the appearance of the classic opioid toxidrome and may result in more significant toxicity than the primary drug.
2. Recurrent toxicity and life-threatening respiratory depression may occur following short-term reversal with naloxone administration, especially in body-stuffers or after ingestion of sustained release formulations.
3. The use of naloxone in the setting of tramadol toxicity is relatively contraindicated due to the occurrence of seizures.
4. The use of black tar heroin has been associated with wound botulism.
5. Poisoning from α_2 agonists like clonidine mimics acute opioid poisoning.



FIGURE 17.26 ■ **Piloerection.** Piloerection may be noted with acute opioid withdrawal. (Photo contributor: Division of Medical Toxicology, University of California, San Diego.)



FIGURE 17.27 ■ **Opioid Mucosal Necrosis.** In this patient, significant necrosis of the soft palate in seen. The necrosis occurred secondary to repeated opioid abuse by snorting crushed oxycodone tablets. (Photo contributor: Lawrence B. Stack, MD.)

ACETAMINOPHEN POISONING

Clinical Summary

Acetaminophen is a widely available analgesic and antipyretic agent. It is commonly found in combination with opioids, decongestants, antihistamines, and other over-the-counter and prescription products. Patients may complain of nausea and vomiting shortly after a toxic ingestion, but may also be asymptomatic. Signs and symptoms of acute liver injury typically occur within 36 hours after acute ingestion. Occasionally, patients present to the emergency department after developing evidence of hepatotoxicity, not realizing that the large ingestion of an acetaminophen-based product is the etiology.

In the overdose setting, acetaminophen exerts its toxic effects via a metabolite that is created via the P-450 enzyme system. The metabolite causes centrilobular necrosis of the liver which may lead to fulminant hepatic failure. Renal failure may also occur. Fatalities from hepatic failure usually occur 3 to 5 days after the ingestion. Treatment includes the administration of N-acetylcysteine (NAC), which can prevent acetaminophen-induced hepatotoxicity if initiated within 8 hours of the acute ingestion.

Management and Disposition

Activated charcoal may be considered in patients who present within 2 hours of acetaminophen overdose. A serum acetaminophen level drawn at 4 hours after a single acute ingestion can be plotted on the Rumack-Matthew nomogram to determine the need for treatment. If the serum level is at or above the treatment line, the patient should be treated with a standard course of oral or intravenously administered NAC. Patients who require administration of NAC should be admitted to the hospital.

Pearls

1. Acetaminophen is a common agent in many over-the-counter medications. Patients who overdose on these medications require routine checking of a serum acetaminophen level to identify those who may need treatment with NAC.
2. The formulation of oral NAC is available in a 20% solution. The 20% solution comprises 20 g of NAC per 100 mL of solution. For the average 70-kg adult, the initial oral loading dose of 140 mg/kg would be 9.8 g, or approximately 50 mL of the 20% solution.
3. To enhance palatability, oral NAC can be diluted into a beverage of choice and served in a cup with a lid and straw.
4. Massive ingestions of acetaminophen may result in an anion gap metabolic acidosis.



FIGURE 17.28 ■ **Acetaminophen Overdose.** Multiple acetaminophen-containing pills are seen in the vomit of an overdose patient. The patient had ingested the pills a few hours prior to presentation in a suicide attempt. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 17.29 ■ **Acute liver failure.** This patient developed hepatic failure with marked jaundice as a result of an intentional acetaminophen overdose. (Photo contributor: R. Jason Thurman, MD.)

SALICYLATE POISONING

Clinical Summary

Salicylates are a common cause of analgesic poisoning. Acute ingestions of large quantities of aspirin may have delayed absorption due to the formulation of the drug or the formation of bezoars. Poisoning may occur with chronic ingestions as well, particularly in older patients.

Early effects after ingestion include GI irritation which may lead to nausea and vomiting. Classically, salicylate-poisoned patients present with a mixed acid-base picture. Central stimulation of the respiratory drive results in a primary respiratory alkalosis. As a result of disrupted energy mechanics and decreased adenosine triphosphate (ATP) production, metabolic acidosis and lactate accumulation occur. The initial pH of the patient's serum may be acidemic or alkalemic depending on the predominant acid-base disorder at the time of blood sampling. Ketonuria may also be noted. Hyperthermia occurs due to the generation and release of heat secondary to uncoupling of oxidative phosphorylation. Coma and seizures indicate severe nervous system toxicity and are associated with poor outcomes. Increased capillary permeability may result in NCPE and cerebral edema.

Management and Disposition

Fluid resuscitation to replace volume depletion is paramount early in the presentation. Since salicylate is a weak acid, alkalinizing the serum to a pH between 7.45 and 7.55 range traps the salicylate in an ionized form, decreasing entry into the CNS. Similarly, urinary alkalinization prevents tubular reabsorption, thereby enhancing the renal elimination of the salicylic acid. Because of the underlying metabolic acidosis and bicarbonate-induced hypokalemia, potassium replacement is usually needed in order to alkalinize the urine. Hemodialysis should be considered for deterioration in the acid-base status of the patient, renal failure, NCPE, or cerebral edema. A rapidly rising serum salicylate concentration is another consideration for dialysis. Patients with chronic ingestions may meet clinical criteria for extracorporeal elimination even with serum salicylate levels in the 40 to 50 mg/dL range. Admission should be strongly considered for any toxic salicylate ingestion.

Pearls

1. Oil of wintergreen is typically 98% methylsalicylate (1400 mg/mL). One teaspoon (5 mL) provides the equivalent 7000 mg of salicylic acid.
2. If a patient with severe salicylism must be intubated, careful attention should be made to mimic the minute ventilation of the nonparalyzed patient. If a respiratory acidosis is allowed to occur, the patient will become severely acidemic which allows the salicylate to further distribute into the tissues and poison the mitochondria.



FIGURE 17.30 ■ **Aspirin Bezoar.** Pill bezoar found in the GI tract of a patient who ingested approximately 750 enteric-coated aspirin tablets. At the time of death, approximately 13 hours after ingestion, the serum salicylate level was 128 mg/dL. More than 300 partially digested pills remained in the GI tract on postmortem. (Photo contributor: Jared M. Orrock, MD.)

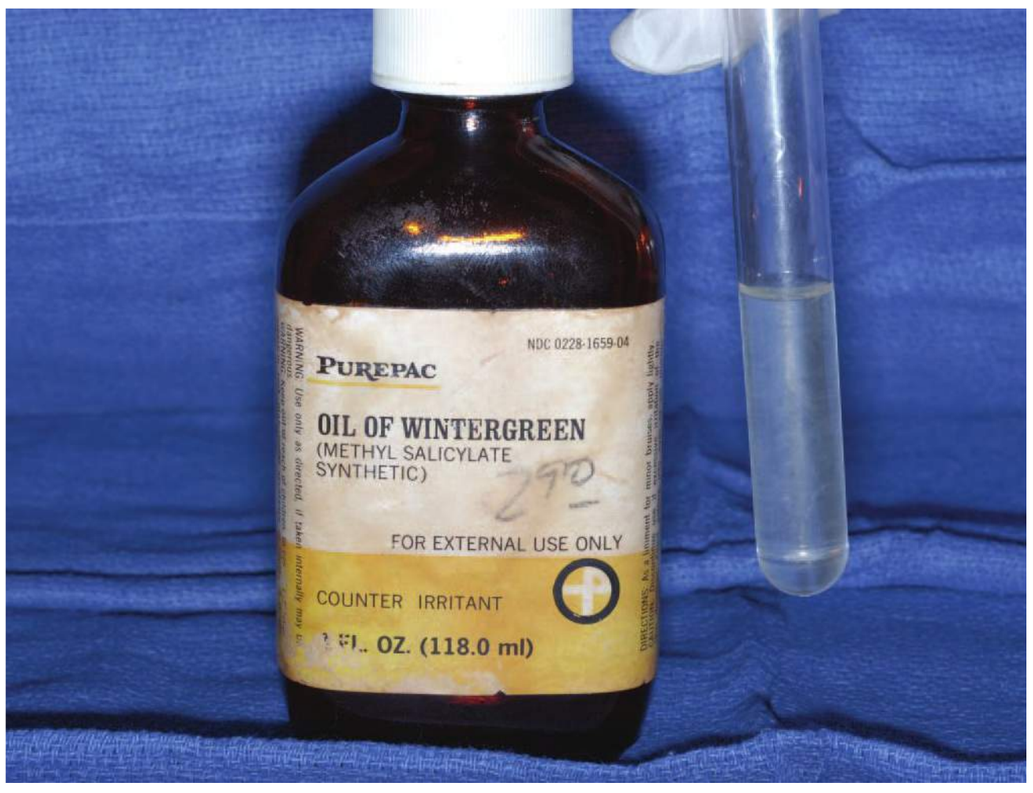


FIGURE 17.31 ■ **Oil of Wintergreen.** Severe salicylism may occur from ingestion of products that contain a high concentration of oil of wintergreen (methylsalicylate). This bottle of oil of wintergreen is a 98% solution, which contains the equivalent of 7000 mg of salicylate per teaspoon. (Photo contributor: R. Jason Thurman, MD.)

3. While acetazolamide administration results in alkalinization of the urine, the excretion of the bicarbonate into the urine comes at the expense of promoting acidemia, which could further drive the salicylate into the CNS and enhance toxicity.
4. The agent diflunisal will cause markedly elevated serum salicylate levels in the absence of other clinical or biochemical indicators of toxicity.

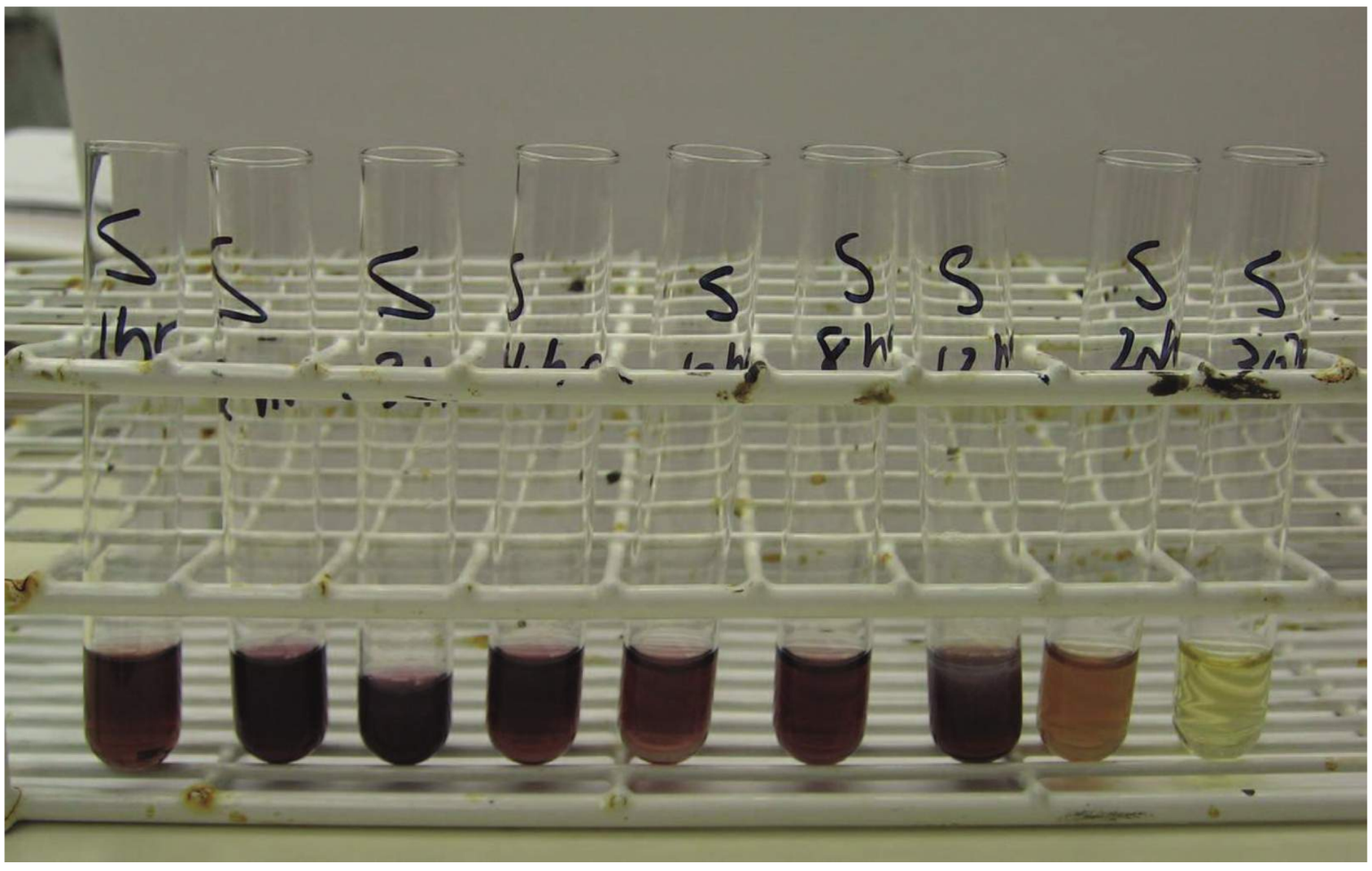


FIGURE 17.32 ■ **Trinder Reagent.** In the presence of salicylates, the addition of Trinder reagent to urine specimen will yield a purple color. This picture demonstrates the reaction to Trinder reagent from urine samples collected serially 1 hour to 30 hours after ingestion of 650 mg aspirin. (Photo contributor: Sheila Dawling, PhD.)

TOXIC ALCOHOL INGESTION

Clinical Summary

The commonly available toxic alcohols include ethylene glycol, methanol, and isopropanol. Ethylene glycol is a sweet-tasting liquid commonly found in antifreeze, as well as in brake fluid. Methanol is used in lock deicers, windshield wiper fluid, and industrial solvents. Isopropanol is commonly marketed as "rubbing" alcohol, although it is also found in nonstreaking glass and window cleaners, soaps, cosmetics, and antifreezes.

The parent toxic alcohols have the potential to intoxicate, but are not otherwise toxic to end organs. Sequential metabolism via alcohol dehydrogenase and aldehyde dehydrogenase produces the organic acids responsible for end-organ toxicity and metabolic acidosis. Ethylene glycol is metabolized to glycolic and oxalic acids; the former is responsible for the acidosis, while the latter is responsible for calcium oxalate deposition in the renal tubules and delayed acute renal failure (24-72 hours postingestion). Hypocalcemia may occur with severe intoxication. Cranial nerve palsies may also occur 5 to 20 days after ingestion. Although less intoxicating than ethanol, methanol is metabolized to formic acid, which is responsible for both acidosis and direct retinal toxicity. Patients often report blurred or dim vision ("snowstorm") prior to development of objective signs, including optic disc hyperemia, pupillary dilation, and poor accommodation. Pancreatitis and delayed basal ganglia lesions may occur. Isopropanol metabolism is limited to ketone formation and does not result in significant acidosis.

Management and Disposition

Decontamination options are limited, as activated charcoal does not bind alcohol and GI absorption is rapid. Emergency management is directed toward supportive care, diagnosis of the agent, and prevention of further metabolism. Methanol, ethylene glycol, and isopropanol levels may not be readily available. However, care should be taken in interpreting ancillary data, such as urinary fluorescence and the osmolar gap. Both ethanol and fomepizole competitively inhibit alcohol dehydrogenase. Both of these reduce metabolism of the parent compound, but do nothing for the toxic metabolites. Administration of folate (methanol) or pyridoxine and thiamine (ethylene glycol) may inhibit organic acid production or increase degradation by shunting metabolism to alternate pathways. Hemodialysis is indicated for signs of end-organ toxicity (eg, anion gap acidosis, renal failure, mental status changes) and should be considered for elevated toxic alcohol levels.

Pearls

1. Provided adequate metabolism has occurred, isopropanol ingestion will demonstrate the presence of large amounts of ketones on a urine dipstick assay.
2. Only a few sips of concentrated methanol or ethylene glycol are required to produce toxicity in a toddler;

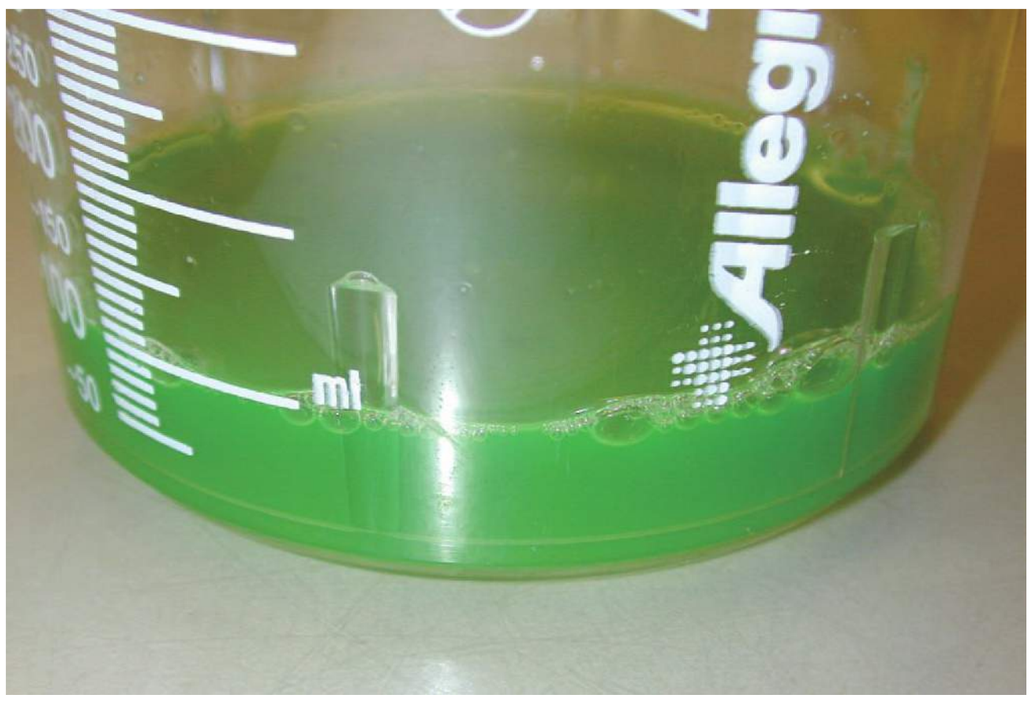


FIGURE 17.33 ■ **Antifreeze.** Addition of fluorescein to antifreeze gives colorless ethylene glycol its green appearance. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)

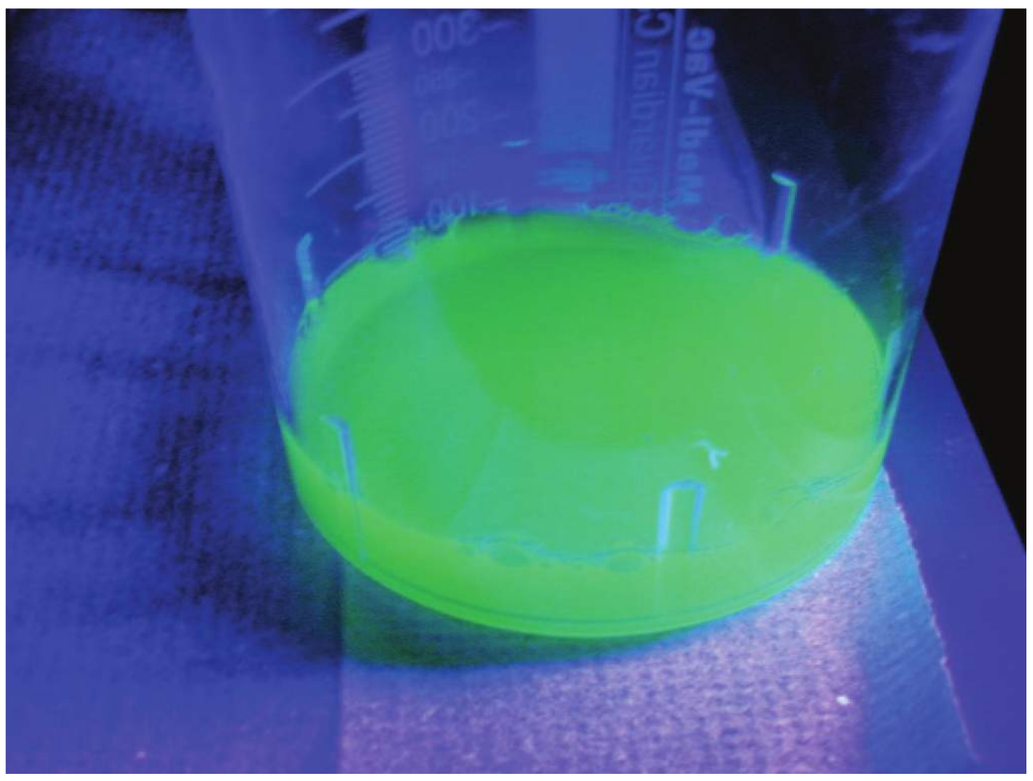


FIGURE 17.34 ■ **Antifreeze Fluorescence.** Application of a black light to antifreeze will demonstrate the fluorescence in body fluids, provided fluorescein has been added. This sample was obtained from the emesis of an overdose patient. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)

these ingestions should be viewed as a "one pill can kill" exposure.

3. Coingestion of ethanol may delay development of eventual toxicity due to preferential blockade of alcohol dehydrogenase.
4. Both ethanol and fomepizole inhibit metabolism of the parent compounds.
5. Due to the occurrence of hypocalcemia in ethylene glycol poisoning, careful monitoring of ionized calcium is critical when using sodium bicarbonate in these patients.

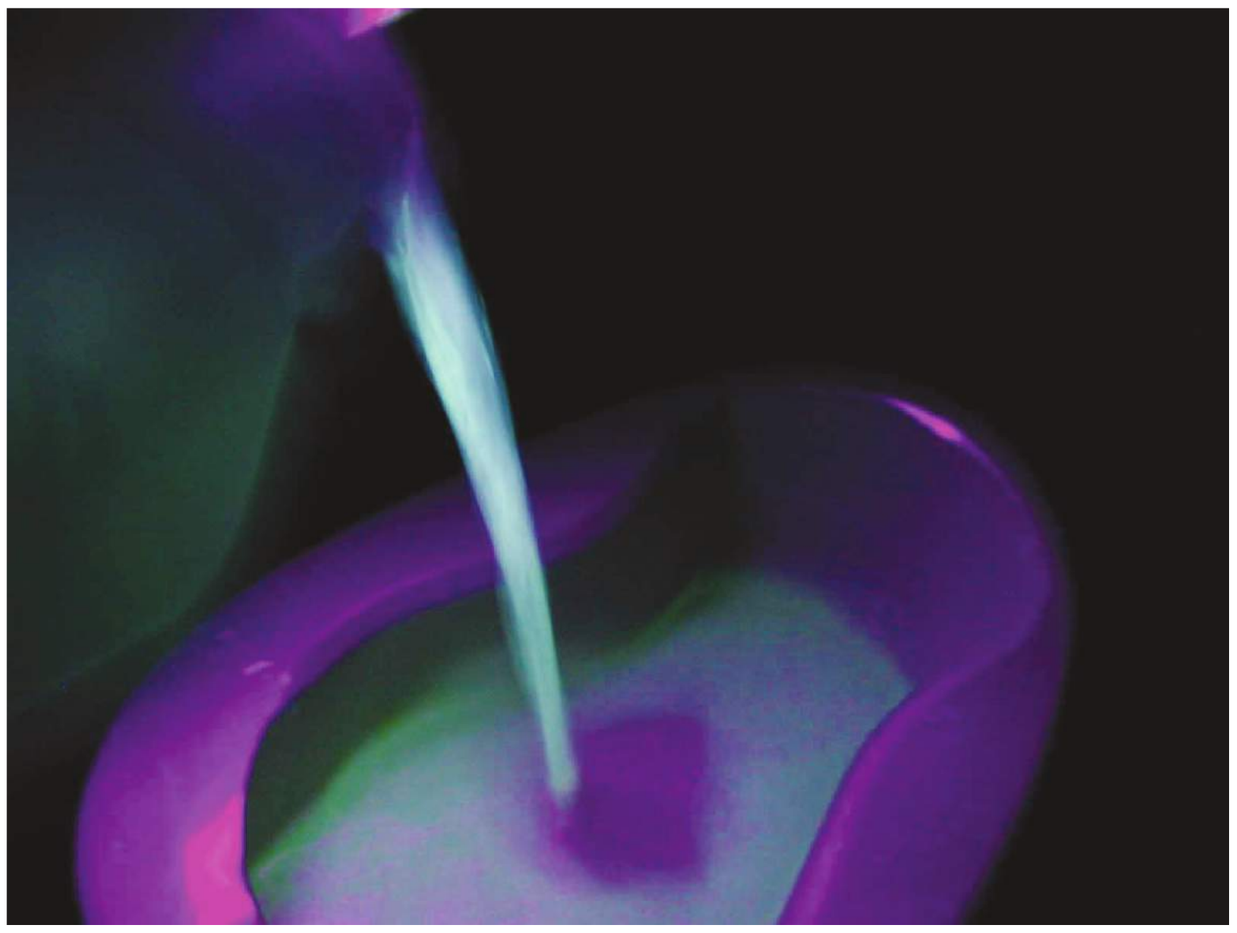


FIGURE 17.35 ■ **Urine Fluorescence.** Under black light, the urine of this ethylene glycol overdose patient shows a bright fluorescence. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)

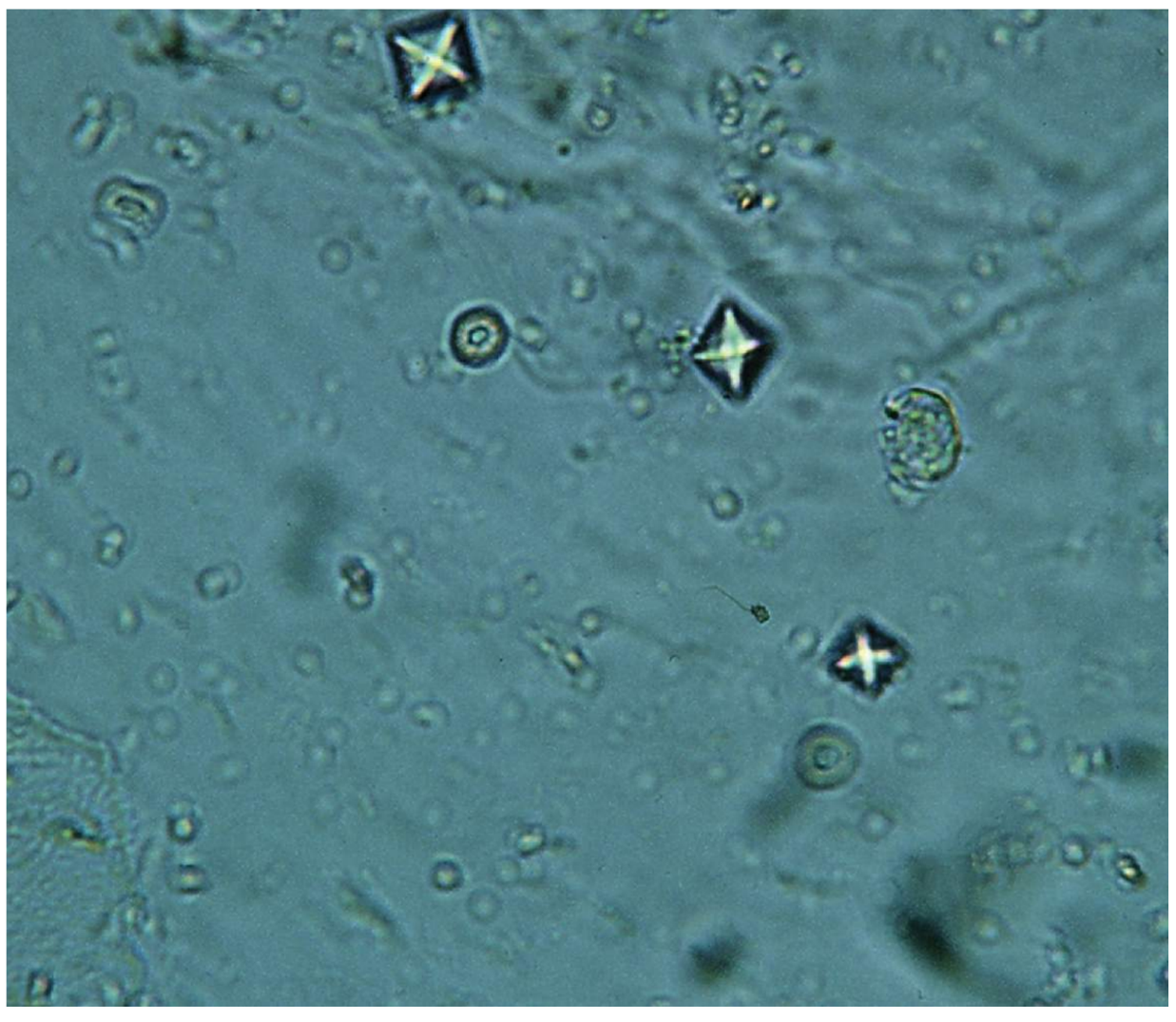


FIGURE 17.36 ■ **Calcium Oxalate Crystals.** Calcium oxalate crystals may be seen in the urine of the patient who ingested ethylene glycol and metabolized the parent compound to create oxalic acid. (Reproduced with permission from Strasinger SK, Di Lorenzo MS. Urinalysis and Body Fluids. 4th ed. Philadelphia, PA: F.A. Davis Company; 2001.)

TRICYCLIC ANTIDEPRESSANT POISONING

Clinical Summary

Despite the advent of the newer antidepressant agents (eg, serotonin-specific reuptake inhibitors), tricyclic antidepressant (TCA) toxicity remains a significant cause of poisoning morbidity and mortality in the United States. TCAs exert effects on multiple systems, including voltage-gated sodium channels, potassium channels, H_1-histamine receptors, D_2-dopamine receptors, M_1-muscarinic receptors, α_1-adrenergic receptors, and the GABA-A receptor complex.

TCA toxicity is related to pharmacological effects on the myocardium, CNS, and vasculature. M_1-muscarinic receptor blockade may result in an anticholinergic toxidrome. CNS toxicity may range from sedation to coma. Seizures, agitation, and delirium may occur. Inhibition of voltage-gated sodium channels results in characteristic widening of the QRS complex. A limb-lead QRS duration greater than 120 ms is associated with an increased incidence of seizures, while a limb-lead QRS duration greater than 160 ms is associated with an increased incidence of ventricular dysrhythmias. Similarly, in adults, a terminal R wave in lead aVR greater than or equal to 3 mm is associated with increased risk of seizure or dysrhythmias.

Management and Disposition

Signs and symptoms of significant overdose typically occur early. All patients presenting after TCA overdose should receive continuous cardiac monitoring and an ECG. Aggressive airway management may be indicated. No specific antidote exists for TCA poisoning. Benzodiazepines are the agent of choice for seizures. Management of QRS widening involves intravenous administration of sodium bicarbonate; controversy exists regarding optimal method of administration (intermittent dosing versus continuous infusion). Symptomatic patients should be admitted to the intensive care unit (ICU) due to the potential for rapid deterioration.

Pearls

1. In one study, half of all patients presenting to emergency department with trivial signs of poisoning had catastrophic deterioration within 1 hour.
2. Flumazenil and physostigmine are both contraindicated in the management of patients with ECG evidence of TCA poisoning.
3. Other xenobiotics that may cross-react with the TCA immunoassay on the urine drug screen include diphenhydramine, carbamazepine, cyclobenzaprine, and quetiapine.

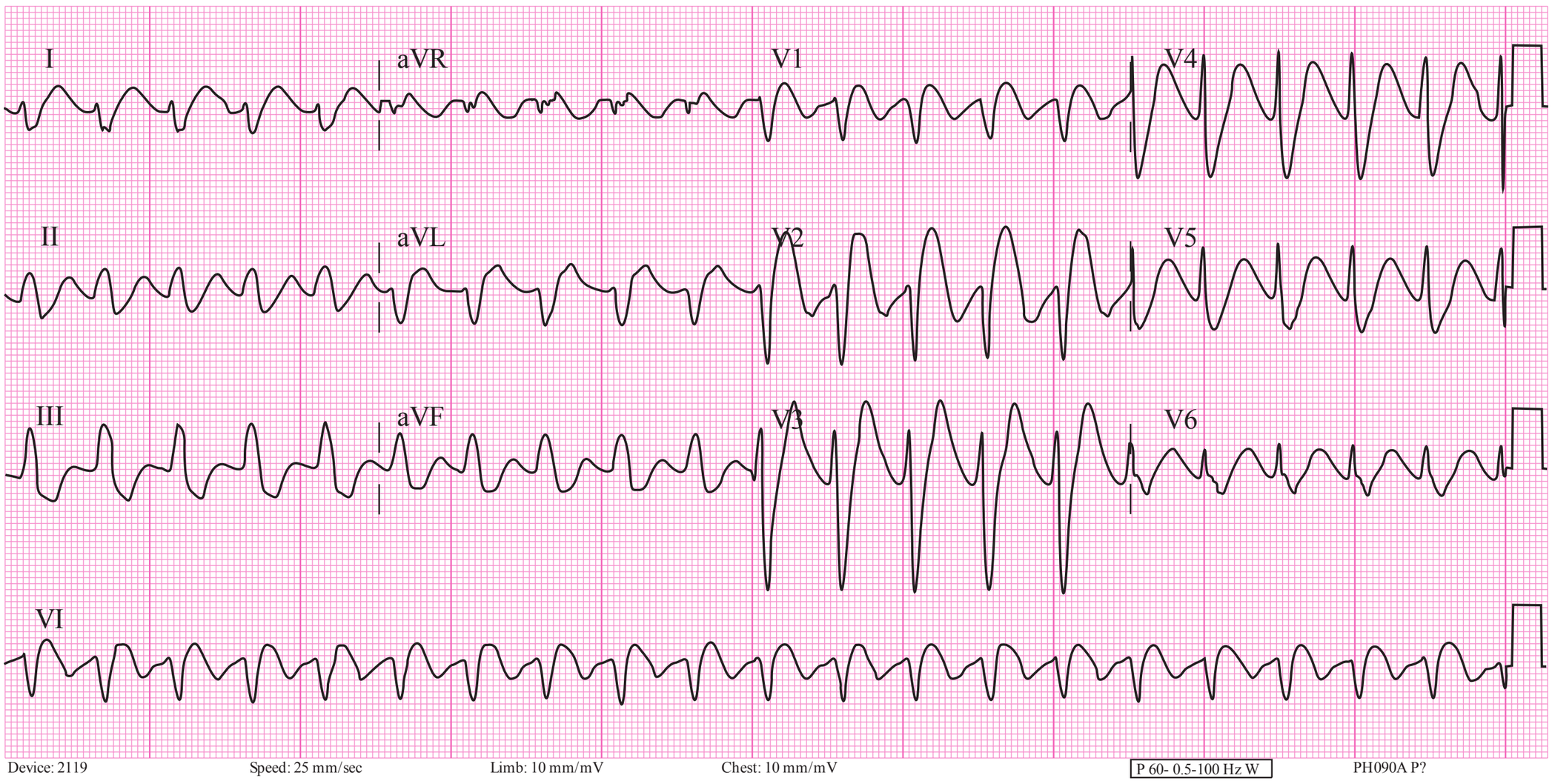


FIGURE 17.37 ■ **TCA Cardiotoxicity.** A 12-lead EKG of a patient who ingested a massive quantity of amitriptyline, demonstrating QRS widening. The patient presented awake and alert, but rapidly became obtunded. (Photo contributor: Thomas Babcock, MD and Clay Smith, MD.)

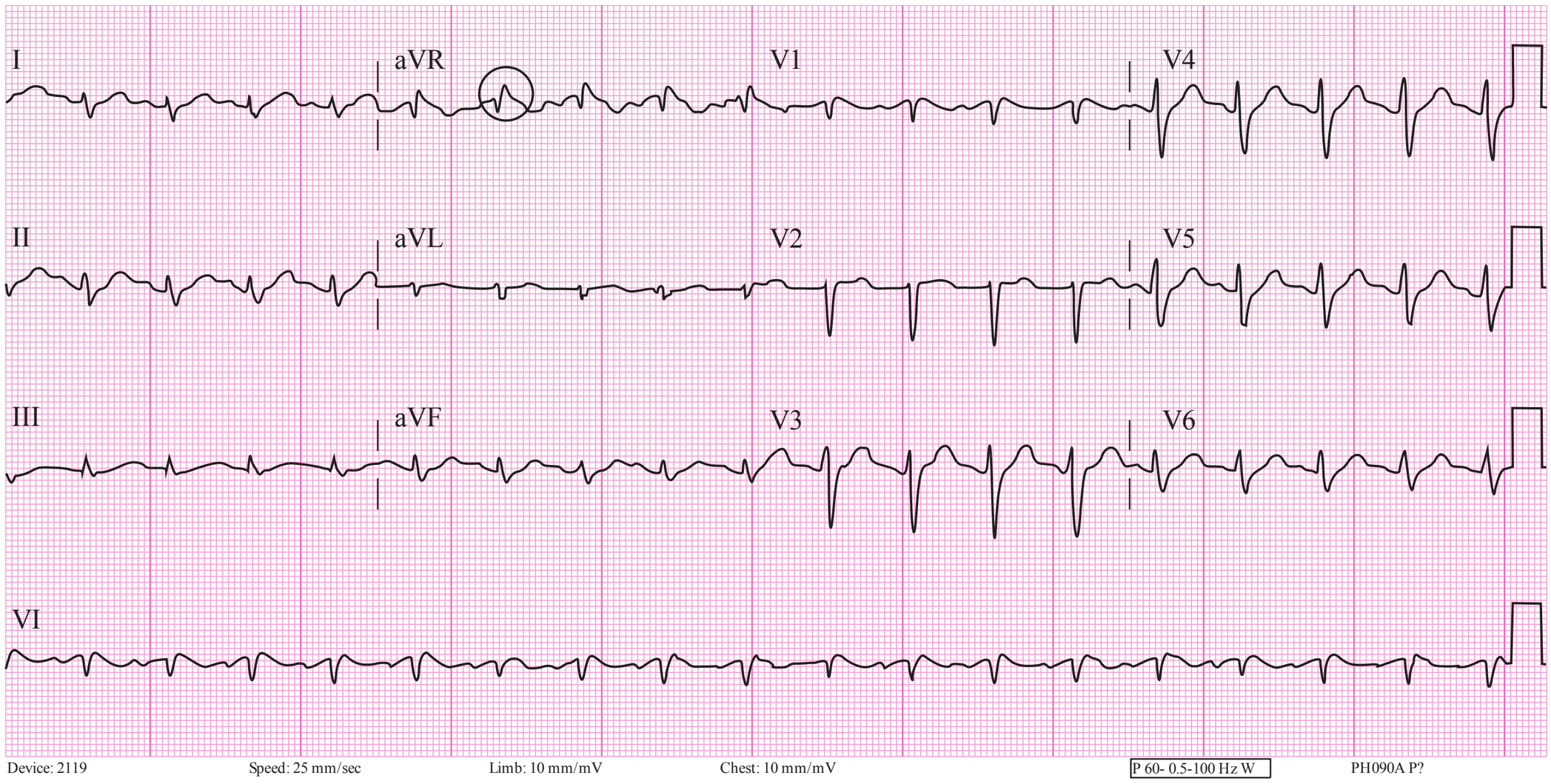


FIGURE 17.38 ■ **TCA Cardiotoxicity—Treated.** Repeat 12-lead EKG of the patient from Fig. 17.37 approximately 2 hours and 45 minutes after the first EKG. A total of 12 amperes of sodium bicarbonate had been administered intravenously. This EKG demonstrates the terminal R wave changes in aVR associated with sodium channel–blocking effects (circle). (Photo contributors: Thomas Babcock, MD, and Clay Smith, MD.)

POISONING BY β-BLOCKER AND CALCIUM CHANNEL BLOCKER AGENTS

Clinical Summary

β-Blockers and calcium channel blockers have various clinical indications, including the management of hypertension, myocardial infarction, and cardiac dysrhythmias, as well as the treatment of noncardiovascular conditions (eg, glaucoma, thyrotoxicosis, migraine headache prophylaxis). β-Blocking agents may be selective for B_1-adrenergic receptors, or nonselective. With therapeutic use, the commonly available calcium channel blockers are selective for the membrane-bound L-type calcium channel. Inhibition of this channel prevents influx of extracellular calcium.

Toxicity presents as an exaggeration of clinical effects, with significant toxicity manifesting predominantly as bradycardia and hypotension. β-Blocker toxicity may result in hypoglycemia, especially in children. Certain agents, such as propranolol, are associated with CNS toxicity (including seizures and CNS depression) and fast sodium channel blockade (analogous to TCA toxicity). Calcium channel blocker toxicity is associated with hyperglycemia, believed to be secondary to impaired insulin release (a calcium-dependent process) and impaired peripheral utilization.

Management and Disposition

The use of aggressive GI decontamination (eg, whole-bowel irrigation) has been advocated for sustained release preparations. In addition to standard Advanced Cardiac Life Support (ACLS) measures, glucagon has been used as a specific antidote for β-blocker toxicity. No treatment has been universally successful in the management of severe calcium channel blocker toxicity. Calcium, glucagon, high-dose insulin euglycemia, and intravenous lipid emulsion have all been tried with variable success. Management of these patients should involve early consultation with a poison control center or toxicologist.

Pearls

1. Topical β-adrenergic blocker administration (eg, for glaucoma) can result in significant systemic toxicity.
2. Development of toxicity may be appreciably delayed after ingestion of sustained-release formulations.
3. The presence of hyperglycemia versus hypoglycemia may help differentiate calcium channel blocker poisoning from β-blocker poisoning, respectively.
4. Due to the potential for significant local tissue toxicity, calcium chloride therapy should optimally be administered through a central venous catheter.

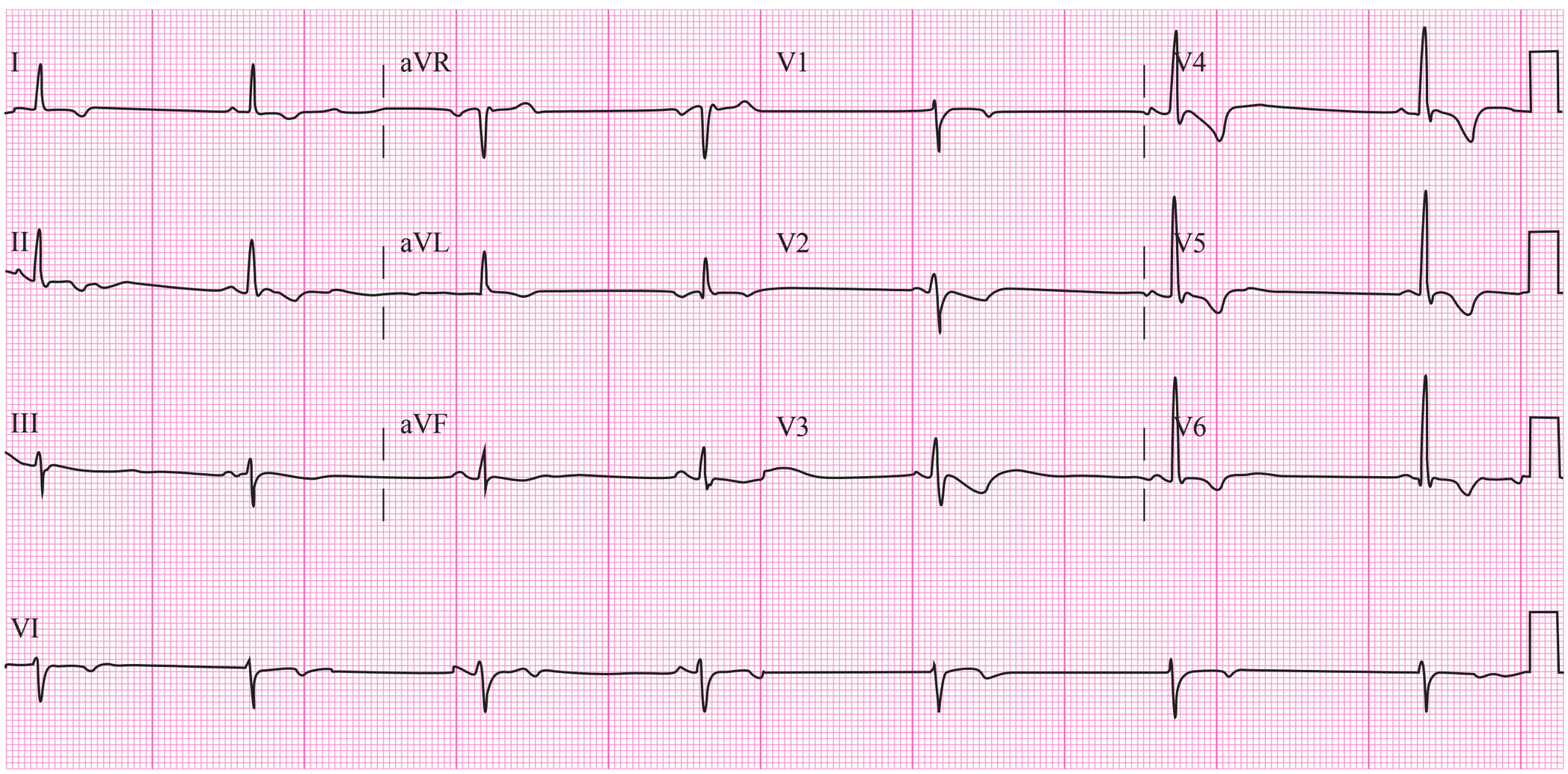


FIGURE 17.39 ■ **β-Blocker Overdose.** Atenolol poisoning resulting in severe bradycardia. (Photo contributor: Saralyn R. Williams, MD.)

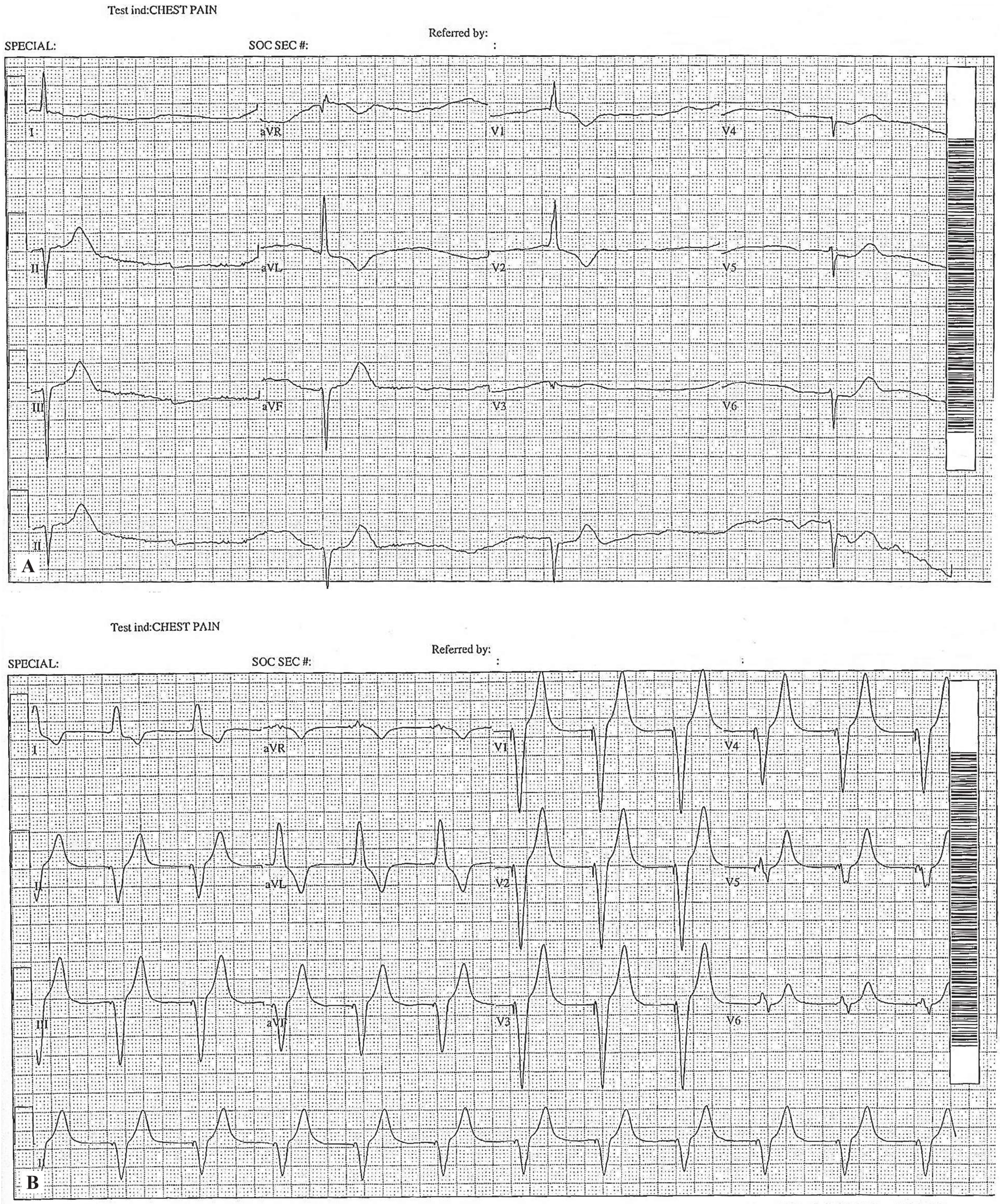


FIGURE 17.40 ■ **Calcium Channel Blocker Overdose.** (**A**) Verapamil poisoning causes profound negative inotropy and chronotropy. The 12-lead EKG demonstrates the bradycardia that may occur. (**B**) The same patient after transvenous pacing was initiated. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)

INHALANT ABUSE

Clinical Summary

Inhalant abuse, the intentional inhalation of vapors for the purpose of becoming "high," is more common among adolescents. Sniffing refers to the inhalation of the agent directly from a container, such as model airplane glue. Huffing involves placing solvent on some type of fabric and inhaling the vapors from the fabric. Bagging is the name given to the technique of spraying the solvent into a bag and then rebreathing from the bag. Occasionally the bag is placed over the head, potentially resulting in asphyxiant death. Inhalants are rapidly absorbed via the lungs and readily cross the blood-brain barrier. Initial effects include euphoria and occasional hallucinations. CNS depression may occur. Acute cardiotoxicity may also occur and is thought to be the cause of "sudden sniffing death." The cause of death is thought to be due to increased myocardial sensitization that promotes dysrhythmogenesis in the setting of a catecholamine surge. A defatting dermatitis may be evidence on the hands due to chronic exposure to solvents. Chronic effects from inhalant abuse include leukoencephalopathy, cardiomyopathy, cerebellar degeneration, and neuropathy.

Management and Disposition

Clues to the diagnosis of inhalant abuse are the presence of spray paint on the fingers or the face. Due to the increased solvent content in metallic-colored paints, gold and silver spray paint are particularly popular. Cardiac dysrhythmias are associated with a poor prognosis. Current recommendations suggest the use of β-blockers to treat ventricular dysrhythmias. Consider electrolyte abnormalities and acid-base status, particularly with toluene-based products. Benzodiazepines may be used for treatment of agitation.

Pearls

1. Chronic abuse of nitrous oxide (N_2O) may result in a megaloblastic anemia and distal axonal sensorimotor neuropathy that may be a result of irreversible oxidation of cobalamin (vitamin B_{12}). In addition to being used medicinally as "laughing gas," nitrous is abused from "whippets," which are the cartridges of compressed air used for whipping cream canisters.
2. Chronic abuse of toluene may result in potassium wasting renal tubular acidosis. Some of the carburetor cleaners that contain toluene also contain methanol.
3. Amyl, butyl, and isobutyl nitrites are strong oxidizers and may produce methemoglobinemia. These may be sold as "poppers."
4. Carbon monoxide poisoning may occur after methylene chloride inhalation.

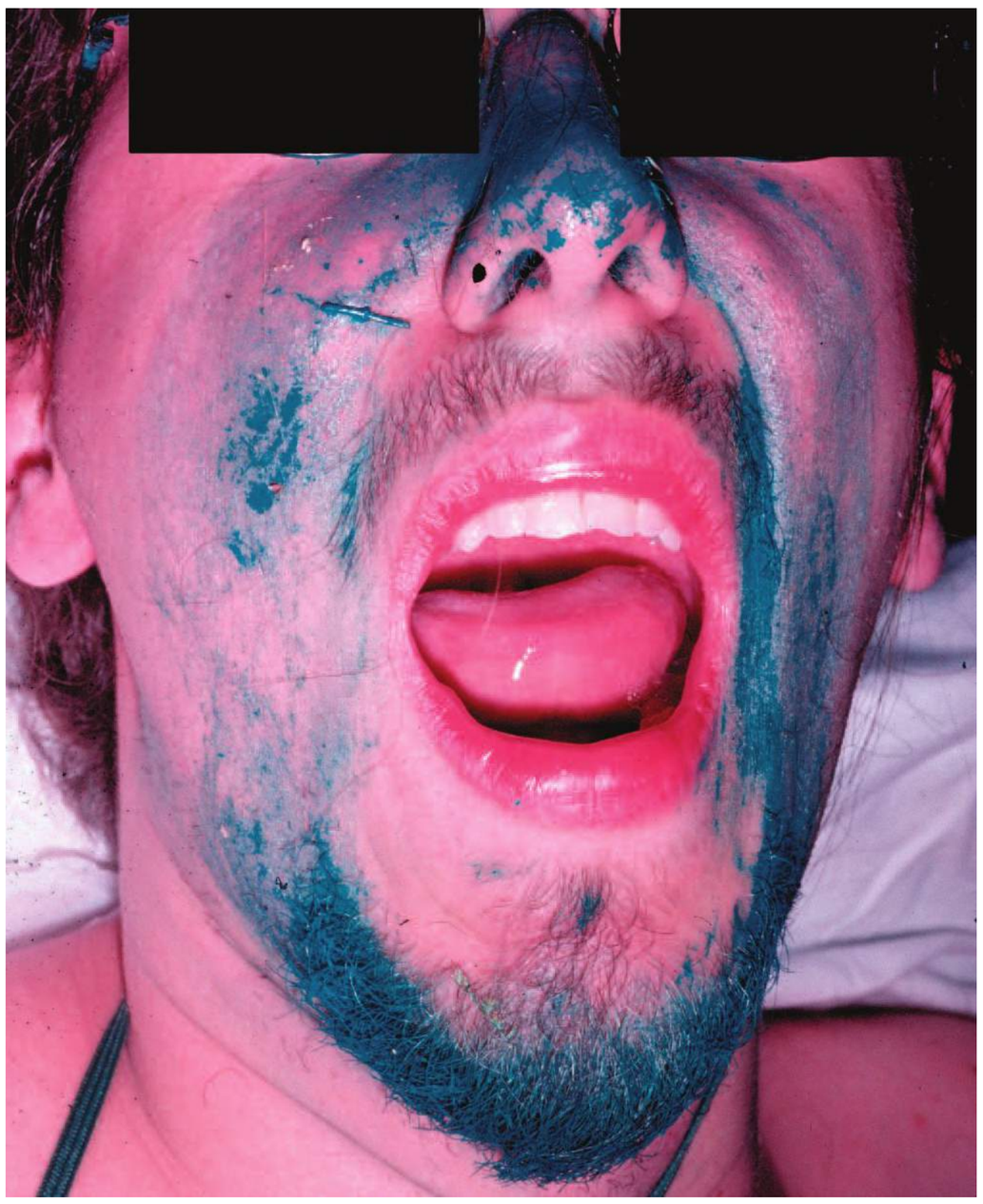


FIGURE 17.41 ■ **"Huffing."** Patients who huff spray paints may present with the paint on their face and hands. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 17.42 ■ **"Huffing."** The hand of the patient in Fig. 17.41. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 17.44 ■ **"Metallic Paint Abuse."** Gold and silver metallic paints are particularly popular for inhalant abuse due to the increased solvent content in the paint and a greater "high." (Photo contributor: R. Jason Thurman, MD.)



FIGURE 17.43 ■ **"Bagging."** Silver paint lining the perioral area in a patient abusing the paint by "bagging." (Photo contributor: R. Jason Thurman, MD.)



FIGURE 17.45 ■ **Motor Neuropathy of the Hand.** The hands of a chronic huffer demonstrate the muscle wasting in the left hand in addition to a mild defatting dermatitis on the palm of the hand. (Photo contributor: Saralyn R. Williams, MD.)

METHEMOGLOBINEMIA

Clinical Summary

Methemoglobin occurs when the iron atom in deoxyhemoglobin loses an electron, resulting in a ferric (Fe^{3+}) ion instead of the usual ferrous (Fe^{2+}) state. Ferric iron can no longer bind to oxygen, thereby reducing the oxygen-carrying capacity of hemoglobin. The presence of methemoglobin also shifts the oxygen hemoglobin dissociation curve to the left resulting in decreased release of oxygen to tissues. Infants are more susceptible to the development of methemoglobinemia due to reduced enzyme activity. Illnesses in infants such as diarrhea, dehydration, and acidosis may induce methemoglobin due to oxidant stress.

The most common causes of methemoglobin are acquired rather than congenital. Common pharmaceutical agents that cause methemoglobin include sulfonamides, dapsone, phenazopyridine, chloroquine, benzocaine, prilocaine, and more rarely lidocaine. Nitrites, which are used in the older cyanide antidote kit, induce methemoglobin.

Clues to the diagnosis include the patient who appears cyanotic and does not improve with the administration of oxygen. The pulse oximeter reading will drop to the mid 80% range but does not correlate with the percent of methemoglobin. The blood may appear chocolate in color and does not become red with exposure to oxygen. The arterial blood gas will demonstrate a normal partial pressure of oxygen with a resulting normal calculated arterial saturation. Methemoglobin may be measured via a co-oximeter using either arterial or venous heparinized blood.

Management and Disposition

Any patient who appears cyanotic should initially be treated with administration of supplemental oxygen and advanced airway management as appropriate. In general, any patient who is symptomatic from methemoglobinemia or has a level exceeding 25% to 30% should be treated with methylene blue. Methylene blue is available in a 1% solution and is administered as a 1 to 2 mg/kg dose intravenously over 5 minutes. This may be repeated if there is no initial response in 20 to 30 minutes. Patients who have methemoglobinemia from dapsone or aniline dyes may have recurrence and require additional dosing of methylene blue.

Pearls

1. High doses of methylene blue (5-7 mg/kg) may cause paradoxical methemoglobinemia and hemolysis.
2. The intravenous administration of methylene blue may interfere with the reading of the pulse oximeter and cause the reading to decrease transiently.
3. Methylene blue accelerates the ability of nicotinamide adenine dinucleotide phosphate (NADPH) methemoglobin reductase to reduce the ferric iron of methemoglobin back to a ferrous iron.
4. Methylene blue may not reverse methemoglobin in a patient with G6PD deficiency due to the absence of NADPH and may increase the risk of hemolysis.
5. Methylene blue has monoamine oxidase inhibitor activity; thus, use in patients who are on serotonin reuptake inhibitors may result in a serotonin syndrome.



FIGURE 17.46 ■ **Methemoglobinemia—Cyanosis.** Methemoglobinemia resulted in the cyanotic appearance of this pediatric patient as noted on the hand on the left side of the image compared with the normal adult control on the right. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 17.47 ■ **Methemoglobinemia—"Chocolate Blood."** "Chocolate blood" from an arterial sample of a patient with methemoglobinemia (left) compared to the normal bright red arterial blood (right). (Photo contributor: Kevin J. Knoop, MD, MS.)

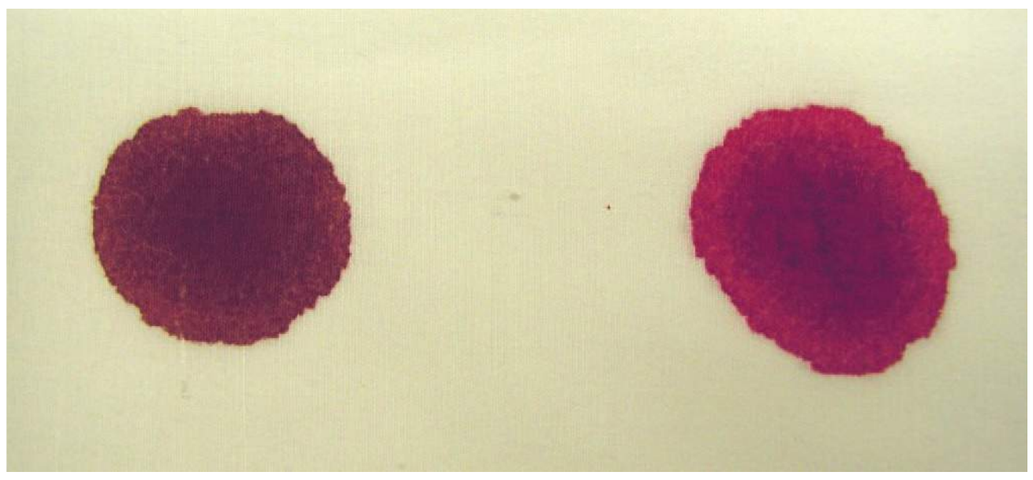


FIGURE 17.48 ■ **Methemoglobinemia—Exposure to Air.** The blood from a patient with methemoglobinemia on the left does not turn bright red upon exposure to air and will remain a "chocolate color" compared to the normal blood sample on the right. (Photo contributor: Louise Kao, MD.)



FIGURE 17.49 ■ **Administration of Methylene Blue.** The intravenous administration of methylene blue may be disconcerting to the patient who may not understand how a "blue dye" will help them. (Photo contributor: Lawrence B. Stack, MD.)

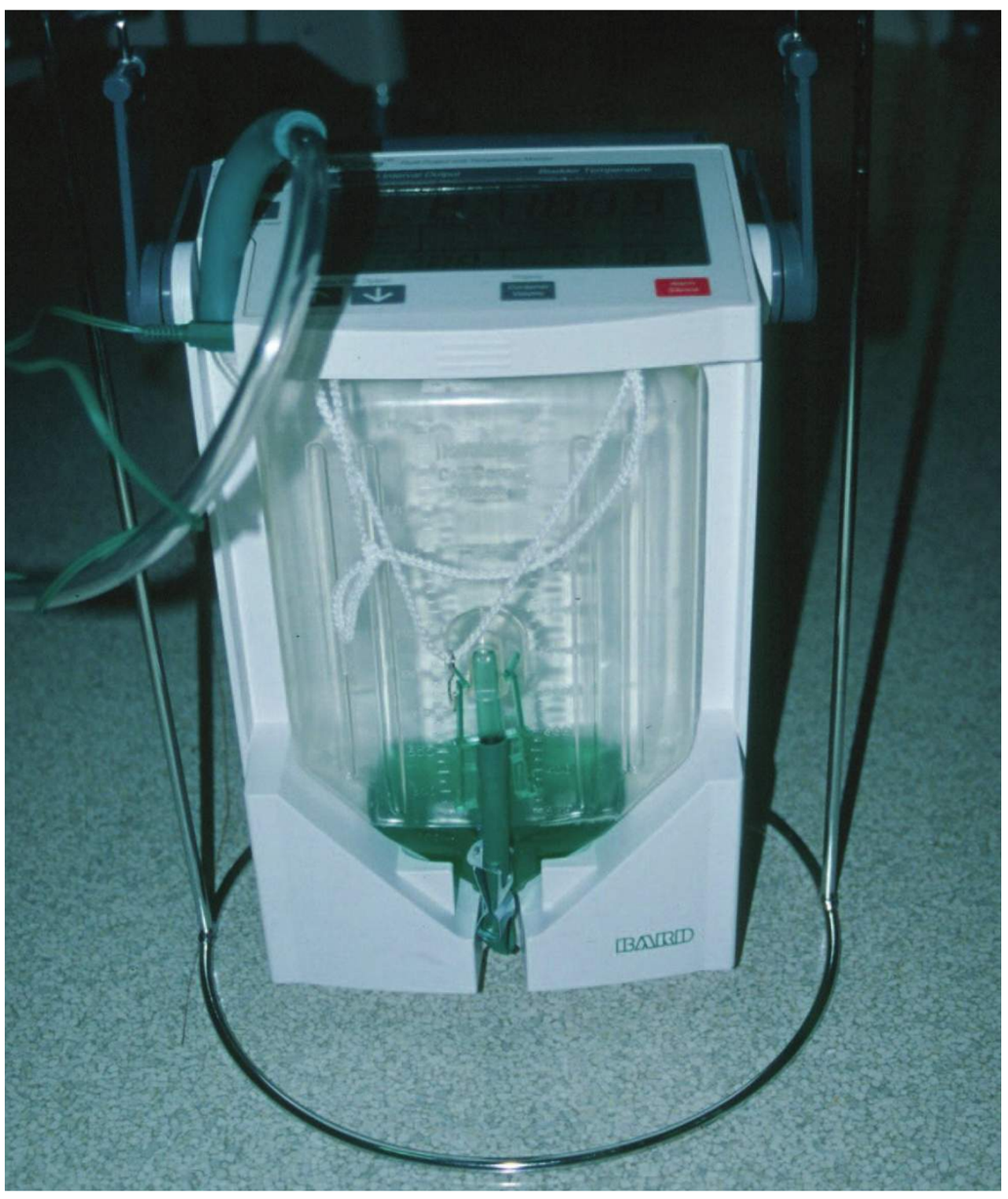


FIGURE 17.50 ■ **Methylene Blue Urine.** Methylene blue is excreted renally and gives a blue-green color to the urine. (Photo contributor: Division of Medical Toxicology, University of California, San Diego.)

CELLULAR ASPHYXIANTS

Clinical Summary

The cellular asphyxiants are a diverse group of substances including carbon monoxide (CO), cyanide, sodium azide, nitrites and other methemoglobin-producing oxidizing agents, and hydrogen sulfide, all of which interfere with the cellular utilization of oxygen. Depending on the substance, the interference may occur at the level of hemoglobin, the electron transport chain, or both. In contrast with the simple asphyxiants, ambient oxygen concentrations are not affected.

CO is a colorless and odorless gas generated from the incomplete combustion of carbonaceous compounds. The affinity of CO for hemoglobin is 250 times greater than that of oxygen. Binding of CO to hemoglobin shifts the oxyhemoglobin dissociation curve to the left, further impairing tissue oxygen delivery. Symptoms of acute poisoning may range from headache to ischemic chest pain, seizures, and CNS depression. Up to 40% of poisoned patients develop delayed neurologic sequelae (DNS); most cases of DNS are associated with an initial loss of consciousness.

Although a nonspecific enzyme inhibitor, cyanide interferes with oxidative phosphorylation. Sources of cyanide include industrial and household chemicals, plants, and structure fires. Clinical manifestations reflect dysfunction of oxygen-sensitive organs, including the CNS and cardiovascular systems. A cyanide toxidrome has been described, consisting of altered mental status, mydriasis, respiratory depression, hypotension, tachycardia, and metabolic (lactic) acidosis.

Management and Disposition

Immediate management focuses upon airway stabilization and antidotal therapy. Carboxyhemoglobin levels should be obtained in patients with suspected carbon monoxide poisoning. Pregnancy status should be determined in females presenting with suspected carbon monoxide poisoning. Blood cyanide levels are not typically available in the immediate care setting. While 100% oxygen is the accepted antidote for acute CO poisoning, controversy persists regarding the mode of administration (normobaric oxygen versus hyperbaric oxygen). Potential indications for hyperbaric oxygen include syncope, altered mental status (especially with evidence of cerebellar dysfunction), acidosis, and pregnancy. Therapy for cyanide poisoning includes intravenous administration of hydroxocobalamin. Hydroxocobalamin has the advantage of raising blood pressure, but also causes intense red skin discoloration and chromaturia.

Pearls

1. The decision to treat acute carbon monoxide poisoning is based upon history and physical examination, and not solely upon carbon monoxide level.
2. Maternal carboxyhemoglobin levels fail to accurately reflect fetal carboxyhemoglobin levels.

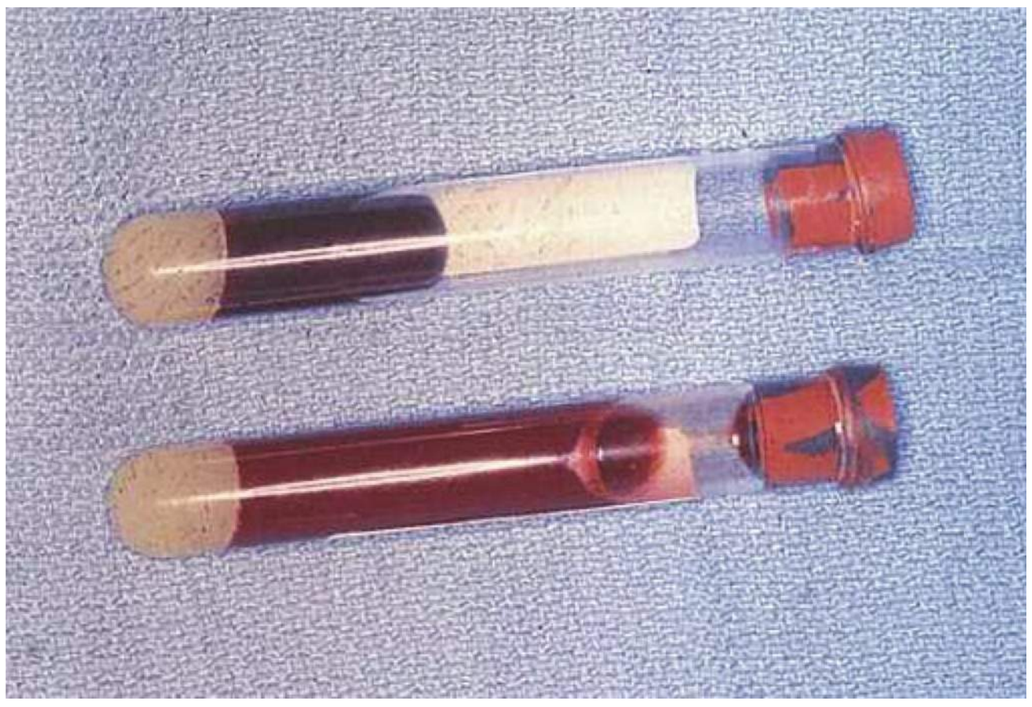


FIGURE 17.51 ■ **Carbon Monoxide Poisoning.** Venous blood samples with the bright red one (bottom sample) taken from a patient with acute carbon monoxide poisoning. The dark red venous blood (top sample) is a control sample from a patient with no carboxyhemoglobin. (Photo contributor: Daniel L. Savitt, MD.)



FIGURE 17.52 ■ **Skin Changes after Hydroxocobalamin.** After administration of therapeutic dosing of hydroxocobalamin, the skin of the patient may exhibit a bright red color and have chromaturia. (Reproduced with permission from Uhl W, Nolting A, Golor G, Rost KL, Kovar A. Safety of hydroxocobalamin in healthy volunteers in a randomized, placebo-controlled study. Clin Toxicol. 2006;44:17-28. Copyright © Informa Healthcare.)

3. Methylene chloride and nickel carbonyl are metabolized to carbon monoxide, resulting in on-going exposure and potential for toxicity.
4. In victims of structure fires who present without severe burns, a plasma lactate greater than 10 mmol/L correlates with cyanide level greater than 40 mumol/L.
5. Empiric therapy with hydroxocobalamin may be considered for victims of structure fires in whom suspicion exists for cyanide poisoning.
6. CO poisoning, as well as cyanide toxicity, may cause selective bilateral injury to the globus pallidus.



FIGURE 17.53 ■ **Bilateral Globus Pallidus Lesions.** Selective bilateral injury to the globus pallidus may be seen with cellular asphyxiants such as carbon monoxide or cyanide poisoning. (Photo contributor: Lawrence B. Stack, MD.)

VANCOMYCIN-INDUCED RED MAN SYNDROME

Clinical Summary

Vancomycin is a glycopeptide antibiotic that has activity against gram-positive bacteria. It has little to no activity against gram-negative bacteria or mycobacteria. It is poorly absorbed after oral administration, although it may be used orally for treatment of pseudomembranous colitis. Intravenous administration is the most common route. This is well tolerated with minimal burning at the site of the IV; however, rapid infusion may occasionally cause degranulation of mast cells and basophils. As a result, the patient experiences erythematous flushing, particularly of the face and neck, hence the name "red man syndrome." Tachycardia and hypotension may occasionally be seen.

Management and Disposition

Slowing the intravenous infusion usually resolves the flushing. Increasing the dilution of vancomycin in solution may also assist with preventing the flushing. Diphenhydramine has been used for treatment and may be used as a pretreatment.

Pearls

1. Other syndromes known to causing flushing include scombroid poisoning, disulfiram reactions, and hydroxocobalamin infusions.
2. Concomitant administration of aminoglycoside with vancomycin may increase the risk of nephrotoxicity.

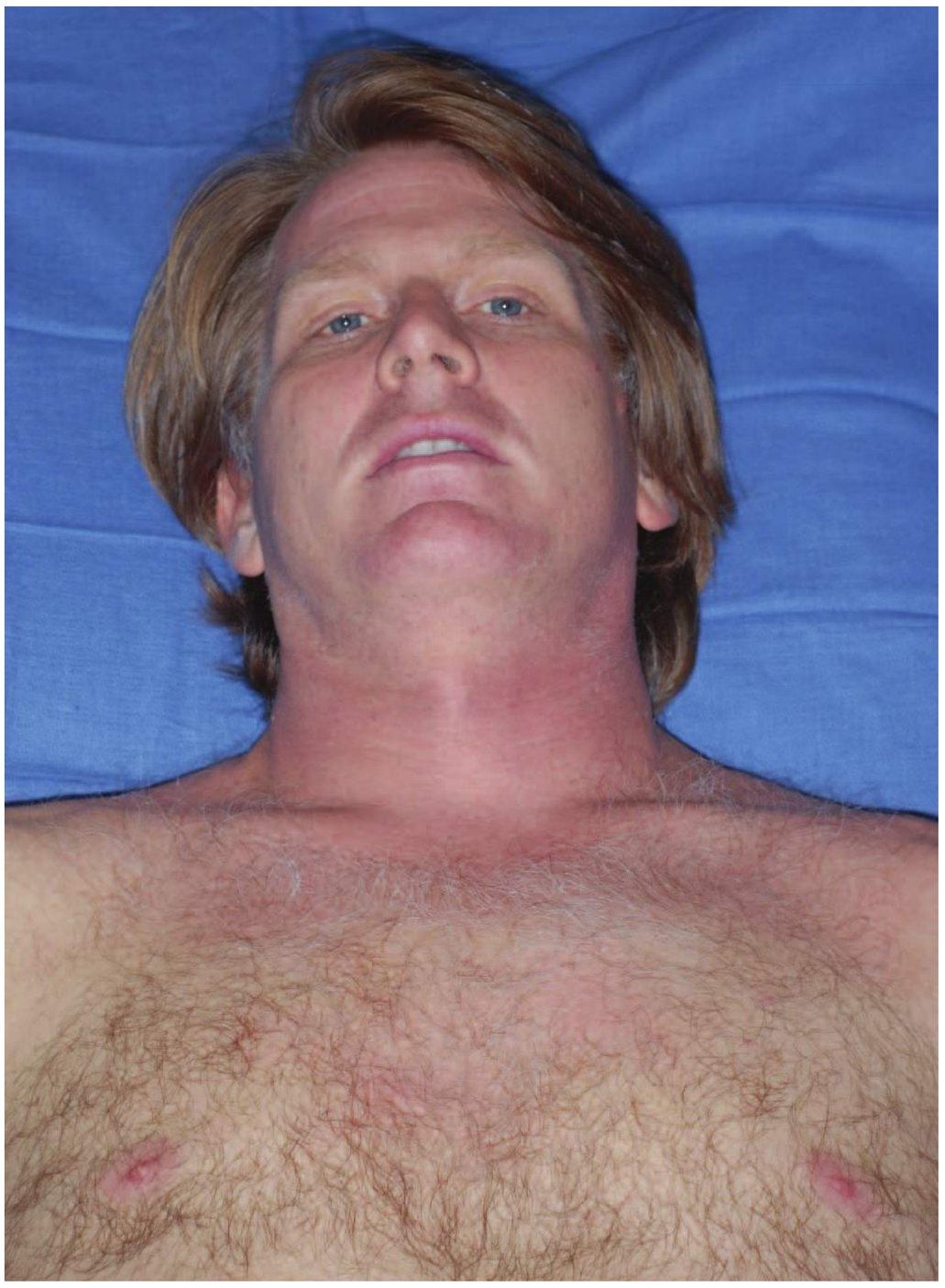


FIGURE 17.54 ■ **Red Man Syndrome.** Facial and neck flushing are manifestations that may be seen with red man syndrome from intravenous vancomycin infusion. (Photo contributor: R. Jason Thurman, MD.)

Clinical Summary

Botulinum is a potent neurotoxin that is derived primarily from Clostridium botulinum, although a few other clostridial species produce a homologous neurotoxin. Botulinum toxin blocks release of Ach, which results in decreased activation of muscarinic and nicotinic receptors. Initial effects may include nonspecific findings such as nausea and vomiting, constipation, and throat complaints. The classic neurologic findings are due to the lack of receptor activation of the nicotinic receptor at the neuromuscular junction. Dysarthria, dysphagia, diplopia, and mydriasis progress to a descending symmetric paralysis.

The common types of botulism include foodborne (ingestion of preformed toxin), infantile (in vivo production of toxin), and wound botulism (in vivo production of toxin). While foodborne has the features classically described, infantile botulism manifests as the constipated, floppy baby. Wound botulism is most commonly associated with "skin popping," a technique of subcutaneous injection of an illicit drug, usually black tar heroin. Diagnosis is initially a clinical one, with subsequent verification via a murine assay of the presence of botulinum toxin in a patient sample. This assay is performed through either the state department of health or the Centers for Disease Control and Prevention.



FIGURE 17.55 ■ **Infantile Botulism.** The floppy-constipated baby is a classic presentation of infantile botulism. (Photo contributor: Centers for Disease Control and Prevention.)

Management and Disposition

Any patient in whom botulism is suspected should be admitted to the hospital. Careful monitoring of the airway status is important to ensure that the patient has adequate ventilatory capacity. The local health department should be notified to assist with the procurement of botulinum antitoxin for foodborne and wound botulism patients. Human botulinum immune globulin is available through the California Department of Health for the treatment of infantile botulism.

Pearls

1. The initial chief complaint of a patient with wound botulism may be "sore throat" since the patient has dry mucus membranes and difficulty swallowing.
2. Since botulinum toxin does not cross the blood-brain barrier, the mental status should not be affected unless the patient has respiratory insufficiency.
3. Early signs of infantile botulism may be difficulty with feeding since feeding for an infant requires use of the cranial nerves.



FIGURE 17.56 ■ **Wound Botulism.** Wound botulism occurs from the in vivo production of botulinum toxin. It manifests the same neurotoxicity with the ptosis, bulbar paralysis, and respiratory compromise as foodborne botulism. Note the profound ptosis in this patient. (Photo contributor: William H. Richardson, III, MD.)

Clinical Summary

Since the discovery of the cause of a hemorrhagic disorder in Wisconsin cattle in the early 20th century, warfarin and its analogues have been used as rodenticides and pharmacologic agents. Warfarin is actually named after the Wisconsin Alumni Research Foundation. Warfarin-like xenobiotics inhibit the activity of vitamin K 2,3 epoxide reductase, thus affecting the production of the vitamin K–dependent clotting factors II, VII, IX, and X. While the effect on the production of clotting factors occurs soon after absorption, the clinical effects are delayed until the available activated factors are depleted.

Long-acting anticoagulants, known as "Superwarfarins," were developed as rodenticides due to the development of warfarin resistant rodents. Their mechanism of action is the same as warfarin, but they are more potent and exhibit a much longer duration of effect. Poisoned patients may present with any sequelae of an anticoagulated state spanning the spectrum of easy bruising to severe life-threatening hemorrhage.

Management and Disposition

Patients who are over-anticoagulated may require reversal with the administration of vitamin K. Life-threatening hemorrhage may require immediate reversal with administration of fresh frozen plasma. Guidelines for correction of vitamin K antagonists are published by the American College of Chest Physicians. Patients with intentional poisonings



FIGURE 17.57 ■ **Rodenticide Package.** Brodifacoum is a common long-acting anticoagulant (a "superwarfarin") used as rodenticide for mice. Note that the percent of active ingredient is only 0.005%; thus, ingestion of a few pellets by a child is unlikely to cause clinically significant anticoagulation. The patient with this packet ingested a large amount and required long-term reversal. (Photo contributor: R. Jason Thurman, MD.)



FIGURE 17.58 ■ **Warfarin Overdose.** Generalized bruising, as seen in this patient, is typical of a patient with a warfarin overdose. The INR of this patient was >10. (Photo contributor: R. Jason Thurman, MD.)

from the long-acting anticoagulants ("superwarfarins") may require high-dose vitamin K for several weeks to months due to the significantly prolonged effect of the drugs.

Pearls

1. Warfarin is actually a racemic mixture of R and S enantiomers which are metabolized by different CYP p450 pathways. The S enantiomer is more potent that the R enantiomer. Genetic polymorphisms influence the dose needed to achieve a therapeutic level.
2. Single unintentional ingestions of the "superwarfarins" by children under 6 years of age rarely result in clinically significant effects. Repetitive ingestions can result in coagulopathy. If a toddler has a vitamin K–dependent coagulopathy from a superwarfarin ingestion, the diagnosis of Munchausen by proxy should be entertained.
3. If a patient is on chronic warfarin therapy, be wary of the many xenobiotic interactions that can potentiate or antagonize the anticoagulant effect of the drug.



FIGURE 17.59 ■ **Rodenticide Pellets.** Typical appearance of the bluish-green brodifacoum pellets contained in the packaging. (Photo contributor: R. Jason Thurman, MD.)

CAUSTIC INGESTION

Clinical Summary

Caustics are a diverse group of household and industrial products and pharmaceutical agents that cause functional and histological tissue damage through direct contact. They represent the second most common toxic exposure for children 5 years of age or under. These agents are frequently described in terms of pH, with acids typically having a pH less than 3 and alkali (bases) typically having a pH greater than 11. Despite a near-physiologic pH, phenol may produce severe burns due to a high titratable acid reserve (TAR).

Alkali exposure results in a liquefactive necrosis, with deep and progressive tissue damage, predominantly to the esophagus. Endoscopic grading of esophageal burns is similar to thermal burns, ranging from mucosal hyperemia and edema (grade I) to full-thickness burns (grade III). Acid ingestion results in coagulative necrosis, which limits the depth of penetration. Damage is predominantly localized to the gastric mucosa, with pooling of the caustic agent in the antrum.

Management and Disposition

The primary goal of management is airway assessment and stabilization. Hypotension is a grave finding. A serum pH less than 7.20 may indicate the need for surgical exploration. Activated charcoal decontamination is relatively contraindicated. Endoscopy is recommended after large or deliberate caustic ingestion, presence of oral burns, or persistent refusal to take oral liquids. Steroids are occasionally used as an effort to prevention of esophageal strictures in selected patients, but the decision to administer is made based upon endoscopic grading in conjunction with the gastroenterologist.

Pearls

1. The absence of oropharyngeal burns is a poor predictor of distal esophageal injury. The presence of vomiting, drooling, or stridor is more predictive of significant esophageal injury on endoscopy.
2. Analogous to thermal burns after smoke inhalation, upper airway edema and airway obstruction may occur abruptly.
3. Ingestion of muriatic acid (HCl) results in an initial non-anion gap metabolic acidosis.



FIGURE 17.60 ■ **Caustic Oropharyngeal Burns.** This patient suffered caustic burns from the accidental drinking of a highly alkali solution that was stored in a soda can. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 17.61 ■ **Caustic Oropharyngeal Burns.** Close-up image of the alkali burns on the tongue of the patient in Figure 17.60. (Photo contributor: Lawrence B. Stack, MD.)

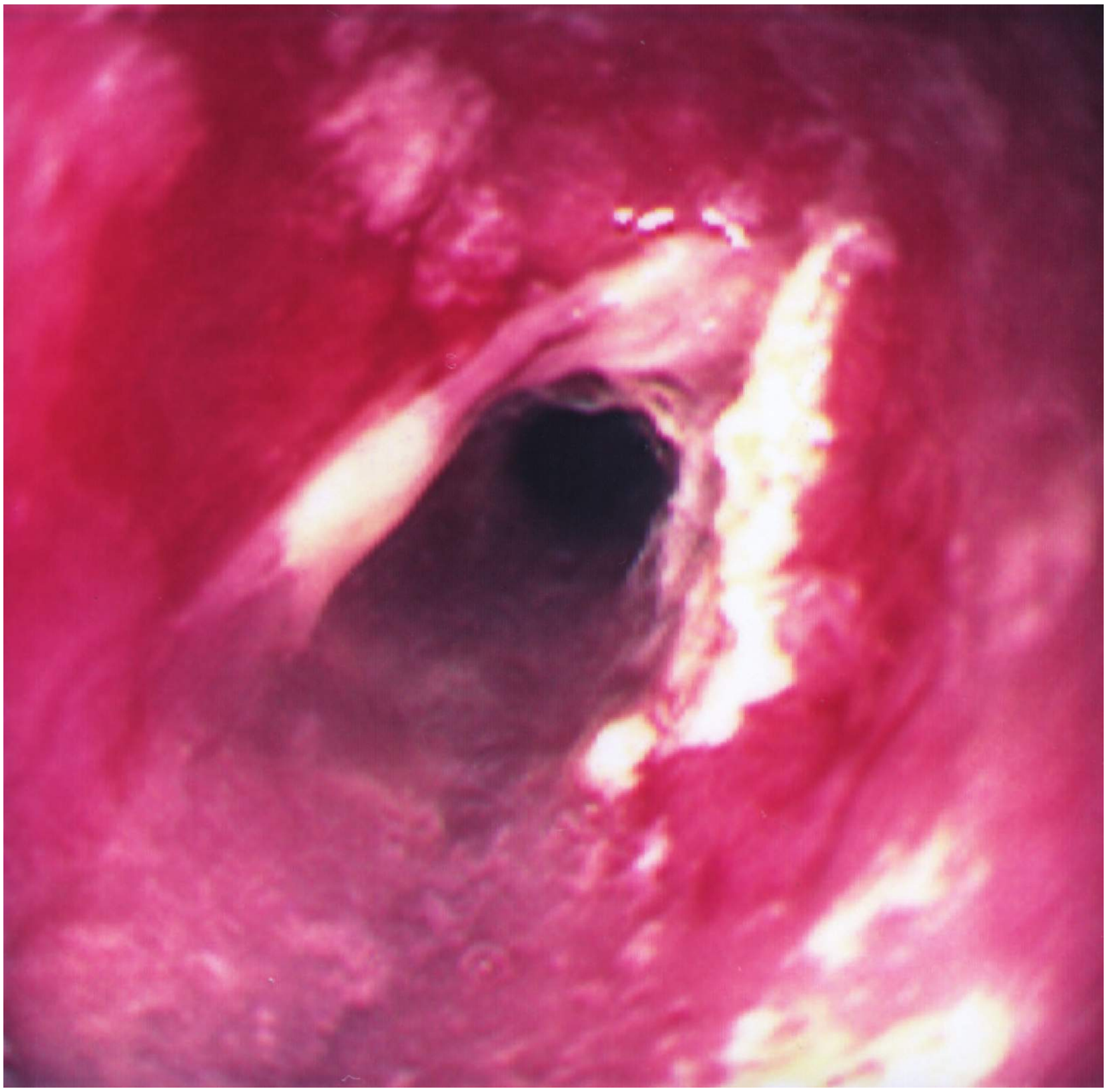


FIGURE 17.62 ■ **Caustic Esophageal Burns.** These esophageal burns were caused by an accidental ingestion of lye in a pediatric patient. (Photo contributor: Philip E. Stack, MD.)

HYDROFLUORIC ACID BURNS

Clinical Summary

Hydrofluoric acid (HF) is a colorless, corrosive liquid available in both commercial (>20%) and household (<20%, typically 6-12%) formulations. Commercially, HF is used in glass etching, electroplating, and semiconductor manufacturing. Consumer products are typically marketed as rust removers and chrome cleaners. Although toxic via the dermal, ocular, pulmonary, and GI routes, the majority of patients present after dermal exposure, typically to the hands and fingers. The severity of local injury depends upon the HF concentration and the extent of exposure.

Symptoms and tissue effects may be appreciably delayed, especially with household formulations. In the setting of hand exposure, pain is typically described as progressive severe unremitting deep burning sensation. Early local erythema is variable. Especially with higher HF concentrations, a pale blanched appearance may develop. In addition to the coagulative necrosis noted with other inorganic acids, HF causes toxicity by the binding and precipitation of calcium ions. In addition to local effects, significant and potentially life-threatening systemic effects may occur including hypocalcemia, hyperkalemia, and hypomagnesemia. Concentrations greater than 50% may cause rapid decompensation with even small dermal exposures (1% total body surface area[TBSA]).

Management and Disposition

Treatment varies depending upon the route of exposure, but is directed toward decontamination, neutralization of the fluoride ion, and pain control. In the setting of ingestion or significant dermal exposure (more than 5% TBSA of a household HF formulation), serum electrolytes, including calcium and magnesium, should be determined. In addition to pain control, management options for hand burns include commercially available calcium gluconate gel, subcutaneous calcium gluconate infiltration, and regional intravenous and intra-arterial calcium gluconate infusion.

Pearls

1. An expedient 2.5% calcium gluconate gel can be made by adding 3.5 g calcium gluconate powder to 5 oz (150 mL) of water-based lubricant. Alternatively, 10 crushed 1-g calcium carbonate antacid tablets may be added to 20 mL water-based lubricant. The resultant gel may be placed in a rubber glove and placed on the patient's hand for topical treatment.
2. With significant dermal exposure or ingestion, prolonged cardiac and serum electrolyte monitoring may be required; sudden, delayed cardiac arrest has occurred.
3. Replacement of calcium and magnesium may require substantially larger doses than typically required.



FIGURE 17.63 ■ **Hydrofluoric Acid Burns.** Hydrofluoric acid burn due to application of a rust remover agent. The patient presented hours later after initial use with severe, deep pain in the thumb and index finger. (Photo contributor: Karen Rogers, MD.)



FIGURE 17.64 ■ **Hydrofluoric Acid Burn—Nail beds.** Hydrofluoric acid can seep underneath the nail beds, resulting in blanched discoloration. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 17.65 ■ **Hydrofluoric Acid Burn Treatment.** Expedient topical therapy for hydrofluoric acid burns can be made using crushed calcium containing antacids mixed with surgical jelly. The jelly may be placed in a rubber glove, then the glove on the patient's hand. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)

ARSENIC POISONING

Clinical Summary

Arsenic, a tasteless and odorless metalloid, is well absorbed by multiple routes of administration. Although arsenic exists in both inorganic and organic species, only the inorganic form is responsible for toxicity. Contaminated soil and water with inorganic arsenic are the primary sources of exposure to the general population. Chronic arsenic poisoning due to contaminated water continues to be a global health issue.

Arsenic inhibits multiple enzymes critical to the production of ATP. It inhibits pyruvate dehydrogenase complex, decreases the citric acid cycle, and decreases gluconeogenesis. Acute arsenic poisoning typically starts with acute onset of nausea, vomiting, abdominal pain, and "rice water" diarrhea. Acute encephalopathy, acute renal failure, lung injury, and death may occur. Later findings in survivors include alopecia, rash, Mees lines, and neuropathy. Chronic toxicity results in dermatologic changes such as hyperpigmentation or hypopigmentation. Hyperkeratosis may occur on the skin, particularly the palms and soles. Peripheral vascular disease (blackfoot disease) may occur. Arsenic is a known carcinogen and is associated with skin and lung cancers.

Management and Disposition

After an acute ingestion, an abdominal radiograph may demonstrate radiopaque material in the GI tract. Supportive care with maintenance of electrolytes is important. A 24-hour urine collection in a metal-free container is the optimal method for determining arsenic burden. However, the acutely ill patient will likely require chelation before urine results are available. Chelation is usually initiated with intramuscular dimercaprol (British anti-Lewisite, BAL). Succimer, an oral analogue of the BAL, may be useful in subacute poisoning. Patients with suspected acute arsenic poisoning should be admitted to an ICU.

Pearls

1. When a nonselective heavy metal urine test is performed for possible arsenic exposure, the measured arsenic typically reflects the presence of nontoxic organic species from dietary sources such as seafood, rather than toxic inorganic species. As such, urine should either be speciated, or the patient advised to refrain from seafood prior to urine collection.
2. Intravenous arsenic trioxide is approved by the Food and Drug Administration for treatment of acute promyelocytic leukemia unresponsive to other therapies.
3. Acute arsenic poisoning is one of the few heavy metals that is directly cardiotoxic as it blocks delayed rectifier channels. Torsades de pointes has been described in the setting of acute arsenic poisoning.



FIGURE 17.66 ■ **Chronic Arsenic Poisoning.** Hyperpigmentation of the skin is a clinical finding from chronic arsenic poisoning. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 17.67 ■ **Chronic Arsenic Poisoning.** Hyperpigmentation and hypopigmentation along with hyperkeratosis are findings on the palms of patients with chronic arsenic poisoning. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 17.68 ■ **Chronic Arsenic Poisoning.** The patchy hair loss seen in this photograph is from chronic arsenic poisoning. (Photo contributor: Selim Suner, MD, MS.)



FIGURE 17.69 ■ **Mees Lines.** Characteristic Mees lines of chronic arsenic poisoning. Note the transverse white lines on all the nails of both hands. Mees lines are often seen in conjunction with polyneuropathy of arsenic poisoning. (Photo contributor: Robert S. Hoffman, MD.)

IRON POISONING

Clinical Summary

Iron is a commonly used pharmaceutical agent and supplement found in prenatal and multivitamins. Although the incidence has declined, acute iron poisoning remains a significant cause of pediatric morbidity and mortality. The toxic dose depends upon the quantity of elemental iron in the preparation, which in turn depends upon the iron formulation. While the minimum toxic dose remains controversial, ingestion of more than 40 mg/kg elemental iron may result in toxicity, while ingestion of more than 60 mg/kg elemental iron is associated with severe morbidity and possible mortality.

Iron toxicity is classically described as a four-stage process. Stage I develops within the first few hours of ingestion and reflects the direct caustic effects of iron on the GI tract. Signs and symptoms may include abdominal pain and GI bleeding. Stage II, variably present, is a redistributive or quiescent phase; although the initial GI symptoms resolve during this phase, acidosis and end-organ toxicity may still develop. With significant toxicity, individuals may progress directly from Stage I to Stage III. Stage III is the phase of overt shock, metabolic acidosis, and end-organ dysfunction (including delayed hepatic failure). Individuals who survive Stage III may rarely progress to Stage IV, gastric outlet obstruction secondary to the initial caustic insult of Stage I.

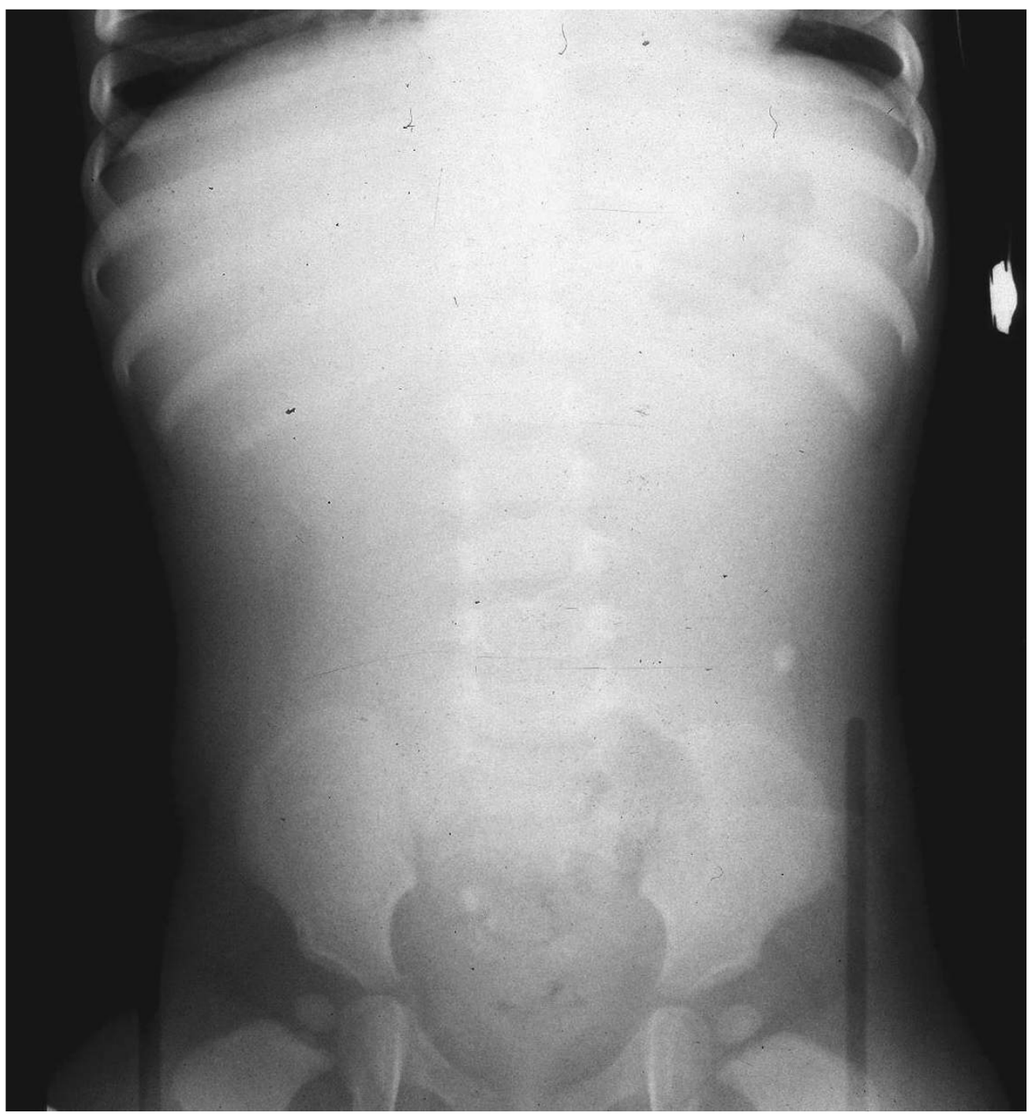


FIGURE 17.70 ■ **Radiopaque Iron.** KUB radiograph of a patient with an acute iron ingestion, demonstrating radiopaque foreign bodies as noted in the left mid-quadrant and right lower pelvis. (Photo contributor: Saralyn R. Williams, MD.)

Management and Disposition

Activated charcoal does not bind iron. Whole-bowel irrigation has been advocated for substantial ingestions and for patients with evidence of iron tablets on abdominal radiographs. Serum iron levels peak between 2 and 6 hours postingestion. Levels obtained more than 6 hours after ingestion are unreliable due to tissue redistribution. Patients with evidence of iron toxicity (eg, persistent vomiting, acidosis, altered mental status, and hypotension) or a 4- to 6-hour postingestion serum level more than 500 mcg/dL should receive chelation with deferoxamine.

Pearls

1. Acute iron ingestion and the risk for toxicity must be assessed based upon the quantity of elemental iron ingested, not the total amount ingested.
2. The absence of radiopaque materials on abdominal radiographs is not a reliable indicator to exclude potential iron toxicity; liquid and pediatric (chewable) formulations are not typically radiopaque.
3. Despite the potential for anaphylactoid reactions and hypotension, patients requiring chelation therapy should receive deferoxamine via the intravenous route.



FIGURE 17.71 ■ **Vin Rose Urine; Iron Poisoning.** A rare example of the progression of coloration of vin rose urine (from the excreted ferrioxamine complex) over 15 hours of chelation with deferoxamine is shown. Deferoxamine chelates free iron (but not iron present in the transferrin, hemoglobin, hemosiderin, or ferritin) (Reproduced with permission from Strange GR, Ahrens WR, Schafermeyer RW et al. Pediatric Emergency Medicine. 3rd ed. New York, NY: McGraw-Hill; 2009: Fig. 127-1.)

Clinical Summary

Although the prevalence of markedly elevated lead levels in the population has been declining, acute and chronic lead poisoning still occur. Lead is well absorbed by the lungs and less well via the GI tract. Lead paint in older homes is a continued source of lead exposure. Other possible exposures may occur from occupational exposures, retained lead bullets in synovial fluid, jewelry, lead-painted toys, fishing weights, ceramic glazes, and cosmetics. Severe lead poisoning in adults has also been associated with ingestion of contaminated moonshine.

Lead poisoning affects multiple organ systems. Neurotoxicity may range from subtle personality changes to encephalopathy and cerebral edema. At the societal level, even small lead burdens are associated with statistically significant decreases in intelligence quotient. Motor neuropathy such as foot drop and wrist drop may be seen in adult patients, especially after occupational exposure. Microcytic anemia may occur and basophilic stippling of the red cells may be seen. Hypertension and an acute nephropathy may occur. Abdominal pain may be described by patients but, unlike other heavy metal poisonings, constipation is more likely than diarrhea. Radiographic "lead lines," bands of increased density on long bones metaphyses, may be seen in young children. These densities are not due to deposition of lead but rather increased calcium deposition.

Management and Disposition

Whole-blood lead level is the primary measure of lead exposure, but is not usually available in real time. Radiographic studies may demonstrate radiopaque substances from ingested jewelry or paint chips in children. Lead encephalopathy must be aggressively managed. Dimercaprol (BAL) is administered intramuscularly and $CaNa_2EDTA$ is later given intravenously. Chelation with oral succimer is currently recommended in asymptomatic children with levels between 45 and 70 mcg/dL. Reducing the exposure in children is paramount to treatment and the source of the lead may be elusive.

Pearls

1. One source of lead exposure in children is through the occupation of the parent. Workers in a lead dust environment will bring home the lead dust on their clothes and shoes.
2. Imported eye cosmetics with lead have been a source of pediatric exposures in certain ethnic groups.
3. Azarcon and greta are lead-based remedies that are used to treat diarrheal illnesses.
4. Adults usually require much higher blood lead levels than children before encephalopathy occurs.

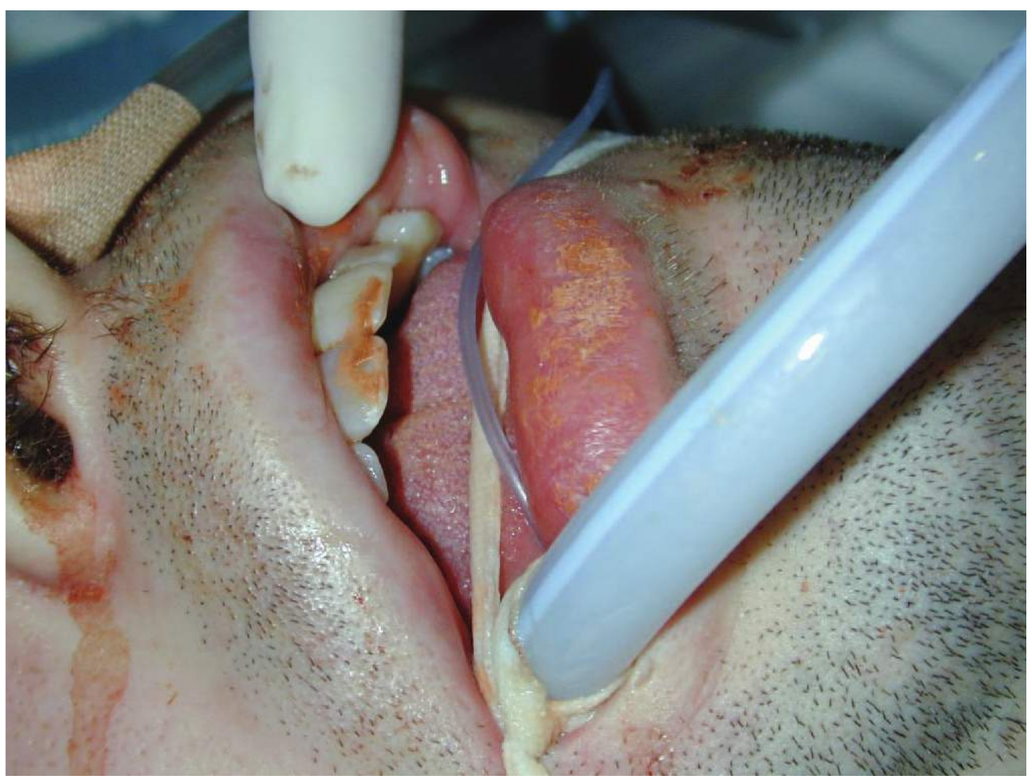


FIGURE 17.72 ■ **Acute Lead Poisoning.** This patient presented with acute encephalopathy after ingesting lead-based, tangerine-scented gloss glaze used for making pottery. The substance is present on his teeth, lips, and nose. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)

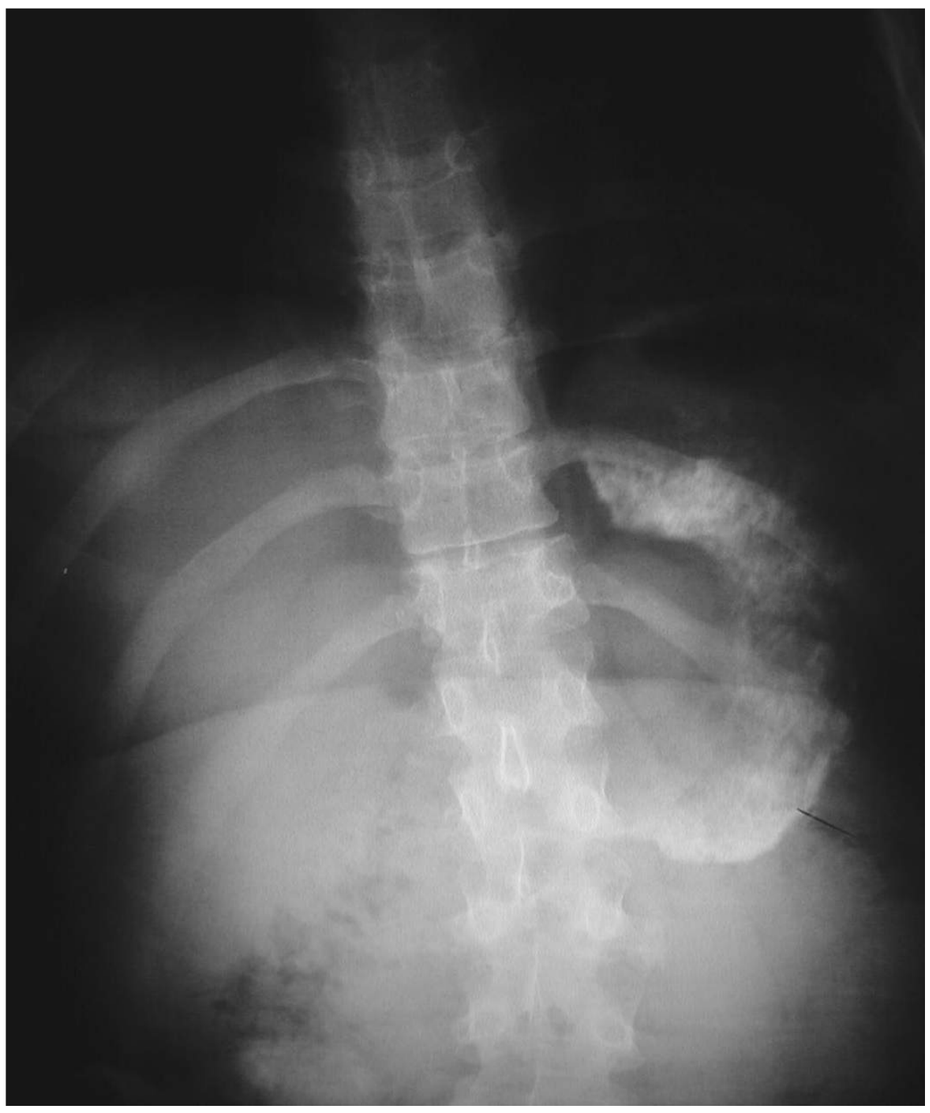


FIGURE 17.73 ■ **Radiopaque Lead.** The abdominal x-ray of the patient in Fig. 17.72. Lead is radiopaque when ingested. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)



FIGURE 17.74 ■ **Lead Lines.** Lead lines seen in a pediatric patient with chronic lead poisoning. The increased radiographic densities on the metaphyseal growth plates demonstrate radiological growth retardation and increased calcium deposition. (Photo contributor: David Effron, MD.)

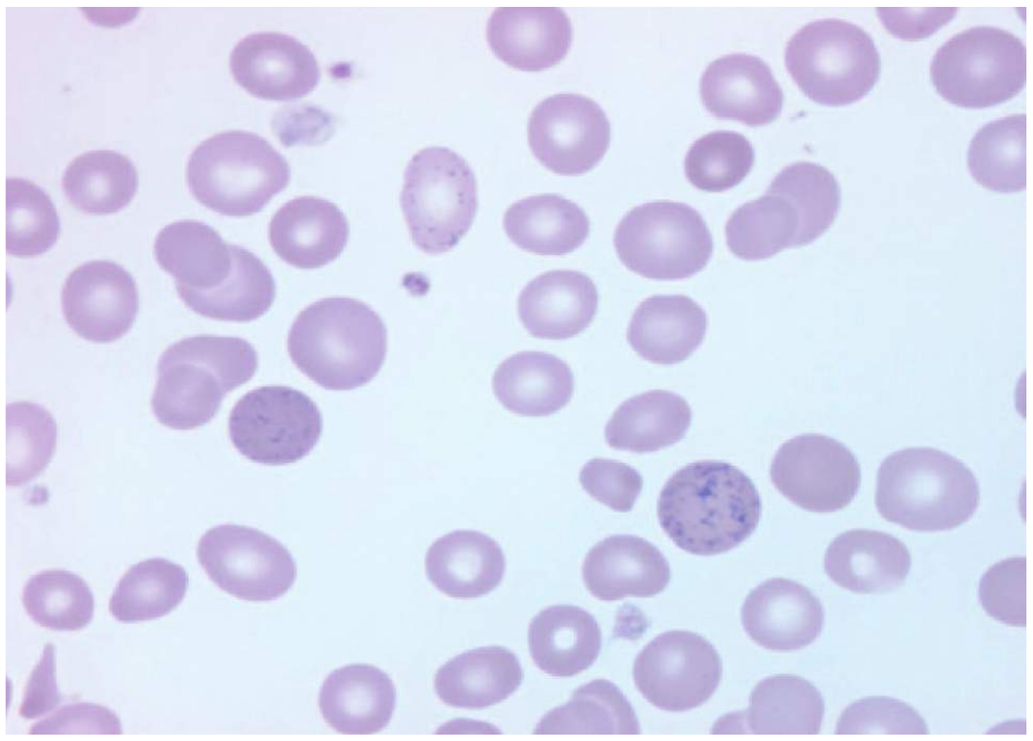


FIGURE 17.75 ■ **Basophilic Stippling.** Basophilic stippling along with a microcytic anemia may be seen in patients with chronic lead poisoning. (Photo contributor: Debbie Bennes, BS, MLT, ASCP.)

Clinical Summary

Mercury occurs in three different forms (elemental, inorganic, and organic), each with its own clinical pattern of poisoning. Elemental mercury ("quicksilver") is found in thermometers and sphygmomanometers. Elemental mercury poisoning is associated with inhalation of volatilized mercurial ions, which may occur after vacuuming or heating. Manifestations include cough, fevers, chills, and dyspnea. Acute interstitial pneumonitis may occur and may progress to severe lung injury and death. Inorganic mercury poisoning usually occurs from the ingestion of the mercurial salts. Initial presentation is acute caustic gastroenteritis that may be hemorrhagic. Renal failure is a prominent finding in these patients. Organic mercury poisoning occurs from ingestion of short-chain alkyl mercurial compounds. Methylmercury distributes into brain tissue and causes neurologic disease such as ataxia, paresthesias, visual difficulties, movement disorders, and speech difficulties. Methylmercury is also a known teratogen.



FIGURE 17.76 ■ **Subcutaneous Mercury.** Lateral radiograph of an ankle demonstrating elemental mercury in the tissues. The patient had a barometer break into his skin. (Photo contributor: Saralyn R. Williams, MD.)

Management and Disposition

After an ingestion of a mercurial substance, a radiograph may demonstrate radiopaque material in the GI tract. If elemental mercury was injected intravenously, mercurial emboli may be seen in the lungs. Local injection in the skin may demonstrate mercury deposition in the soft tissues. Ingestion of elemental mercury rarely results in significant absorption. For inhalational injury due to elemental mercury, respiratory support may be required. Ingestion of inorganic mercury may lead to early cardiovascular collapse as a result of the severe volume depletion. Fluid resuscitation and electrolyte management are critical. Chelation with dimercaprol (BAL) may be initiated early and when the patient is able to take oral medications, the chelator may be switched to succimer. For organic mercury poisoning, oral succimer may be the first-line agent.

Pearls

1. Elemental mercury toxicity has occurred when it is heated and used to extract gold from jewelry.
2. "Mad as a hatter" is a term used to describe the delirium for anticholinergic poisoning; however, the term is derived from the erethism and hatter's shakes from



FIGURE 17.77 ■ **Mercurial Emboli.** Appearance of mercurial emboli in the pulmonary vascular tree on chest x-ray. Though this may be seen from intentional intravenous mercury injection, this patient absorbed the mercury intravenously following an accident involving multiple shattered thermometers. (Photo contributor: John Worrell, MD.)

mercury exposure during the felt-hat manufacturing process in the late 19th and early 20th century.

3. Organic mercury is eliminated via the fecal route, so urine samples for methylmercury will not be reflective of the body burden.
4. BAL is suspended in peanut oil, and so can only be administered as an intramuscular injection, and to nonpeanut-sensitive individuals.



FIGURE 17.78 ■ **Mercury Salts.** Mercury salts were used as topical antiseptics. Ingestion of inorganic mercury is caustic to the GI tract and causes rapid renal failure. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)



FIGURE 17.79 ■ **Mercury Tablets.** An example of the tablets from the bottle in Fig. 17.78. Although faded with age, the coffin-shaped tablets contain the word "poison" on one side and the "skull-and-crossbones" on the other. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)

Clinical Summary

Mushrooms are the fruiting bodies of certain fungi. While many species of mushrooms can cause toxicity when ingested, only a few contain amatoxins and account for most fatalities attributed to mushroom ingestion. Examples include Amanita phalloides (the "death cap") and Amanita ocreata (the "destroying angel") species. Amatoxin poisoning results in severe symptoms of gastroenteritis at least 6 to 24 hours after ingestion. Symptoms include nausea, vomiting, profuse watery diarrhea, and abdominal pain. GI symptoms may last 12 to 24 hours and are followed by a latent period of apparent improvement. This period is followed by a rise in liver enzymes and bilirubin. Fulminant hepatic failure and renal failure may become apparent.

The more common mushroom exposures include those with GI toxins. Ingestions result in an acute nausea, vomiting, and diarrhea that occurs within 2 hours of ingestion (eg, <6 hours of ingestion). Examples of mushrooms include the Chlorophyllum molybdites which looks like a toasted marshmallow in the grass. Other types of mushroom poisonings include gyromitrin-containing mushrooms that are misidentified for the popular morels. Gyromitrin mushrooms cause status epilepticus that is responsive to high-dose pyridoxine. Coprine-containing mushrooms only cause toxicity when ethanol is co-ingested as they cause a disulfiram reaction.

Management and Disposition

For the GI toxins containing mushrooms, management is supportive care. For the amatoxin containing mushroom, administration of activated charcoal may be recommended depending on the time since ingestion. Specific interventions that may be helpful but are yet unproved include charcoal hemoperfusion, high-dose cimetidine, high-dose penicillin, high-dose ascorbic acid, silibinum, and NAC. Consultation with the local poison center is recommended.

Pearls

1. A single "death cap" may contain enough amatoxin to kill an adult.
2. Cooking mushrooms does not substantially alter their toxicity.
3. Not all Amanita species of mushrooms cause hepatotoxicity when ingested. Some Amanita species are hallucinogens and one causes renal failure.
4. Be wary of using the "6-hour rule" with mushroom scavengers who may have ingested multiple species of mushrooms.



FIGURE 17.80 ■ Amanita phalloides. The "death cap" produces amatoxins and accounts for most of the fatalities due to mushroom ingestion. (Photo contributor: Edward J. Otten, MD.)



FIGURE 17.81 ■ Amanita ocreata. These mushrooms were samples of Amanita ocreata provided by a family who had ingested them and subsequently developed significant hepatotoxicity. (Photo contributor: Division of Medical Toxicology, University of California, San Diego.)



FIGURE 17.82 ■ **Morel Mushroom.** Morels are considered a delicacy; however, inexperienced mushroom hunters may collect a "false morel" which contains gyromitrin. Ingestion of gyromitrin results in status epilepticus that is responsive to intravenous pyridoxine. (Photo contributor: Saralyn R. Williams, MD.)



FIGURE 17.84 ■ **Coprinus Mushrooms.** Coprinus mushroom cause toxicity when ethanol is co-ingested, resulting in a disulfiram-like reaction. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)



FIGURE 17.83 ■ Chlorophyllum molybdites**.** This mushroom is ubiquitous and is a common culprit for mushroom toxicity. The patient typically presents with acute nausea vomiting less than 2 hours after ingestion. (Photo contributor: Saralyn R. Williams, MD.)

Clinical Summary

Cardiac glycosides (CGs) are found in the leaves, flowers, and seeds of Nerium oleander (common oleander), Thevetia peruviana (yellow oleander), Digitalis purpurea (foxglove), Strophanthus gratus (ouabain), Convallaria majalis (lily of the valley), Apocynum cannabinum (dogbane), Urginea maritima and Urginea indica (squill), and Cheiranthus cheiri (wallflower). If ingested, they produce clinical findings similar to digoxin toxicity. The drinking of foxglove and oleander tea may be a cause of CG toxicity. Therapeutic effects occur from inhibition of the cardiac cell membrane sodium-potassium ATP pump, resulting in increased automaticity, decreased conduction through the atrioventricular (AV) node, and improved inotropy.

Toxic effects are an exaggeration of therapeutic effects. Bradydysrhythmias may result from impaired pacemaker function. Tachydysrhythmias may occur from increased automaticity. Nausea, vomiting, abdominal pain, confusion, depression, and fatigue may be present. Headaches, paresthesias, weakness, scotomas, and visual color disturbances (yellow halos around lights) may also be seen.

Management and Disposition

Atropine may be initially given for bradydysrhythmias. Refractory bradydysrhythmias require pacing. Ventricular tachydysrhythmias have been treated with phenytoin or lidocaine when digoxin-specific Fab fragments are not available. Activated charcoal is the preferred method of decontamination. Cardioversion should be avoided in CG toxicity. Digoxin-specific Fab fragments are the treatment of choice for life-threatening dysrhythmias or CG-induced hyperkalemia.

Pearls

1. Treat CG overdose from plant exposure in the same way as an acute digoxin overdose. Higher doses of digoxin-specific Fab fragments may be required.
2. Calcium should be avoided in treating CG-associated hyperkalemia, as it may worsen ventricular arrhythmias.
3. Dysrhythmias characterized by increased automaticity coupled with the presence of conduction disturbances are highly suggestive of CG toxicity.



FIGURE 17.85 ■ Nerium oleander **(Common Oleander).** A common decorative plant in subtropical climates often seen lining roads and highways. Flowers may be white, yellow, red, or purple. Drinking a tea brewed from the leaves of oleander results in severe cardiac glycoside poisoning. (Photo contributor: Saralyn R. Williams, MD.)



FIGURE 17.86 ■ Digitalis purpurea **(Purple Foxglove).** The ornamental plant. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 17.87 ■ Convallaria majalis **(Lily of the Valley).** While both beautiful and fragrant, lily of the valley contains multiple types of toxic cardiac glycoside compounds. (Photo contributor: Saralyn R. Williams, MD.)

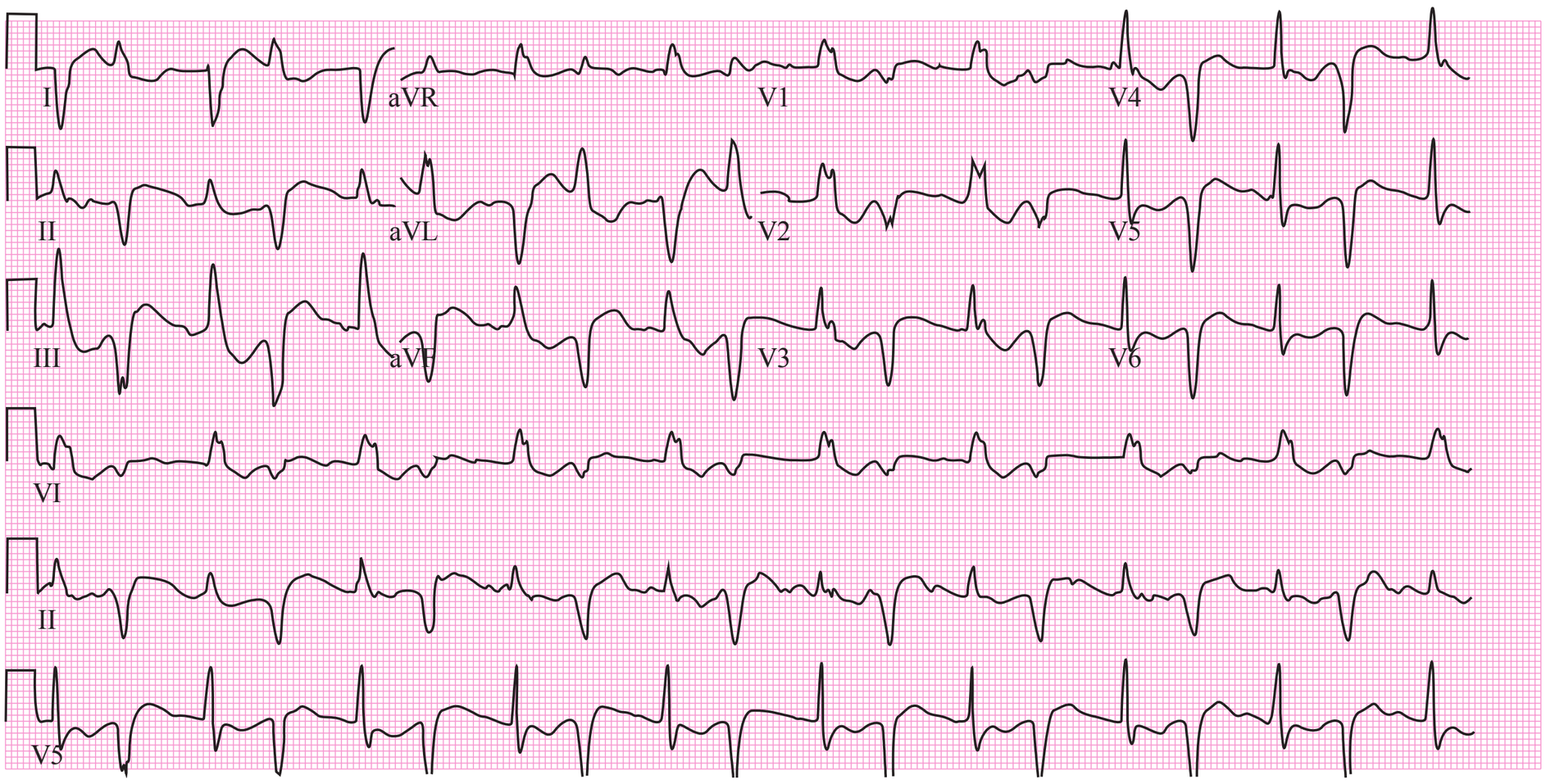


FIGURE 17.88 ■ **Bidirectional Ventricular Tachycardia.** An example of bidirectional ventricular tachycardia that occurred in a patient with digitalis toxicity. (Photo contributor: Binh Ly, MD.)

Clinical Summary

Many common houseplants such as dieffenbachia (dumbcane) and peace lily cause irritant effects when ingested owing to large amounts of insoluble oxalate crystals in its leaves. The oxalate crystals are highly irritating, and those who ingest the leaves experience painful burning of the lips, tongue, mouth, and esophagus. Marked swelling of the tongue, lips, and oropharynx can occur, and airway patency may become a major issue in managing these patients. Ocular exposures may occur as well, resulting in painful burning, erythema, and eyelid swelling. Fortunately, these calcium oxalate crystals are not absorbed and the hypocalcemia associated with soluble oxalates is not an issue.

Management and Disposition

Topical anesthetics are helpful in controlling severe pain from burning mucous membranes. Management is largely supportive, as the painful oral burns experienced with these exposures usually limit ingestion. As with any oropharyngeal burn, airway issues must be addressed. A period of observation is appropriate to make sure that airway compromise does not occur with continued swelling. If leaves are swallowed and the patients are symptomatic, GI consultation should be considered to assess the extent of esophageal injury. GI decontamination is usually not necessary.

Pearls

1. Performance of nasopharyngoscopy may be helpful in assessment of airway patency for more posterior burns.
2. Patient should be instructed not to swallow topical anesthetics, as toxicity may result with extensive use.



FIGURE 17.89 ■ Dieffenbachia. Dieffenbachia is a common houseplant because of its colorful appearance and ease of indoor growth. (Photo contributor: Kevin J. Knoop, MD, MS.)



FIGURE 17.90 ■ **Peace Lily.** The attractive foliage and blooms make this a popular house plant; however, it is not a true lily. (Photo contributor: Saralyn R. Williams, MD.)

JIMSONWEED INGESTION

Clinical Summary

Jimsonweed (Datura stramonium) is a toxic plant that contains tropane alkaloids consisting of atropine, scopolamine, and hyoscyamine compounds. Ingestion may occur through the drinking of tea made from the leaves or flowers of jimsonweed or from eating the plant's seeds or leaves. Poisoned victims demonstrate an anticholinergic toxidrome resulting from the antimuscarinic receptor effects of atropine and scopolamine. Patients may exhibit altered mental status, xerostomia, xeroderma, xerophthalmia, blurred vision, mydriasis, tachycardia, decreased bowel and bladder motility, and hyperthermia.

Management and Disposition

Treatment initially consists of assessing the ABCs (airway, breathing, circulation) and stabilization measures. Hypotension resulting from tropane alkaloid ingestion usually responds to fluid boluses. Vasopressor agents are rarely necessary. Activated charcoal may be recommended early after ingestion but may not change outcome as the toxins are absorbed rapidly. Whole-bowel irrigation is contraindicated with intestinal ileus and must be considered with great caution in jimsonweed ingestion due to decreased bowel motility. Physostigmine may be of benefit to treat severe anticholinergic toxicity, but may be better utilized as a diagnostic agent after consultation with the poison center. Severe agitation and psychosis may be treated with benzodiazepines and carefully administered properly dosed physostigmine.

Pearls

1. Examination of the axillae for xeroderma may be helpful to detect peripheral anticholinergic syndrome and distinguish between anticholinergic and sympathomimetic toxidromes.
2. Administering 1% pilocarpine eye drops does not reverse anticholinergic mydriasis.
3. Jimsonweed toxicity should be considered in the differential diagnosis of children and adolescents presenting with acute altered mental status, especially when accompanied by signs of anticholinergic toxicity.



FIGURE 17.91 ■ **Jimsonweed.** Jimsonweed seed pod with dried seeds. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)



FIGURE 17.92 ■ **Jimsonweed-Induced Xerostomia.** A severe case of xerostomia from the antimuscarinic effects of jimsonweed ingestion. Note the associated flushing of the patient's cheek. (Photo contributor: R. Jason Thurman, MD.)

Clinical Summary

Peyote (Lophophora williamsii) is a cactus plant found primarily in the southwestern United States. The cactus contains a significant amount of mescaline, a potent hallucinogen with structural similarities to norepinephrine. Peyote buttons and seeds are frequently ingested for recreational use, but are also used in the religious ceremonies of some Native American groups. Toxicity of peyote is generally mild and self-limited, but hypotension and respiratory depression can occur. Mescaline induces some sympathomimetic effects due to its similarity to norepinephrine; marked visual hallucinations and a sense of depersonalization follow. These effects are often accompanied by unpleasant GI symptoms such as severe nausea and vomiting. Full recovery from these symptoms usually occurs within a few hours.

Management and Disposition

Treatment of peyote ingestion is largely supportive; severe toxic effects are uncommon. Marked agitation may be managed with benzodiazepines.

Pearls

1. An individual peyote button contains about 45 mg of mescaline; a mescaline dose of 5 mg/kg usually produces psychotropic effects.
2. Botulism poisoning has been reported from the ingestion of dried peyote buttons.



FIGURE 17.94 ■ **Peyote Cactus.** An individual crown of peyote. (Photo contributor: Martin Terry, PhD.)



FIGURE 17.93 ■ **Peyote Cactus Patch.** Patch of peyote crowns growing in the desert. (Photo contributor: Martin Terry, PhD.)



FIGURE 17.95 ■ **Peyote Button.** This desiccated button of peyote is the form that is ingested for recreational or religious purposes. (Photo contributor: Martin Terry, PhD.)

TOXALBUMIN INGESTION

Clinical Summary

The jequirity pea (Abrus precatorius) and castor bean (Ricinus communis) belong to a family of poisonous plants that contain toxalbumins. The chief toxin of the jequirity pea is abrin, which is structurally very similar to the toxin ricin of the castor bean. Ingestion of jequirity peas and castor beans rarely results in toxicity, as a majority of the plant toxin is concentrated within the hard shell of the seeds. However, when these seeds are chewed or the shell is digested, symptoms of severe gastroenteritis follow within 1 to 3 days. Nausea, vomiting, abdominal pain, and diarrhea are common but in severe cases may be accompanied by hemorrhagic gastritis and hematemesis, seizures, arrhythmias, marked dehydration, CNS depression, and even death. Unfortunately, because of the colorful attractive nature of jequirity peas and castor beans, most cases of ingestion occur in the pediatric age group. Because of the very high potency of these toxins, they are occasionally used for homicidal purposes and growing concern exists for their potential utilization as an agent of bioterrorism.

Management and Disposition

Treatment of jequirity pea and castor bean ingestions is largely supportive, as there is no specific antidote for abrin or ricin. Gastric decontamination may be considered and may include activated charcoal and whole-bowel irrigation. In asymptomatic patients, decontamination, careful observation, and close follow-up are adequate. With symptoms of toxicity, however, admission is recommended, as the potential for marked clinical worsening is present.



FIGURE 17.96 ■ **Castor Bean Plant.** The castor bean plant is large and leafy; it may reach a height of 10 to 12 ft. (Photo contributor: Alex Wilson.)

Pearls

1. Most jequirity pea and castor bean ingestions are benign, as the vast majority of toxin resides within the undigested shell of the plant.
2. The toxalbumins abrin and ricin are structurally similar to botulinum toxin, cholera toxin, diphtheria toxin, and insulin.
3. Severe allergic reactions with anaphylaxis have been reported with handling of the seeds of castor bean and are also seen among workers in factories where castor oil is produced.
4. Castor bean plant is commercially cultivated as a source of castor oil. Such oil has been used for centuries as a purgative and as a lubricant for machines.



FIGURE 17.97 ■ **Castor Bean.** Typical appearance of the castor bean. (Photo contributor: Alex Wilson.)



FIGURE 17.98 ■ **Jequirity Pea.** Jequirity peas are also known as rosary peas, Indian beans, Buddhist's beads, crab's eyes, and prayer beads. They are about 5 mm in diameter and have a colorful glossy shell, usually red with a black center, although black and white may also be seen. (Photo contributor: Kevin J. Knoop, MD, MS.)

Clinical Summary

Levamisole is a levo enantiomer of tetramisole and has immunomodulatory properties. It was first used as an anthelminthic for human and veterinary use. Initial human use in the 1970s for inflammatory conditions resulted in publications of levamisole-associated agranulocytosis and vasculitis. In 2003, the United States Drug Enforcement Agency identified levamisole as an adulterant in cocaine. In 2009, case reports of agranulocytosis and vasculitis associated with levamisole-contaminated cocaine were published. The dermatologic manifestations may include retiform purpura with possible skin necrosis and tend to appear on the ears and nose but can affect any area.

Complications have been reported from both cocaine hydrochloride and crack cocaine and all routes of use. The reason for adulterating the cocaine with levamisole is not clear. Theories include that the levamisole enhances the effects of cocaine via one of several potential mechanisms: enhancing noradrenergic neurotransmission, inhibiting monoamine oxidase, inhibiting acetylcholinesterase, and stimulating ganglionic nicotinic receptors.

Management and Disposition

Initial considerations for the differential diagnosis for agranulocytosis or the vasculopathy should be broad. The xenobiotic exposure history for the patient must also be carefully reviewed, inclusive of drugs of abuse. For both the agranulocytosis and the cutaneous vasculopathy, serologic testing assists with the differential diagnosis; however, no one classic pattern is diagnostic for levamisole as the etiologic agent.

In the setting of agranulocytosis, neutropenic fever may occur and should be managed accordingly with antibiotics. Deaths from infectious complications have occurred. Cessation of exposure to the levamisole is imperative—which means cessation of cocaine use.

Pearls

1. Case reports of levamisole-associated complications suggest that there is a high level of recurrence of complications upon reexposure to levamisole-contaminated cocaine.



FIGURE 17.99 ■ **Levamisole-Induced Vasculitis (Ear).** The vasculitis associated with levamisole has a predilection for involving the ear. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 17.100 ■ **Levamisole-Induced Vasculitis (Nose).** Levamisole induced vasculitis can induce significant tissue necrosis requiring reconstructive grafts. (Photo contributor: R. Jason Thurman, MD.)

2. Based on case reports, the ears and nose tend to manifest the skin necrosis more often than other areas.
3. Urine drug screens usually check for the cocaine metabolite benzoylecgonine, which may be detectable in the urine for up to 3 days after cocaine exposure. Adulterants such as levamisole are not a part of the routine drug screens. Detection for levamisole requires additional techniques such as gas chromatography-mass spectrometry, liquid chromatography, or tandem mass spectrometry.



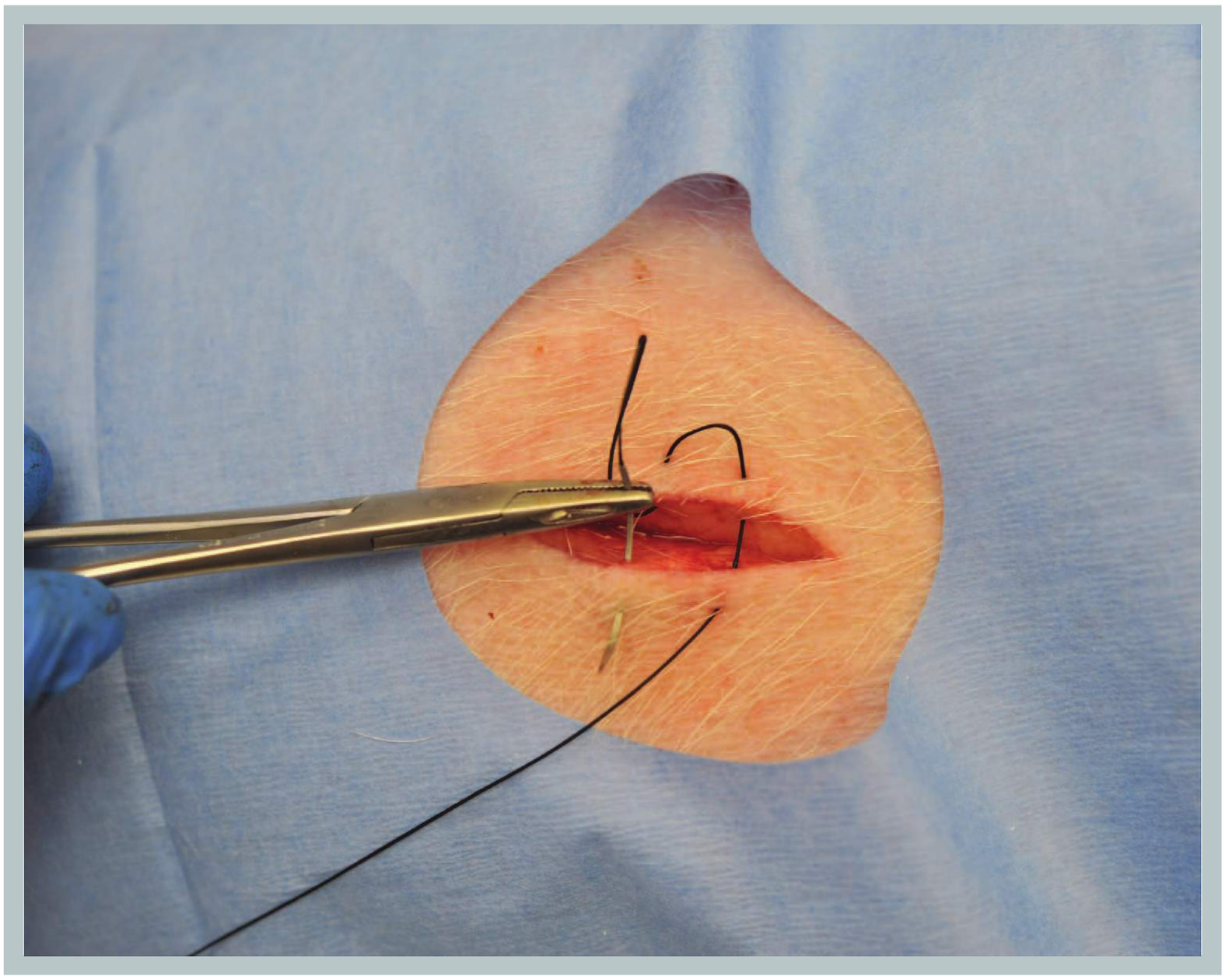
FIGURE 17.101 ■ **Levamisole-Induced Vasculitis.** Typical appearance of the purpuric lesions of levamisole-induced vasculitis. These lesions were acute and involved the leg of the patient in Fig. 17.100 who also had necrotic lesions from a previous case of levamisole-induced vasculitis. (Photo contributor: R. Jason Thurman, MD.)

Chapter 18

WOUNDS AND SOFT TISSUE INJURIES

Scan QR code or go to www.ematlas.com for video related to chapter

Chan W. Park
Michael L. Juliano
Dana Woodhall



Horizontal Mattress Suture. (Photo contributor: Michael L. Juliano, MD.)

The authors acknowledge Matthew D. Sztajnkrycer and Alexander T. Trott for portions of this chapter written for the second edition.

WOUND CLEANING AND IRRIGATION

Clinical Summary

The goals of minor wound care are to achieve optimal wound aesthetics and infection prevention. For most uncomplicated wounds, irrigation is the most effective means of reducing bacterial count. However, debridement may be necessary in contaminated wounds since devitalized tissue may impair the wound's ability to resist infection.

Management and Disposition

Preliminary wound management begins with assessment, adequate hemostasis, foreign-body removal, and irrigation. Provide adequate analgesia prior to wound cleansing. In simple well-vascularized wounds, tap water is as effective as normal saline or sterile water. In contaminated wounds, povidone-iodine diluted 1:10 with normal saline may help with disinfection. Bacterial-static solutions, such as nonionic surfactant cleaner, may also reduce bacterial inoculum. Solutions containing ionic detergents (eg, Betadine surgical scrub) should not be used as it is toxic to wound tissue. If necessary, wound scrubbing should be done gently to avoid damaging viable tissue.

Irrigation is the most effective means of reducing bacterial inoculum; 500 to 1000 mL of fluid or 60 mL/cm of wound length is adequate for most uncomplicated wounds. The recommended irrigation pressure of 5 to 8 lb per square inch (PSI) can be accomplished by attaching an 18- or 19-gauge intravenous (IV) catheter sheath, or a commercially available splash shield, to a 20- or 30-mL syringe. A bulb system is suboptimal; it generates only 0.5 to 1 PSI. Debris that cannot be irrigated can be scrubbed or sharply debrided. The tissue should appear pink and viable; a scant amount of fresh bleeding indicates good vascular supply. High-pressure irrigation (≥25 PSI) may be necessary in highly contaminated or complicated wounds requiring operating room washout. However, it offers no advantage for routine wounds.



FIGURE 18.1 ■ **Wound Soaking.** Soaking is an appropriate method for loosening debris and coagulated blood but is not a substitute for irrigation. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)

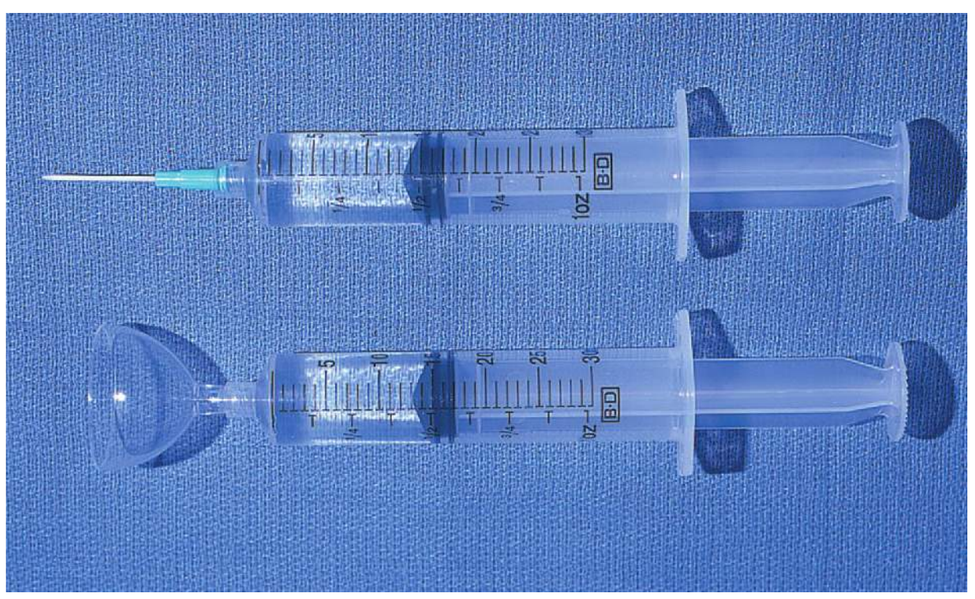


FIGURE 18.2 ■ **High-Pressure Irrigation Devices.** Ideal pressure for routine wound irrigation is 5 to 8 PSI. This can be achieved with a 30-mL syringe attached to an 18- or 19-gauge IV catheter sheath (top) or splash shield (bottom) (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)

Pearls

1. Universal precautions, including gloves and face shield, should always be observed.
2. Antibiotics are no substitute for thorough wound cleansing and irrigation.
3. Shaving the eyebrow for wound repair is contraindicated due to the unpredictable pattern of hair regeneration.

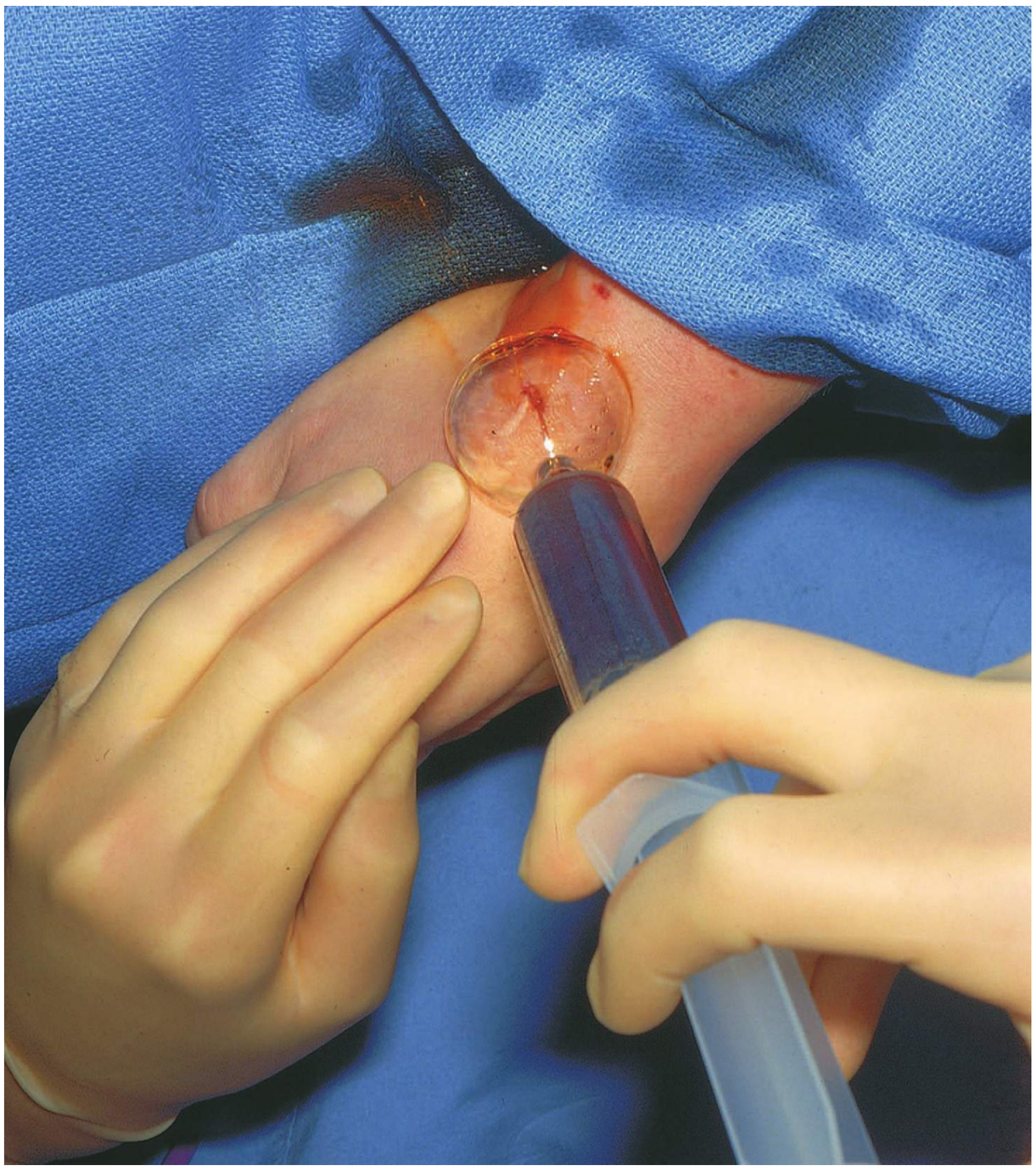


FIGURE 18.3 ■ **Wound Irrigation.** After adequate anesthesia, a wound is thoroughly irrigated. Even with the shield, there can be significant splatter and potential for body fluid exposure. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)

Clinical Summary

Proper wound assessment and preparation are essential to good wound management. Always consider the age and mechanism of injury, risk for contamination or foreign body, risk to the nerve, blood vessel and tendon, tetanus status, and identifying comorbid conditions that may affect wound healing.

Management and Disposition

Patient compliance is integral to achieving adequate wound exploration. The use of local or regional anesthesia is usually sufficient. However, procedural sedation may be required in some. Document neurovascular status prior to any anesthetic administration. Application of direct pressure is often the easiest way to achieve hemostasis. Other methods include the use of blood pressure cuff or tourniquet to achieve temporary hemostasis. Anesthetic solution containing epinephrine (1:100,000 dilution) may help constrict small vessels; however, particular caution should be exercised when using any vasoconstrictors in areas of end arterial circulation (eg, fingers, nose, toes, ears, and penis).

While hemostats or other self-restraining devices such as retractors may help achieve adequate exposure, care must be exercised to avoid damaging the dermis and the tissue's vascular integrity. If exposure is still not adequate despite hemostasis and separation, the wound margins may be slightly extended with fine iris scissors or scalpel to allow better visualization. The wound is extended from one end, through the epidermis and dermis only, to avoid further injury to underlying structures. Once the superficial fascia has been exposed, it may be carefully and bluntly dissected using forceps or scissors.

Pearls

1. Never probe a wound blindly or blindly attempt to control bleeding with hemostats.
2. At 1:100,000 dilution, epinephrine-containing anesthetics may be used with caution in areas of end arterial circulation such as the ear, fingers, nose, toes, and penis.
3. If epinephrine-induced tissue ischemia occurs, injection of phentolamine around the area of ischemia may help restore flow.

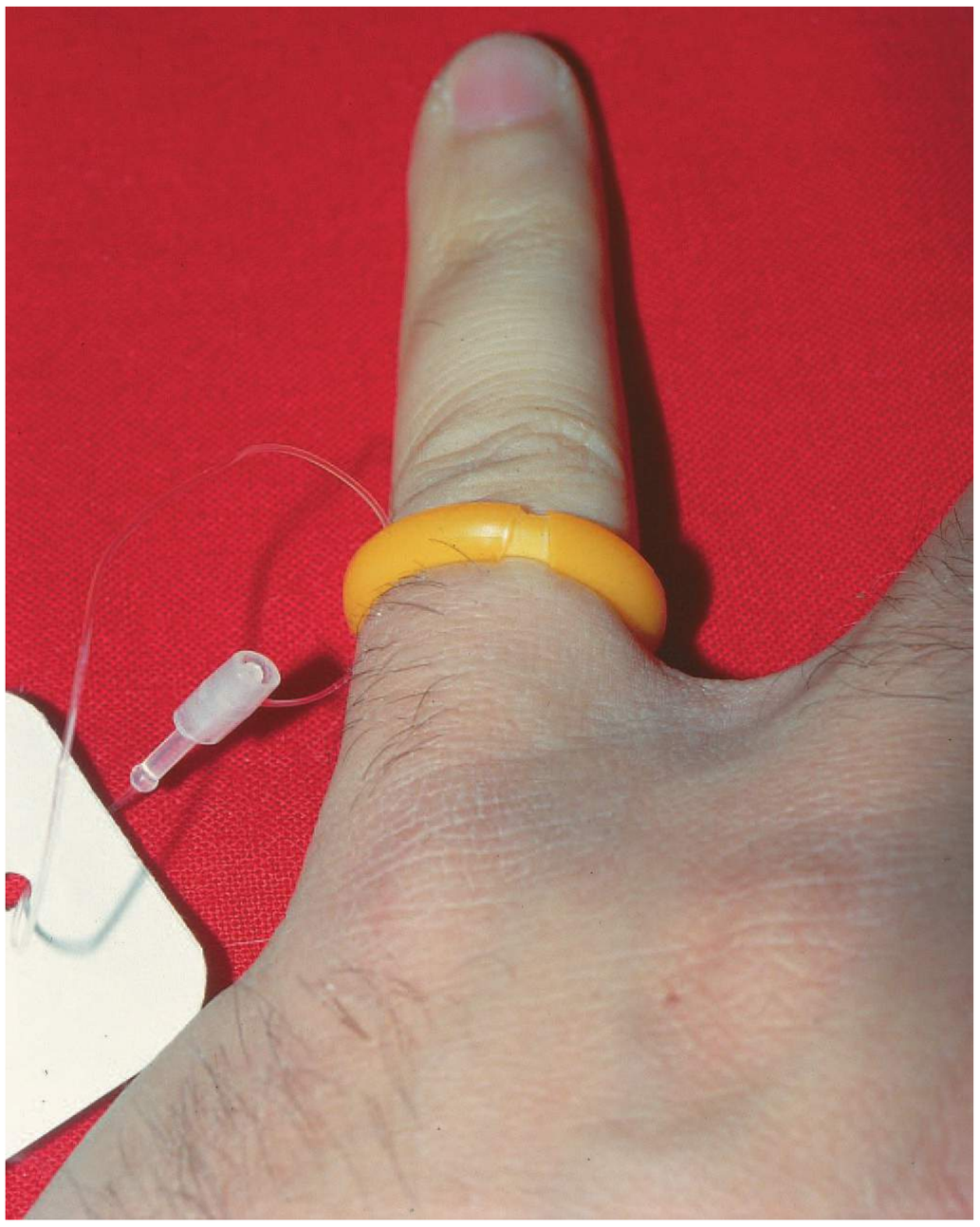


FIGURE 18.4 ■ **Finger Ring Tourniquet.** The ring tourniquet is an effective means of hemostasis. Removal after the procedure is important to prevent finger ischemia. Another effective method of hemostasis involves using a Penrose drain tightened with hemostats. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)



FIGURE 18.5 ■ **Epinephrine Injection.** Although epinephrine can be used to help achieve hemostasis, it should be used with caution on areas with poor collateral blood supply. Epinephrine has been injected into the right thumb which shows the pallor of finger ischemia. (Photo contributor: Selim Suner, MD, MS.)



FIGURE 18.6 ■ **Wound Exploration.** A blood pressure cuff is an alternative means to obtain hemostasis before wound assessment. (Photo contributor: Alan B. Storrow, MD.)

WOUND FOREIGN BODIES

Clinical Summary

All foreign bodies can become a nidus for delayed infection. All reasonable attempts should be made to remove them. Radiographic evaluation or ultrasound (see related chapter) may assist in locating foreign bodies not directly visualized. Foreign bodies are characterized as being either reactive (eg, organic materials such as wood, bone, and soil) or nonreactive (eg, glass and metal).

Management and Disposition

Patients are often unaware that a foreign body is present in their wound; a high level of clinical suspicion should accompany any injury pattern at risk for foreign-body penetration. Wounds at increased risk for foreign-body penetration include lacerations caused by broken glass, perioral injuries with loss of dentition, and injuries to the hands and feet involving needles, nails, or splinters.

Suspicion of a retained foreign body mandates local wound exploration and the consideration of radiographic or ultrasound evaluation. Nearly 80% of objects can be identified on plain radiographs. More specifically, approximately 90% of glass fragments greater than 2 mm in size can be identified through the use of plain radiographs; fragments as small as 0.5 mm can be identified in 50% to 60% of cases. In situation where plain radiographs are poor, ultrasound may be considered.

Due to their increased risk for delayed infection and poor wound healing, reactive material must be removed. Nonreactive objects, however, may be left in place if reasonable effort to remove it has been unsuccessful, and no potential for harm to a vital structure exists. Glass, however, has the potential for significant irritation and a removal attempt should be pursued.

Pearls

1. The base of the wound must be visualized as many foreign bodies hide there.
2. Common foreign bodies retained in hand wounds are wood splinters, glass fragments, metallic objects, and needles.
3. Missed retained foreign bodies are a very common source of litigation in emergency medicine.

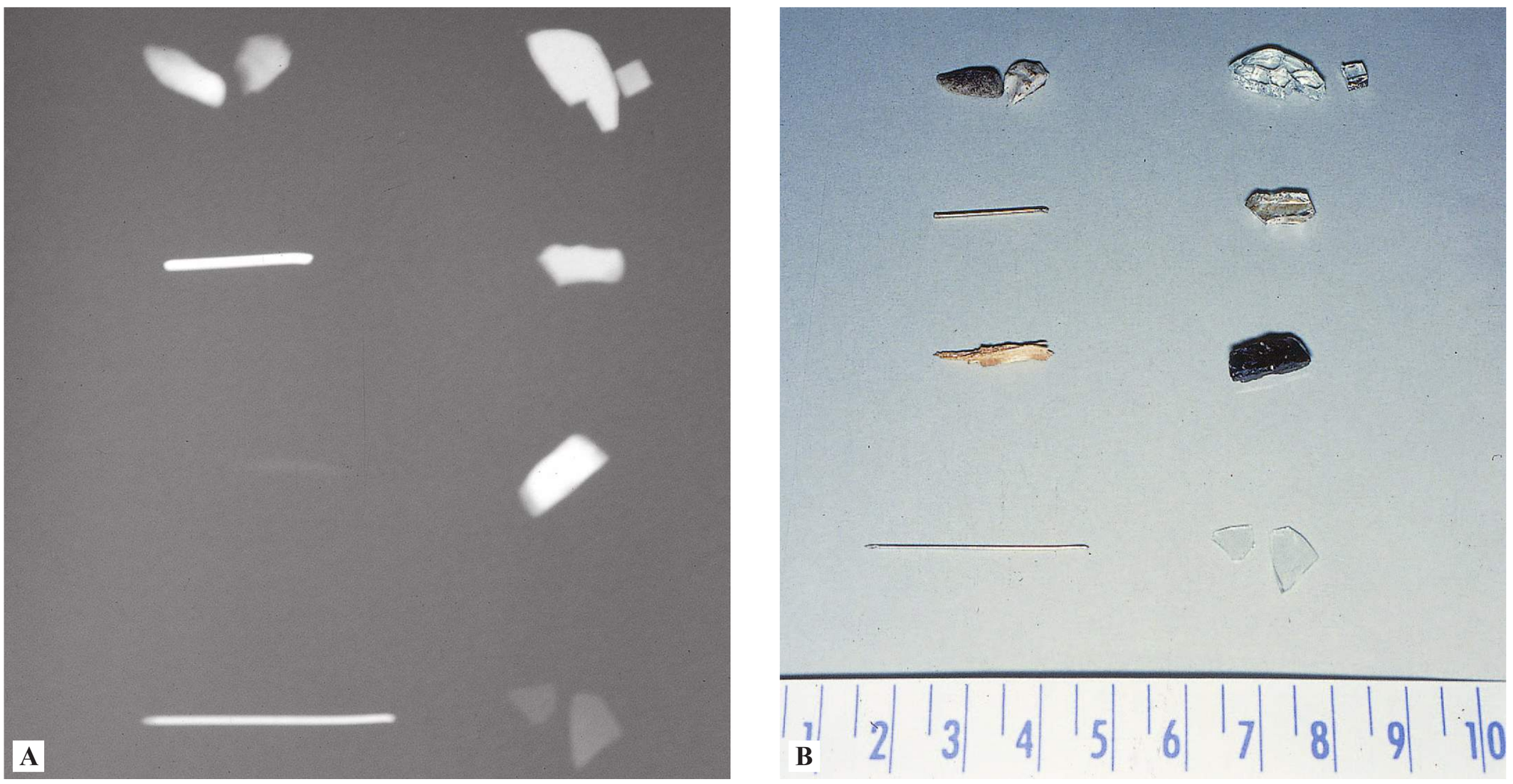


FIGURE 18.7 ■ **Radiodensity of Common Foreign Bodies.** The plain radiograph (**A**) demonstrates the radiodensity of common foreign bodies. Counterclockwise from top left: pebbles, paper clip fragment, wood splinter, hollow needle, light bulb glass, dark ("beer bottle") glass, transparent glass, and automobile windshield glass. Note that, although faint, the wood splinter is visible on plain radiography. Ruler markings (**B**) for the photograph of the corresponding objects are in centimeters. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)

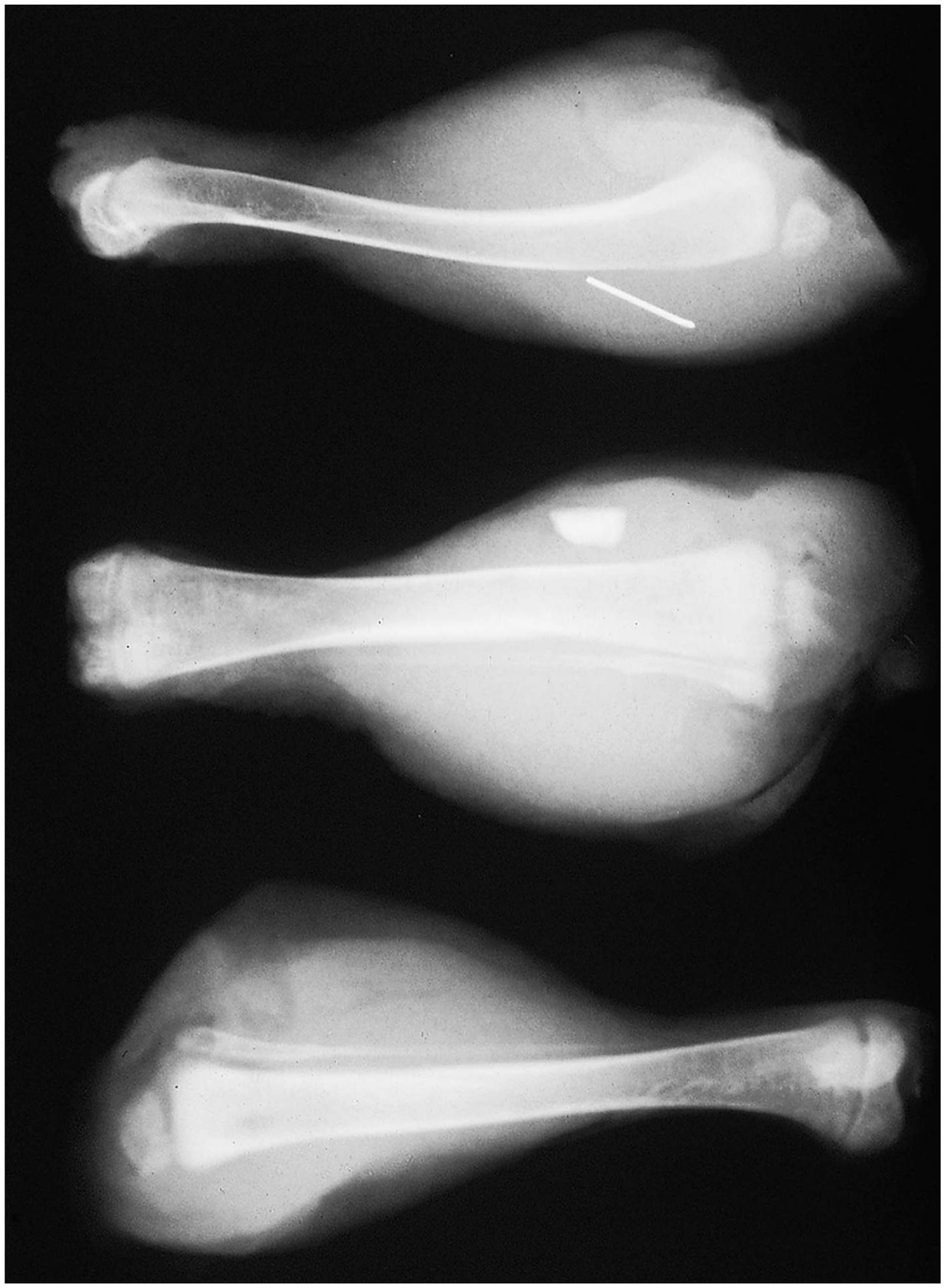


FIGURE 18.8 ■ **Radiodensity of Common Foreign Bodies in Tissues.** The paper clip, dark glass, and wood splinter (top to bottom) imaged in Fig.18.7 were inserted into chicken legs and radiographs taken. The wood splinter is no longer clearly visible within the soft tissue. For purposes of foreign-body localization, a minimum of two radiographic views at 90 degrees to one another are obtained, and the site of the foreign-body entry clearly marked. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)

Clinical Summary

Traumatic surface wounds are caused by one of three mechanisms: shearing, tension, or compression. Such a division helps to guide management decisions involving infection risk and scar formation.

Shearing injuries are caused by sharp objects, such as glass shards or knives, which impart low-energy injury and minimal tissue destruction. A majority of uncomplicated shearing injuries (ie, those not involving neurovascular or anatomically important structures) are repaired primarily in the emergency department (ED). The risk of infection is low and scar formation is typically cosmetically acceptable. Puncture wounds occur from sharp objects that pierce the skin and penetrate into deeper tissues. Such wounds are at a higher risk for infection, foreign-body retention, and underlying structural injury.

Tension or partial avulsion injuries occur when an object strikes the skin at a sharp angle creating a triangular flap. This results in potential vascular disruption, greater tissue destruction, and a higher risk for infection and tissue ischemia. During the repair, vascular supply to the flap must be meticulously preserved; otherwise, the flap may become ischemic.

Crush or compression injuries occur when a blunt object strikes tissue at a right angle, imparting a high degree of kinetic energy. This results in significant tissue destruction of the skin and its underlying supportive fascial layers. Crush injuries are typically ragged, with irregular wound edges and a complex laceration pattern. Despite meticulous wound care and careful primary closure, the resulting scars may be cosmetically poor.

Management and Disposition

First update the tetanus status of all patients requiring wound management. Carefully document the time and functional/neurovascular status at initial evaluation, as this may change over time. Upon optimizing wound preparation, appropriate closure tension must be achieved. If open fracture is suspected



FIGURE 18.9 ■ **Linear Laceration.** A large, uncomplicated, linear leg laceration. Given the depth and gaping, it can be sutured using a layered closure to remove surface tension at the wound edges and promote a more cosmetically acceptable outcome. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 18.10 ■ **Linear Laceration.** A long, linear shearing laceration involving the forehead and scalp, with exposed galea. The wound is explored and palpated for evidence of a depressed or open skull fracture. Closure of large galeal lacerations is recommended to prevent infection. Large frontal galeal lacerations are also repaired to prevent a cosmetic deformity of the frontalis muscle. (Photo contributor: Kevin J. Knoop, MD, MS.)

or confirmed, IV antibiotics are administered and orthopedic consultation obtained. Repair of traumatic wounds depends on the depth, complexity, and location. Deep wounds are closed in layers or by using a vertical mattress technique to remove dead space and relieve tension. Superficial wounds may be repaired with staples, simple interrupted sutures, or running sutures. In certain circumstances, the use of adhesive skin closures or adhesive glues may be adequate.

Pearls

1. Simple, uncontaminated, and uncomplicated wounds caused by clean, sharp objects in otherwise healthy patients may undergo primary closure up to 18 hours from the time of injury with good cosmetic result (see Simple Wound Closures).
2. The vascular supply to a flap is often tenuous; improper closure may further compromise the tissue, especially at the tip. A repair using a corner stitch will help minimize further ischemia (see Complex Wound Closures).
3. Crush injuries have an increased susceptibility to infection. Thorough cleansing, copious irrigation, and judicious debridement are required.



FIGURE 18.12 ■ **Puncture Wound.** A variety of linear objects may result in puncture wounds, in this case a bicycle brake. Radiographic evaluation is mandatory to assess for bone and joint involvement. (Photo contributor: Larry B. Mellick, MD, MS.)



FIGURE 18.11 ■ **Puncture Wound.** A puncture wound to the foot with a contaminated garden instrument. Tetanus status must be carefully addressed in such an injury. A radiograph of the foot demonstrated no associated bony injuries. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)



FIGURE 18.13 ■ **Partial Avulsion Injury.** A typical partial avulsion laceration from a fall onto the edge of a staircase. Note the triangular "flap" in the upper left wound quadrant. Closure of partial avulsion injuries must be particularly meticulous to reduce any further compromise of the flap tip's vascular supply. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 18.14 ■ **Lower Extremity Degloving Avulsion.** A complex degloving injury after entanglement of the lower extremity in a rope while water-skiing. (Photo contributor: Alan B. Storrow, MD.)





FIGURE 18.15 ■ **Ring Injuries.** Rings being caught and forced proximally are a common cause of finger degloving (**A**). Soft tissue injury due to tourniquet effect of a ring in an elderly woman with diabetic neuropathy (**B**). (Photo contributors: A. Selim Suner, MD, MS and B. Lawrence B. Stack, MD.)

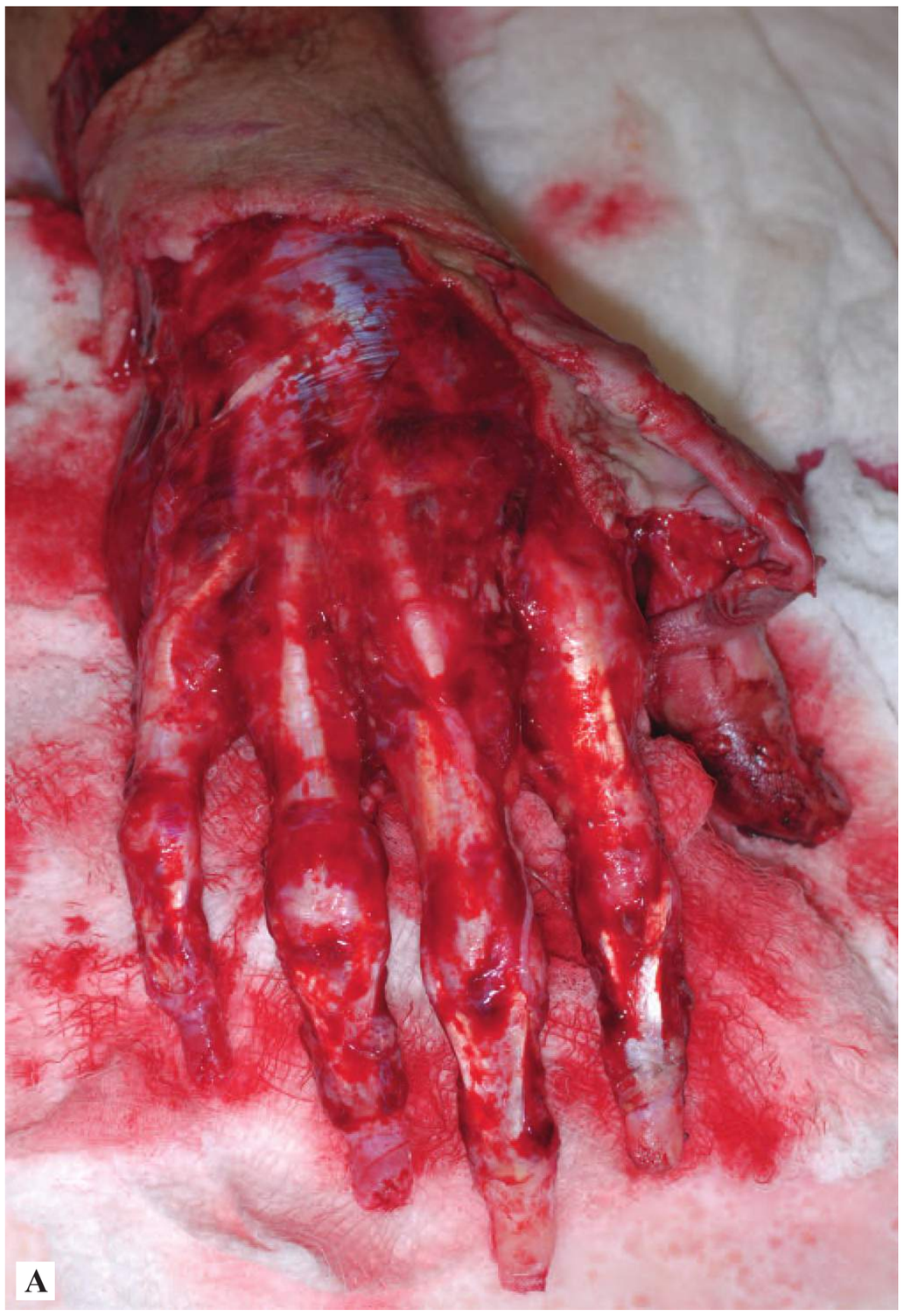




FIGURE 18.16 ■ **Hand Degloving Avulsion.** This extensive degloving (**A**) was caused by an industrial accident and resulted in near-complete removal of the skin of the hand (**B**). (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 18.17 ■ **Crush (Compression) Injury.** Extensive crush injury, with degloving, resulting from an industrial accident. (Photo contributor: David Effron, MD.)

TABLE 18.1 ■ GUIDE TO TETANUS PROPHYLAXIS IN ROUTINE WOUND MANAGEMENT

History of Adsorbed Tetanus Toxoid (Doses)	Clean Minor Wounds		All Other Wounds[a]	
	Tdap or Td[b]	TIG[c]	Tdap or Td[b]	TIG[c]
<3 or unknown	Yes	No	Yes	Yes
≥3 doses	No[d]	No	No[e]	No

[a]Such as (but not limited to) wounds contaminated with dirt, feces, soil, and saliva; puncture wounds; avulsions; and wounds resulting from missiles, crushing, burns, and frostbite.

[b]For children younger than 7 years of age, DTaP is recommended; if pertussis vaccine is contraindicated, DT is given. For persons 7 to 9 years of age, Td is recommended. For persons >10 years, Tdap is preferred to Td if the patient has never received Tdap and has no contraindication to pertussis vaccine. For persons 7 years of age or older, if Tdap is not available or not indicated because of age, Td is preferred to TT.

[c]TIG is human tetanus immune globulin. Equine tetanus antitoxin should be used when TIG is not available. If only three doses of fluid toxoid have been received, a fourth dose of toxoid, preferably an adsorbed toxoid, should be given. Although licensed, fluid tetanus toxoid is rarely used.

[d]Yes, if it has been 10 years or longer since the last dose.

[e]Yes, if it has been 5 years or longer since the last dose. More frequent boosters are not needed and can accentuate side effects.

Source: Table courtesy of the Centers for Disease Control and Prevention. Please see www.cdc.gov for the most current information.

EAR LACERATIONS

Clinical Summary

The ear is composed of a poorly vascularized cartilaginous skeleton covered by tightly adherent skin. Given the paucity of subcutaneous tissue, ear injury that results in hematoma formation can cause pressure necrosis of the cartilage. The goal of repairing an ear laceration involves completely covering the exposed cartilage and preventing hematoma formation.

Management and Disposition

Prior to repair, the area is examined for signs of acute hematoma formation or other associated traumatic injuries. Hemotympanum or Battle sign suggests the presence of a more serious closed head injury, especially basilar skull fracture. Blunt trauma may result in barotrauma resulting in tympanic membrane perforation. Examination can be facilitated by local anesthesia infiltration or, in the case of larger or more complex lacerations, a regional nerve block.

Simple lacerations through the earlobe or the helix can be repaired with interrupted 6-0 nonabsorbable monofilament sutures if the cartilage is not exposed. Simple lacerations that involve the cartilage are primarily repaired by ensuring complete coverage of the exposed cartilage by careful apposition of the overlying skin. The skin generally provides sufficient support, so sutures are not required for the cartilage itself. If the wound is sufficiently irregular and cartilage debridement becomes necessary to avoid undue wound tension, the debridement should be kept to a minimum.

A perichondral hematoma must be drained within 72 hours to prevent potential pressure necrosis which can result in a "cauliflower" ear. Ear wounds are best dressed with a mastoid pressure dressing either primarily or after later hematoma drainage. Such a dressing reduces the chances for future hematoma formation and its complications. Ear sutures are removed in 4 to 5 days.

Pearls

1. Epinephrine-containing anesthetic at a 1:100,000 dilution may be used in ear laceration repair.
2. Hematoma evacuation needs to be rechecked in 24 hours to evaluate for reaccumulation.
3. If cartilage has been exposed or a hematoma drained, antistaphylococcal antibiotic coverage is recommended.
4. Complex lacerations and hematomas of the ear are best cared for in conjunction with a consultant.



FIGURE 18.18 ■ **Ear Laceration.** This patient sustained an uncomplicated, linear pinna laceration. Closure must cover all exposed cartilage. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 18.19 ■ **Ear Laceration.** Complex ear laceration through the helix down through the antihelix. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Lip lacerations may result in significant cosmetic defects if not properly repaired. The lip has two significant anatomic landmarks: the mucosal border, which divides intraoral and external portions of the lip, and the vermilion border, which separates the lip mucosa from the skin of the face. Meticulous alignment of the vermilion border and its associated "white line" is the cornerstone of cosmetic repair. Lip anatomy may be distorted by the kinetic energy of the impact as well as the resultant edema surrounding the wound. Lacerations of the lip's vermilion border may be partial- or full-thickness, compromising the underlying orbicularis oris.

Management and Disposition

Given the high bacterial content of the oral cavity, lip lacerations will not remain clean during repair. The goal of irrigation is to remove clotted blood and gross contaminants such as tooth fragments or dirt. If a fractured tooth is noted, the wound must be explored for fragments. If the tooth or fragment is unaccounted for, then a Panorex or soft tissue radiograph of the face and a chest radiograph should be obtained. Anesthesia for laceration repair is best performed using either an infraorbital (upper lip) or mental (lower lip) nerve block since local infiltration often distorts the tissue and impairs proper alignment of the vermilion border.

If the vermilion border is violated by a superficial laceration, then the first suture, typically 6-0 in size, is placed at the border to reestablish anatomic relationships. Once alignment is judged adequate, simple interrupted sutures are used for completion. If the laceration extends within the oral cavity, absorbable 5-0 sutures are used to close the intraoral component.

With deep or "through and through" lacerations involving the orbicularis oris, the muscle layers are initially approximated with deep, usually 5-0, absorbable sutures. Once the muscle is approximated, the first skin suture is again placed at the vermilion border.

Sutures are removed in 3 to 5 days in children, 4 to 5 days in adults. The patient is advised to eat soft foods, not to apply excessive force to the suture line, and to rinse after eating to prevent the accumulation of food particles.

Pearls

1. Misalignment of the vermilion border by as little as 1 mm may result in a cosmetically noticeable defect.
2. A marking pen may be used to identify landmarks prior to placing the sutures, as suturing itself causes some tissue edema, bleeding, and distortion.
3. Any patient with a lip laceration requires a thorough inspection of the oral cavity for associated trauma, including dental fractures, oral lacerations, and mandibular injuries.



FIGURE 18.20 ■ **Vermilion Border Lacerations.** A complex and a simple vermillion border laceration. (Photo contributor: David Effron, MD.)

TENDON LACERATIONS

Clinical Summary

Tendon injuries are often associated with hand or wrist lacerations. Accurate assessment requires documentation of both motor function and strength. Partial tendon ruptures, including near complete, may still result in normal function.

Management and Disposition

Prior to wound exploration, a thorough exam is performed to assess neurovascular and motor function. All individual flexor and extensor tendons are assessed, including deep and superficial. Abnormal resting posture of the involved extremity can also indicate tendon injury. Tendons are taken through a full range of motion, including re-creation of limb position at the time of insult, in order to detect injuries along the tendon length. Adequate exploration requires excellent hemostasis achieved through direct pressure, or use of a blood pressure cuff or other tourniquet. Initial wound care should include irrigation, exploration for foreign bodies, debridement, antibiotics, and tetanus prophylaxis if indicated.

Partial tendon lacerations are treated conservatively, with splinting in neutral position and follow-up. Isolated extensor tendon lacerations may be repaired in the ED with subsequent specialist follow-up. Flexor tendon lacerations generally require consultation with a hand surgeon or orthopedic surgeon.

Pearls

1. While extensor tendon repair may be accomplished by an emergency care physician with appropriate training and experience, flexor tendon lacerations are a challenging orthopedic problem and require referral.
2. Flexor tendons are weakest approximately 3 weeks after repair.
3. Inability to flex the distal phalanx with intact proximal phalanx extension suggests a flexor digitorum profundus disruption.



FIGURE 18.21 ■ **Flexor Tendon Laceration.** This patient presented after sustaining a laceration to his third and fourth digits (left). The injury was associated with an inability to flex these two digits. Wound exploration revealed the distal segment of the transected flexor tendon apparatus (right). (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)





FIGURE 18.22 ■ **Extensor Tendon Lacerations.** Note the laceration over the third and fourth metacarpals (**A**). Inability to extend the long fingers is evidence of complete extensor tendon disruption. Another extensor tendon laceration associated with inability to extend the fifth digit. (**B**). (Photo contributors: A. Selim Suner, MD, MS and B. David Effron, MD.)



FIGURE 18.23 ■ **Flexor Tendon Laceration.** Wrist laceration and associated complete tendon rupture. (Photo contributor: David Effron, MD.)

ANIMAL BITE WOUNDS

Clinical Summary

Bite wounds (abrasions, lacerations, and punctures) are a frequent emergency care challenge, although most are minor. Dog bites account for the vast majority, followed by cats, humans, and, rarely, other animals (raccoons, foxes, livestock, etc).

Bite wound microbiology is frequently polymicrobial with mixed aerobic and anaerobic bacteria. Pasteurella multocida (a gram-negative anaerobe) has been cultured from up to 80% of cat bites and 25% of dog bites. Eikenella corrodens has been recovered from human bites. Capnocytophaga canimorsus (formerly known as DF-2), a virulent organism which can be normal oral flora in dogs (16%) and cats (18%), can lead to sepsis, disseminated intravascular coagulation, gangrene, or death in susceptible individuals (eg, immunocompromised, asplenic, or diabetic).

The primary site of a bite wound in adults is on the extremities. The hand is at highest risk for developing infection, while the face is the most infection-resistant. Puncture wounds, especially from cats, are also prone to infection. Simple abrasions, regardless of animal, are unlikely to become infected with proper wound care.

Management and Disposition

Management depends on location, animal involved, time elapsed before presentation, and wound type. All wounds should be thoroughly cleaned and debrided. Radiographs should be obtained to exclude bony injury or retained dentition. Contusions and superficial abrasions can be treated with local wound care.

Recommendations vary regarding timing of wound closure. Closure of facial and head wounds can be performed up to 12 hours (and in some reports up to 24 hours). Puncture wounds (especially if caused by a cat), hand lacerations, or high-risk wounds (wounds > 12 hours or clinically infected) should be considered for delayed primary closure. While a linear incision over a puncture wound may facilitate cleaning and exploration, efficacy of this is questioned. Human bites to areas other than the face and head should generally be left open and considered for delayed primary closure. Hand injuries caused by human bites are left open and managed in consultation with a hand specialist (see Chapter 11, Extremity Trauma, Clenched Fist Injury). One should maintain a high index of suspicion for human bite in any laceration near the metacarpals.

All sutured bite wounds should be reevaluated by a healthcare provider within 24 hours. Closed wounds which appear infected on reevaluation should be opened, irrigated, and allowed to close by secondary intention. Cyanoacrylate adhesives should never be used to close a bite wound.

Antibiotic therapy recommendations for bite wounds differ widely. Antibiotics are not recommended for minor wounds. They are recommended for cat bites, hand wounds, and in persons with chronic diseases (eg, diabetes). Empiric therapy is started with broad-spectrum antibiotics such as ampicillin-sulbactam, cefoxitin, or ceftriaxone. Alternatively, ciprofloxacin (or trimethoprim-sulfamethoxazole in children) and clindamycin can be used. Infection by P multocida classically becomes apparent within 24 hours of the bite and is marked by prominent pain, erythema, and swelling.



FIGURE 18.24 ■ **Dog Bite.** Extensive facial wounds secondary to a dog bite. (Photo contributor: David Effron, MD.)



FIGURE 18.25 ■ **Dog Bite.** This patient sustained multiple avulsion injuries to her hand and forearm from a pit bull attack. (Photo contributor: Selim Suner, MD, MS.)

Amoxicillin-clavulanate is the suggested regimen to cover polymicrobial infections and infections caused by P multocida or C canimorsus. Cultures are not recommended for initial treatment. However, they should be obtained in any wound worsening during antibiotic therapy. Tetanus immunization should be appropriately updated.

Rabies infection is rare in the United States; worldwide, dogs are the number one reservoir (see Chapter 21, Tropical Medicine, Rabies). Carnivorous animals (eg, raccoons, skunks, foxes, dogs, or bats) are more likely to be infected. If an animal is suspected as rabid, it should be detained and observed for any signs of rabies; occasionally this requires euthanasia and necropsy. Bats require special attention as many people do not recall being bitten. Healthcare professionals should have a low threshold for instituting rabies prophylaxis in persons with a bat exposure or in whom the animal is not available. Local animal control offices can be contacted for recommendations on appropriate rabies prophylaxis measures.

Pearls

1. All patients with bite wounds from susceptible animals (especially bats) should be assessed for rabies exposure.
2. Given cosmetic concerns and low infection risk, dog bites to the head and face can generally be sutured up to 24 hours after appropriate irrigation.
3. Bites which become infected within 24 hours, especially from cats, should be assumed to be caused by P multocida.



FIGURE 18.26 ■ **Cat Bite.** Note the swelling and erythema to the index finger indicating infection, possibly with P multocida (a prominent and fastidious organism seen commonly with feline bites). (Photo contributor: Kevin J. Knoop, MD, MS.)

Clinical Summary

Fishhook barbs often prevent backing the hook out of the puncture site. Several different methods have been described for removal.

Management and Disposition

Adequate anesthesia, usually local, is essential for removal. Procedural sedation may be needed if a child has a fishhook embedded in a sensitive area (eg, eyelid).

The method used to remove the hook depends primarily on the location of the barb relative to the skin surface and the body part embedded. The most common removal technique is the "push-through and cut." This is recommended when the tip of the fishhook is close to breaking through the skin surface after being embedded (see figures). Care should be taken when performing this maneuver in the hand or face as pushing the fishhook forward may damage nearby structures.

Superficially embedded hooks or hooks with small barbs may be removed in a retrograde fashion, by exerting pressure on the fishhook shaft toward the barb and backing the hook out through the original site of penetration. This technique can be performed manually or with the use of a string.

Once fishhooks are removed, the wound should be cleaned, irrigated, and left open. Antibiotics are usually not necessary; however, treatment (doxycycline) for Vibrio species (especially Vibrio parahaemolyticus) should be considered in wounds contaminated with saltwater.

Pearls

1. Hooks embedded in cartilaginous structures, such as the ear or nose, are best managed with the push-through methods.
2. Hooks that penetrate joint spaces or bone should be managed in consultation with orthopedics.
3. Fishhooks that penetrate the globe of the eye are left in place and emergent ophthalmologic consultation is obtained. The patient is placed in the semirecumbent position and the globe is protected with an eye shield. Pressure patches are contraindicated, as they may extrude intraocular contents.

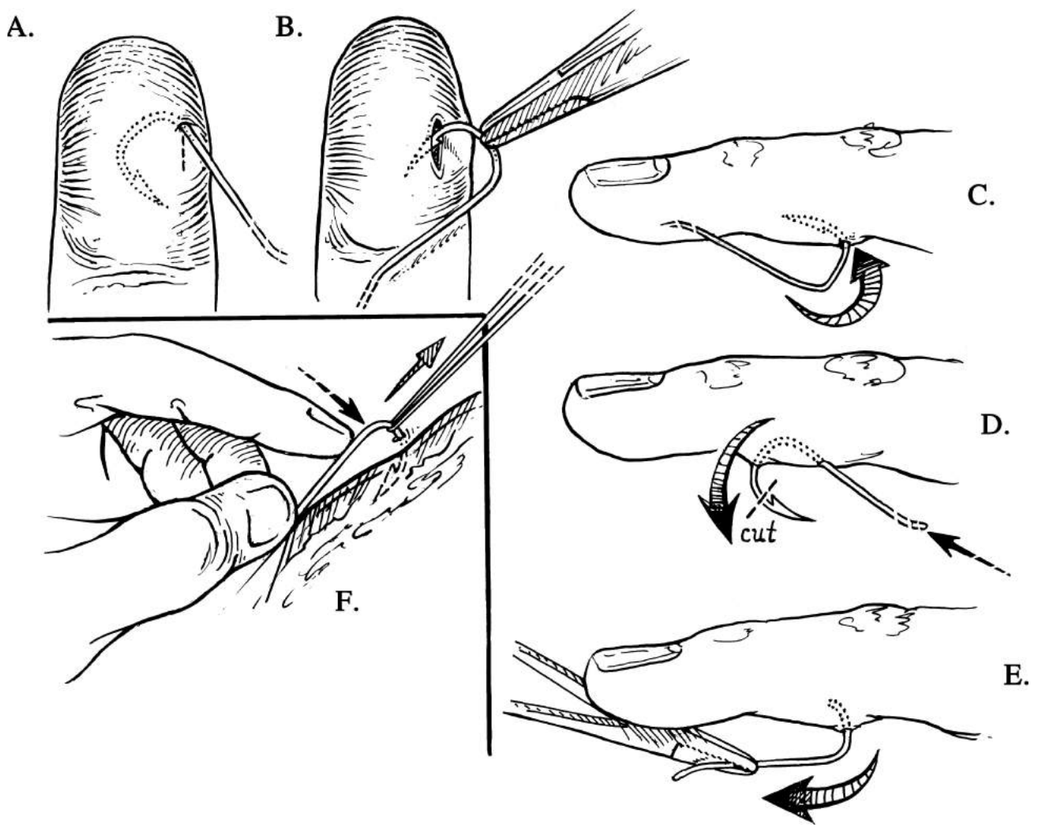


FIGURE 18.27 ■ **Fishhook Removal.** Hooks with small superficially embedded barbs may be carefully backed out through the original puncture site (**A** and **B**). This may require a small incision, made in line with the concavity of the hook curve. The push-through technique is useful for large barbs or those more deeply embedded. The hook is pushed out through the skin, the barb removed, and the remainder of the hook subsequently removed through the original penetration site (**C, D,** and **E**). The traction (string) technique provides an alternative for removing hooks with small barbs. While pressing down on the shaft of the hook, traction is applied with 0 silk or umbilical tape. A swift yank of the cord in the direction opposite the barb will dislodge the hook (**F**). Care is taken to warn bystanders of the potential for the fishhook to fly across the room.



FIGURE 18.28 ■ **Multiple Pronged Fishhook.** Care must be taken when removing this three-pronged hook due to the multiple sharp tips and potential for damaging surrounding structures. (Photo contributor: Selim Suner, MD, MS.)



FIGURE 18.29 ■ **Fishhook Injury and Removal**. The push-through technique was used to remove this hook. Use of a ring cutter proved unsuccessful; a bolt cutter was eventually required to remove the distal portion of this large hook. (Photo contributor: Alan B. Storrow, MD.)

SIMPLE WOUND CLOSURES

Clinical Summary

Most ED lacerations are uncomplicated and can be repaired primarily. Complications (dehiscence, infection, improper healing, scarring) can be minimized by proper suturing techniques, including good wound edge approximation and decreasing wound tension. Wound edge eversion is also essential for proper healing as the edge will flatten with time.

Management and Disposition

Suture techniques must be individualized depending on physician's skill, laceration type, and location. Suture material, size, and duration before removal are determined by anatomic site (Table 18.2).

Deep interrupted absorbable sutures are used, if necessary, to reduce wound tension before superficial repair. Start the deep suture from the bottom of the wound, continue across the top (subdermally), and return to the bottom in finishing. This will leave the knot at the bottom of the wound, decreasing the chance suture material will exit at the surface.

Simple interrupted closures involve single nonabsorbable sutures, each independently tied. The needle should penetrate the skin at 90 degrees and exit the other side of the wound at 90 degrees.

Staples are a rapid means of closing linear wounds. They should not be used on the hands, feet, face, or over joints. Wound edge eversion is also critical to obtain the best outcome.

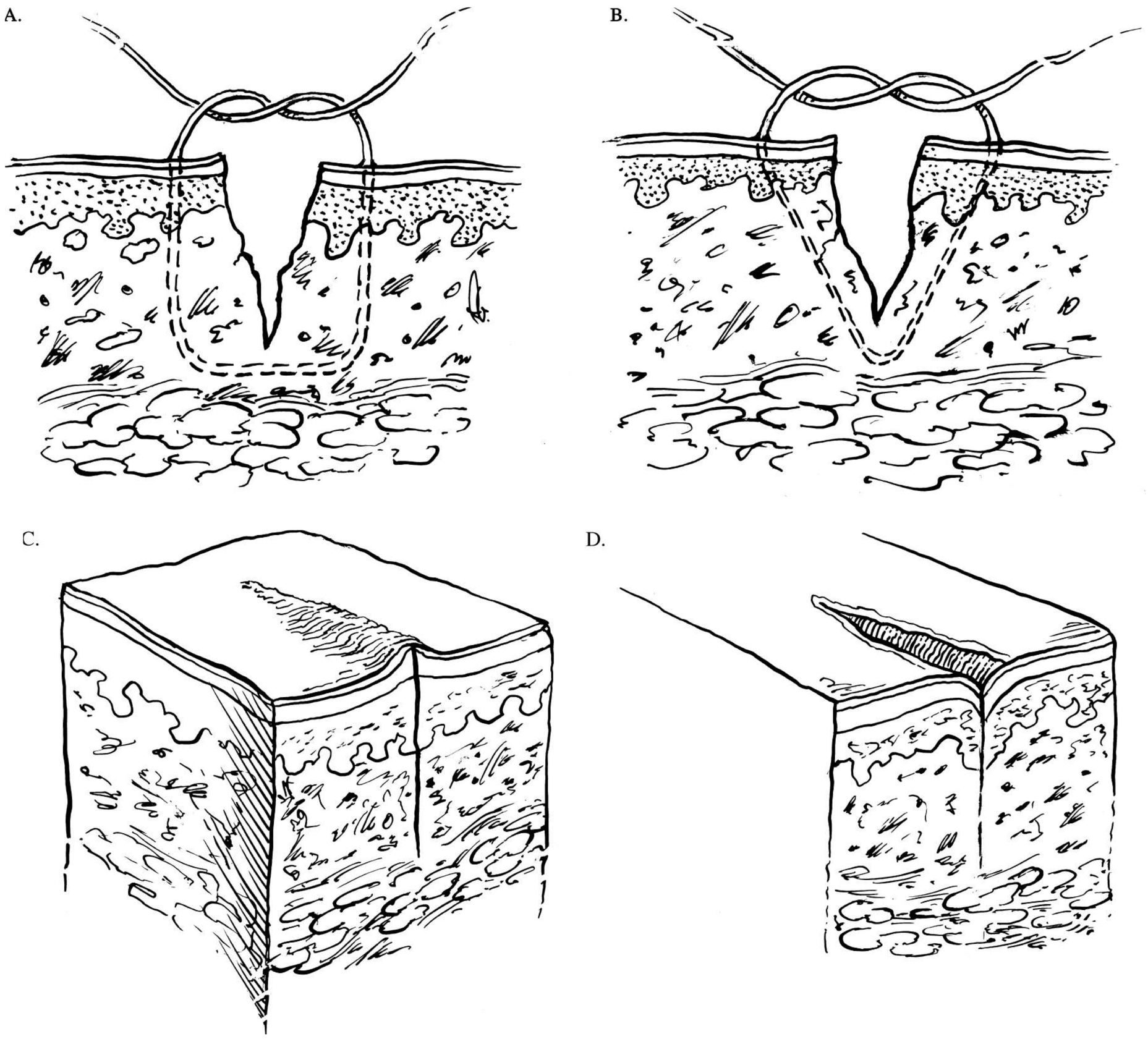


FIGURE 18.30 ■ **Wound Edge Eversion.** Eversion of wound edges is critical for optimal wound healing. For proper eversion, the needle point should enter the epidermis at a 90-degree angle, generating a square or bottle-shaped suture configuration (**A**). This results in a slight rise of the skin edges above the skin plane (**B**). Such eversion will flatten at the level of the skin plane during healing. Entry at a shallower angle (**C**) often leads to wound edge inversion, eventual contraction of the wound edges below the skin plane, and subsequent scar formation (**D**).

Running closure is a rapid technique using several bites along the length of a wound without tying individual knots. Knots are tied only at the beginning and end.

Liquid wound adhesives (eg, cyanoacrylate) have advantages due to speed and lack of a repeat visit for suture removal. Anesthesia may not be required. Small linear lacerations are ideal. The wound must be dry and free of active bleeding for proper adhesion. Antibiotic ointment should not be applied as this will dissolve the adhesive.

Elastic wound closure strips have the same advantages and indications as adhesives. They can be used alone or in conjunction with a tissue adhesive (benzoin resin) applied to the skin on either side of the wound. The edges will curl up over time and can be trimmed by the patient as needed, until they fall off in 2 to 3 weeks. They can also be used after suture removal to give the wound more time to gain tensile strength. Wound closure strips are not recommended in children as they have a tendency to remove them prematurely.

Pearls

1. The bites on both sides of a wound should be equidistant for both optimum wound healing and cosmetic outcome.
2. In gaping wounds, surface tension should be reduced with deep sutures.
3. Antibiotic ointment may be used to remove the adhesive from areas unintentionally glued together by cyanoacrylate.

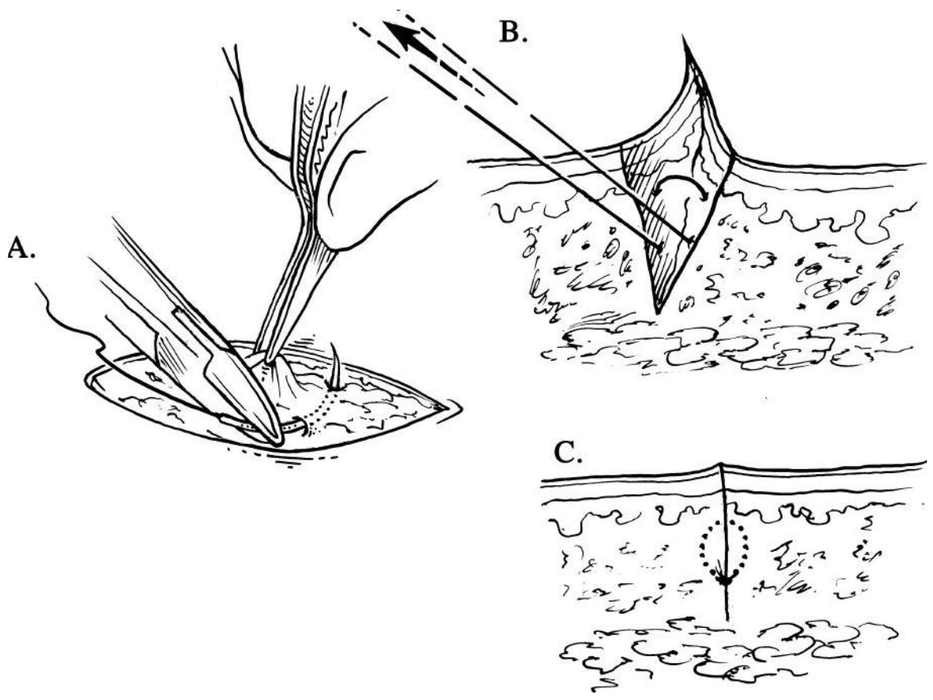


FIGURE 18.31 ■ **Deep Sutures.** Judicious placement of deep sutures allows approximation of the dermis, reduces tension on the wound edges, and may facilitate final superficial closure. The needle is driven from deep within the wound to a superficial level (**A**). On the opposite side of the wound, the needle is driven from superficial to deep (**B**). By having the leading and trailing suture come out on the deep and same side of the superficial cross suture, the knot is buried within the wound (**C**).



FIGURE 18.32 ■ **Simple Interrupted Wound Closure.** An uncomplicated linear laceration generated by a sharp object. For anesthesia and hemostasis, the wound edges are infiltrated with lidocaine-containing epinephrine. The wound is subsequently closed with simple interrupted sutures. Attention is paid to obtaining a degree of wound edge eversion. (Photo contributor: Alan B. Storrow, MD.)

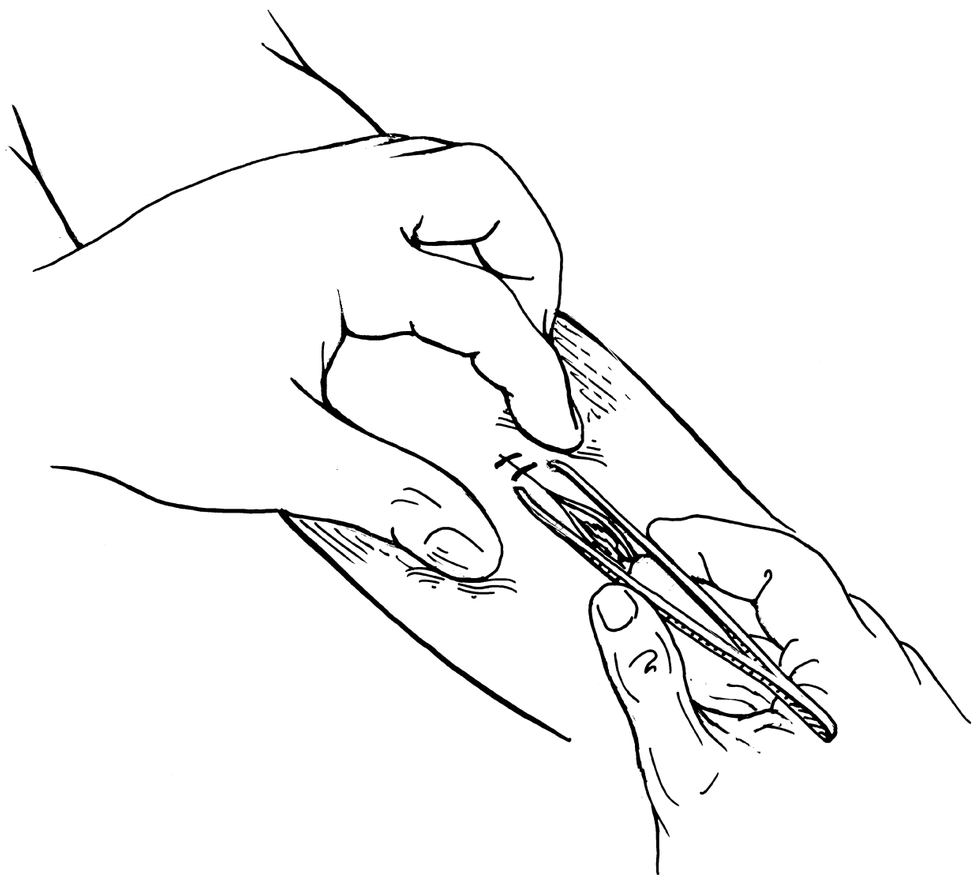


FIGURE 18.33 ■ **Staple Closure.** Meticulous care must be taken when using staples to properly approximate and evert wound edges. This can be facilitated through the use of forceps during staple closure.

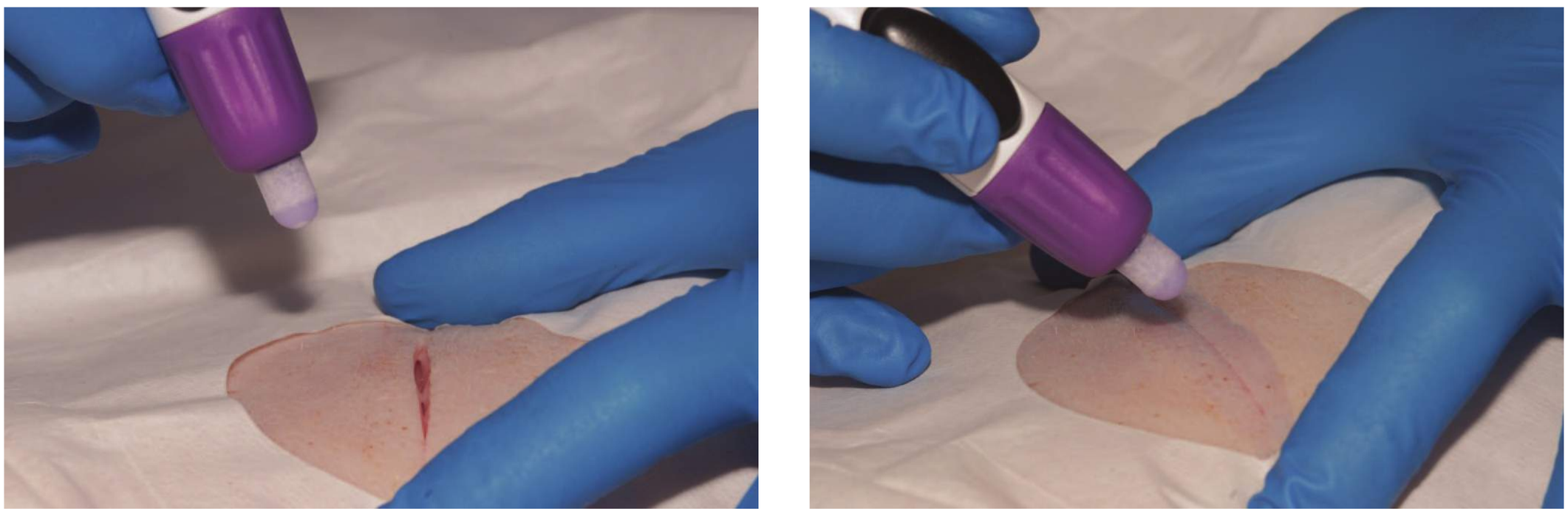


FIGURE 18.34 ■ **Liquid Wound Adhesive Closure.** Wounds that are ideal for this type of closure are small, linear, and hemostatic. Care must be taken to ensure the wound edges are correctly approximated before applying the adhesive. (Photo contributor: Michael L. Juliano, MD.)

TABLE 18.2 ■ SUTURE MATERIALS, SIZE, AND DURATION BY ANATOMIC SITE

Anatomic Site	Skin	Deep	Duration
Scalp	5-0, 4-0 monofilament	4-0 absorbable	6-8 days
Ear	6-0 monofilament	N/A	4-5 days
Eyelid	7-0, 6-0 monofilament	N/A	4-5 days
Eyebrow	6-0, 5-0 monofilament	5-0 absorbable	4-5 days
Nose	6-0 monofilament	5-0 absorbable	4-5 days
Lip	6-0 monofilament	5-0 absorbable	4-5 days
Oral mucosa	N/A[a]	5-0 absorbable	N/A
Face/forehead	6-0 monofilament	5-0 absorbable	4-5 days
Chest/abdomen	5-0, 4-0 monofilament	3-0 absorbable	8-10 days
Back	5-0, 4-0 monofilament	3-0 absorbable	12-14 days
Arm/leg	5-0, 4-0 monofilament	4-0 absorbable	8-10 days
Hand	5-0 monofilament	5-0 absorbable	8-10 days[b]
Extensor tendon	4-0 monofilament	N/A	N/A
Foot/sole	4-0, 3-0 monofilament	4-0 absorbable	12-14 days

[a]Not applicable.
[b]Add 2 to 3 days for joint extensor surfaces.
Source: Adapted with permission from Trott AT. Wounds and Lacerations: Emergency Care and Closure. 2nd ed. St. Louis, MO: Mosby-Year Book; 1997.

Clinical Summary

Horizontal and vertical mattress sutures, as well as the corner stitch (see figures for technique), may be used to manage wounds unable to be closed with simple techniques.

Management and Disposition

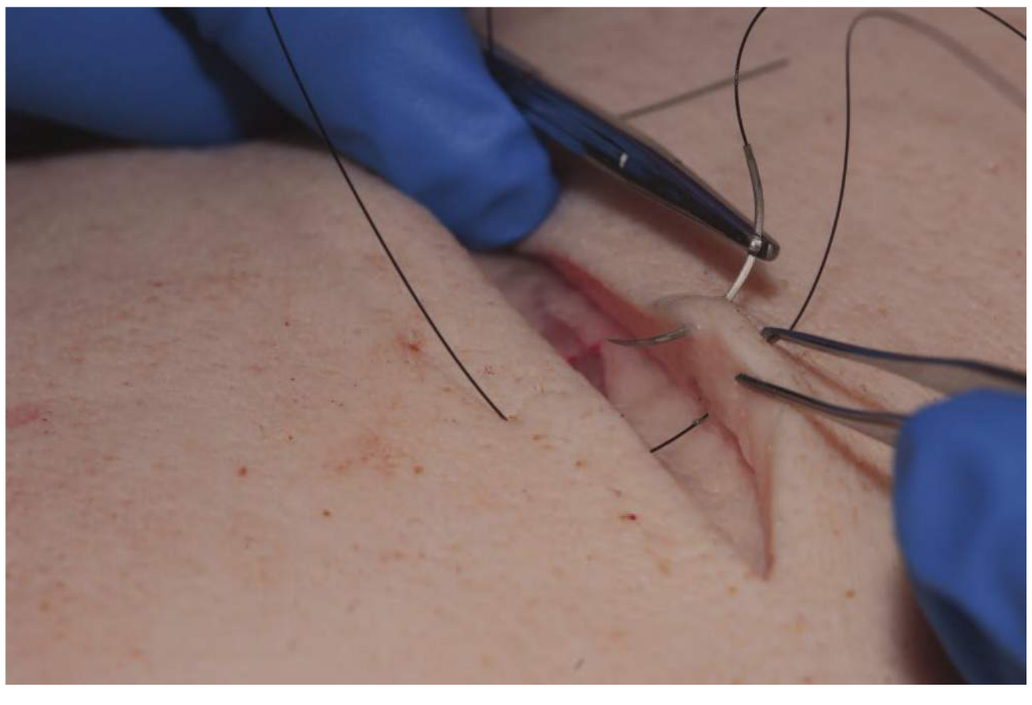
Vertical mattress suture is a useful technique for deep wounds. It reduces wound tension by acting as both a deep and superficial suture.

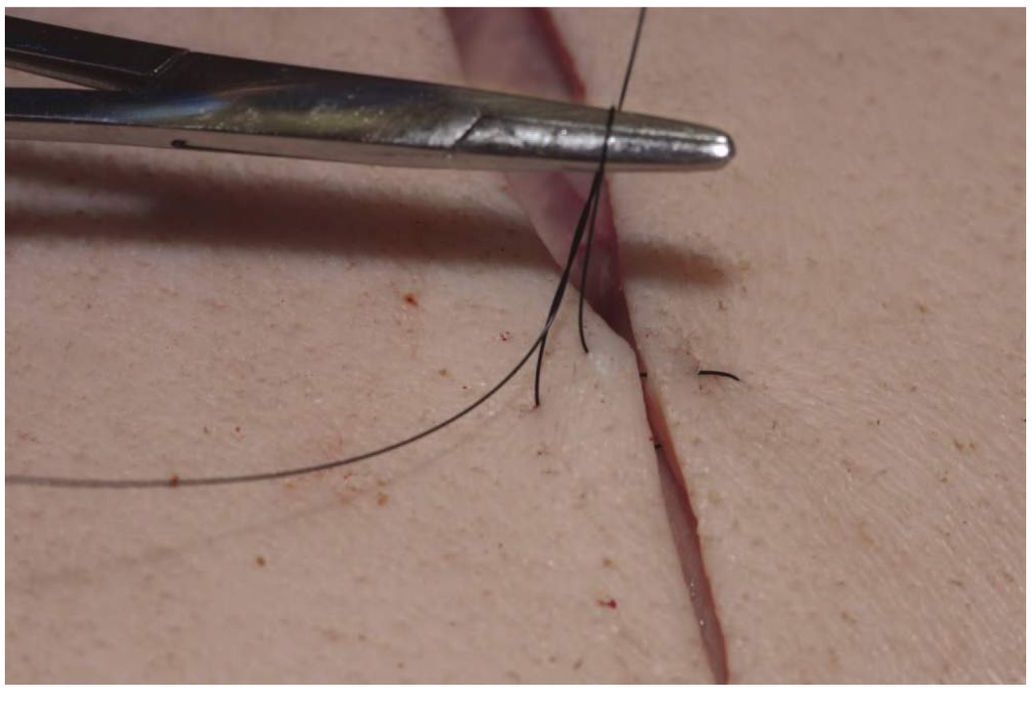
Horizontal mattress suture is best used for wide, gaping wounds with a risk of increased wound tension after closure (eg, lacerations overlying a joint).

Corner stitch is used to close triangular wounds or flaps. A simple interrupted suture cannot be placed to approximate the point of the flap due to a tenuous blood supply and increased chance of dehiscence. Once the corner is secured, simple sutures are used to repair the rest of the wound, with care taken to place the sutures far enough from the tip to optimize circulation.

Pearls

1. Utilization of a mattress suture can aid in wound edge eversion and tension reduction. They are particularly useful in areas where the deep subcutaneous tissues are too fragile for deep sutures (eg, over a joint or shin).
2. A single corner stitch may be used to close several corners of a stellate wound.





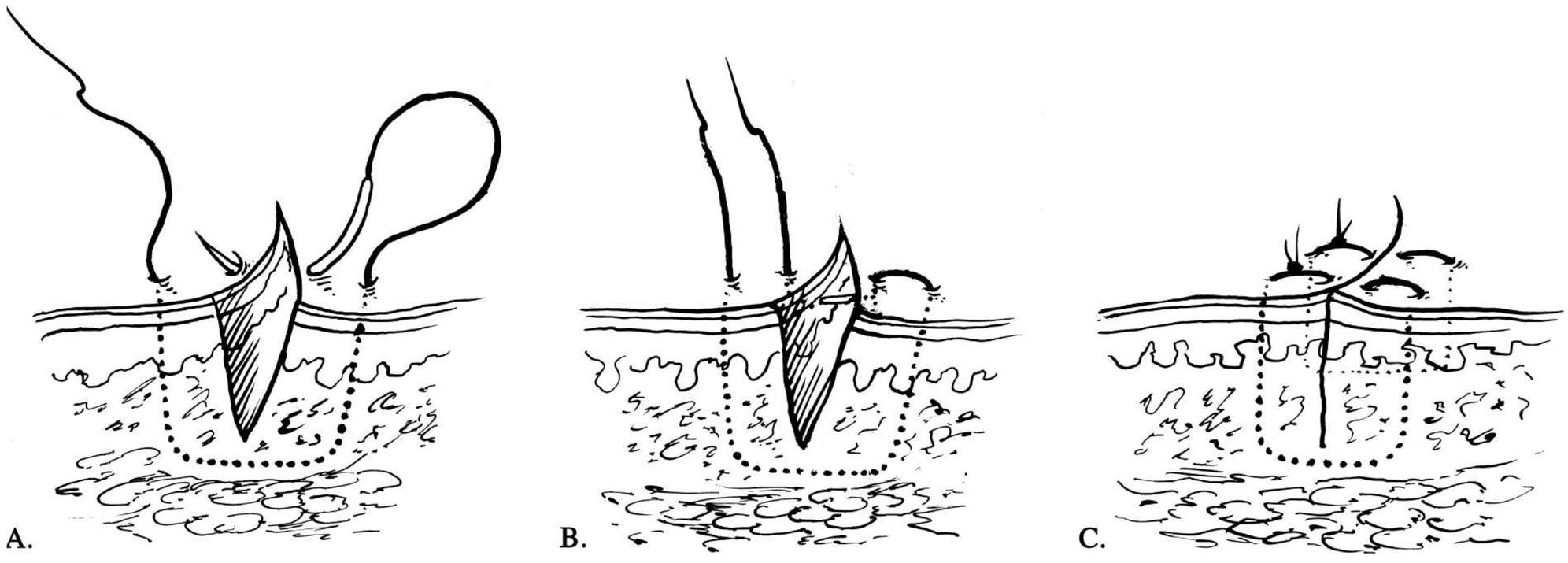
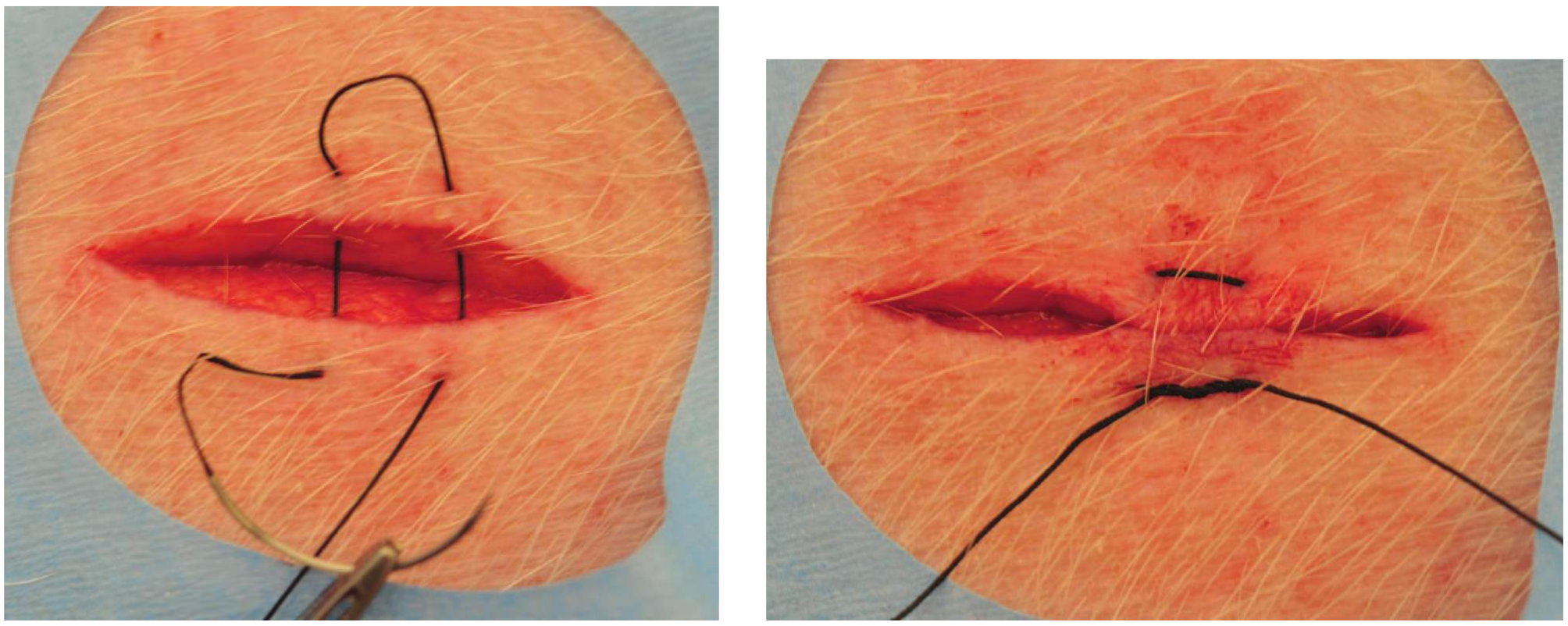


FIGURE 18.35 ■ **Vertical Mattress Suture.** The suture is placed by first taking a large deep bite of tissue approximately 1 cm away from the wound edge and exiting at the same location on the other side of the wound. A second small superficial bite is then performed in the reverse direction (**A**). When the bites are complete (**B**), tying results in nice apposition of the wound edges (**C**). This technique is especially useful in areas of lax skin, such as the elbow or dorsum of the hand. (Photo contributor: Michael L. Juliano, MD.)



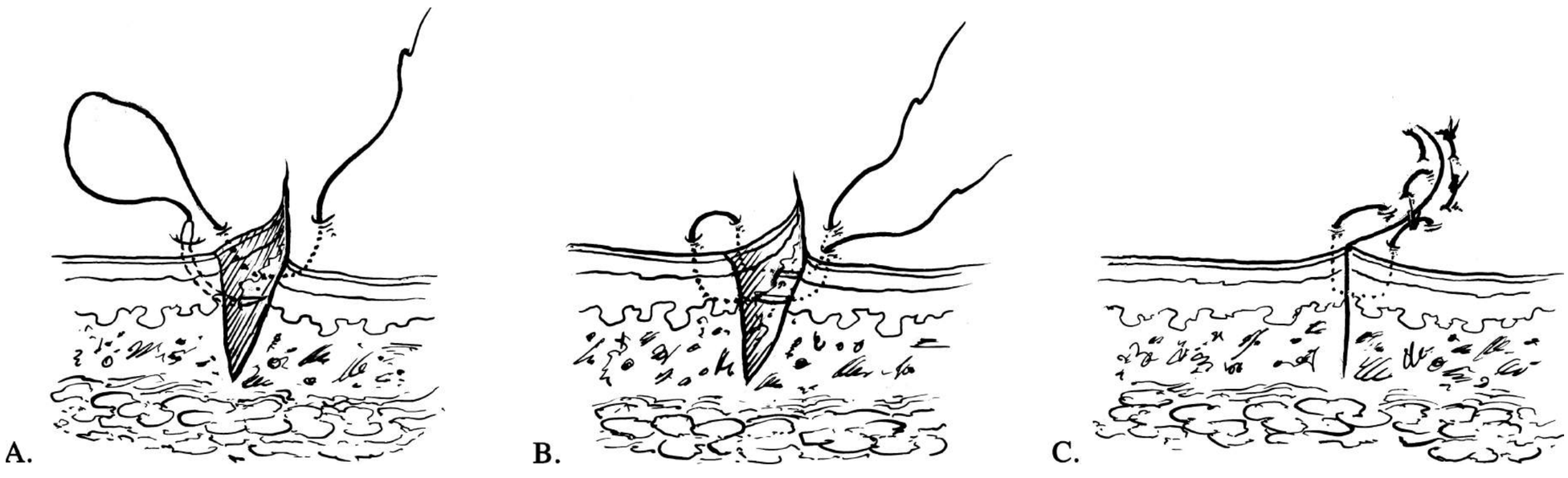


FIGURE 18.36 ■ **Horizontal Mattress Suture.** Useful in achieving wound edge eversion, the horizontal mattress suture begins with a standard suture throw. A second bite is taken approximately half a centimeter from the first exit (**A**) and brought through at the original starting edge, half a centimeter from the original entry point (**B** and **C**). (Photo contributor: Michael L. Juliano, MD.)

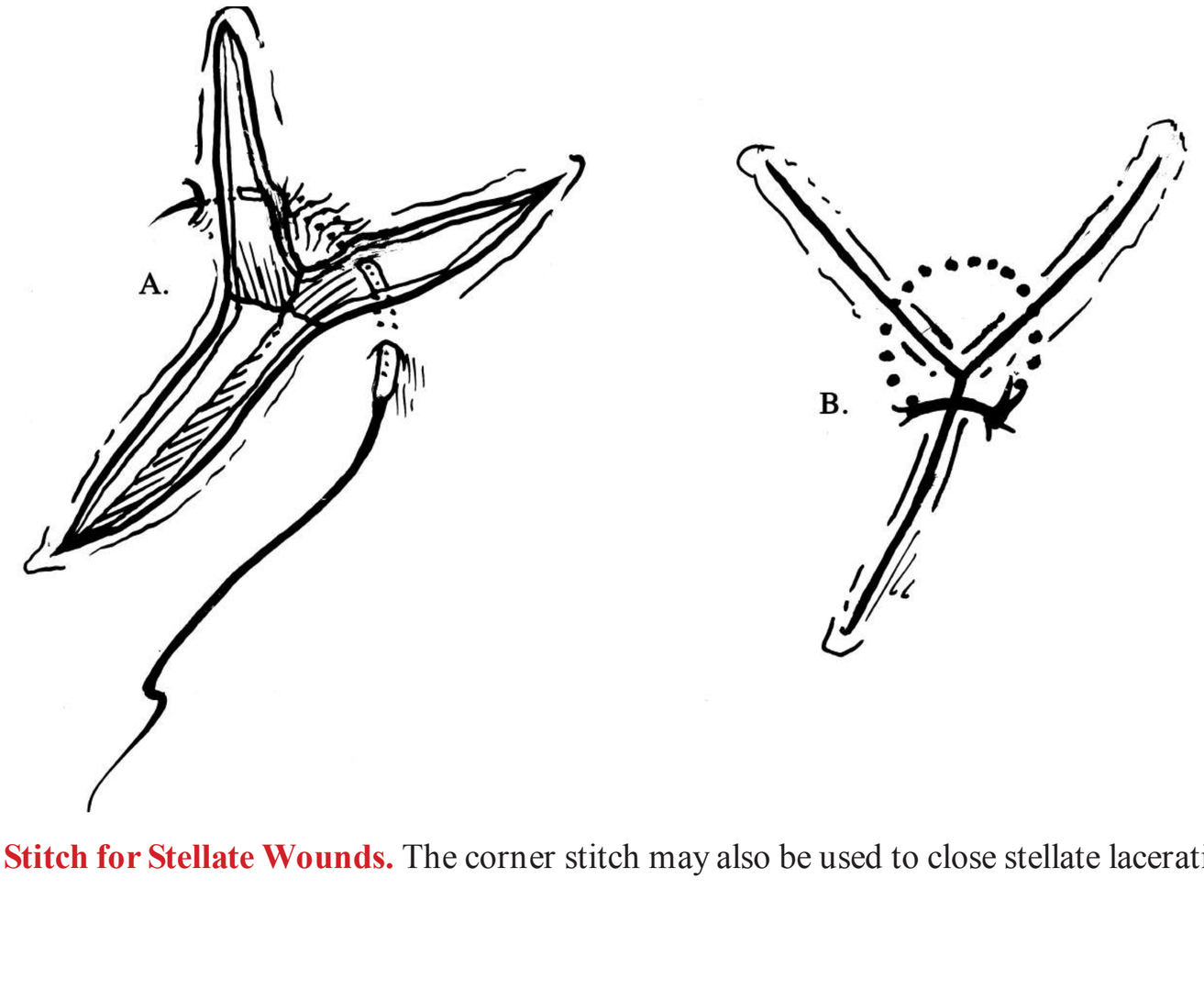
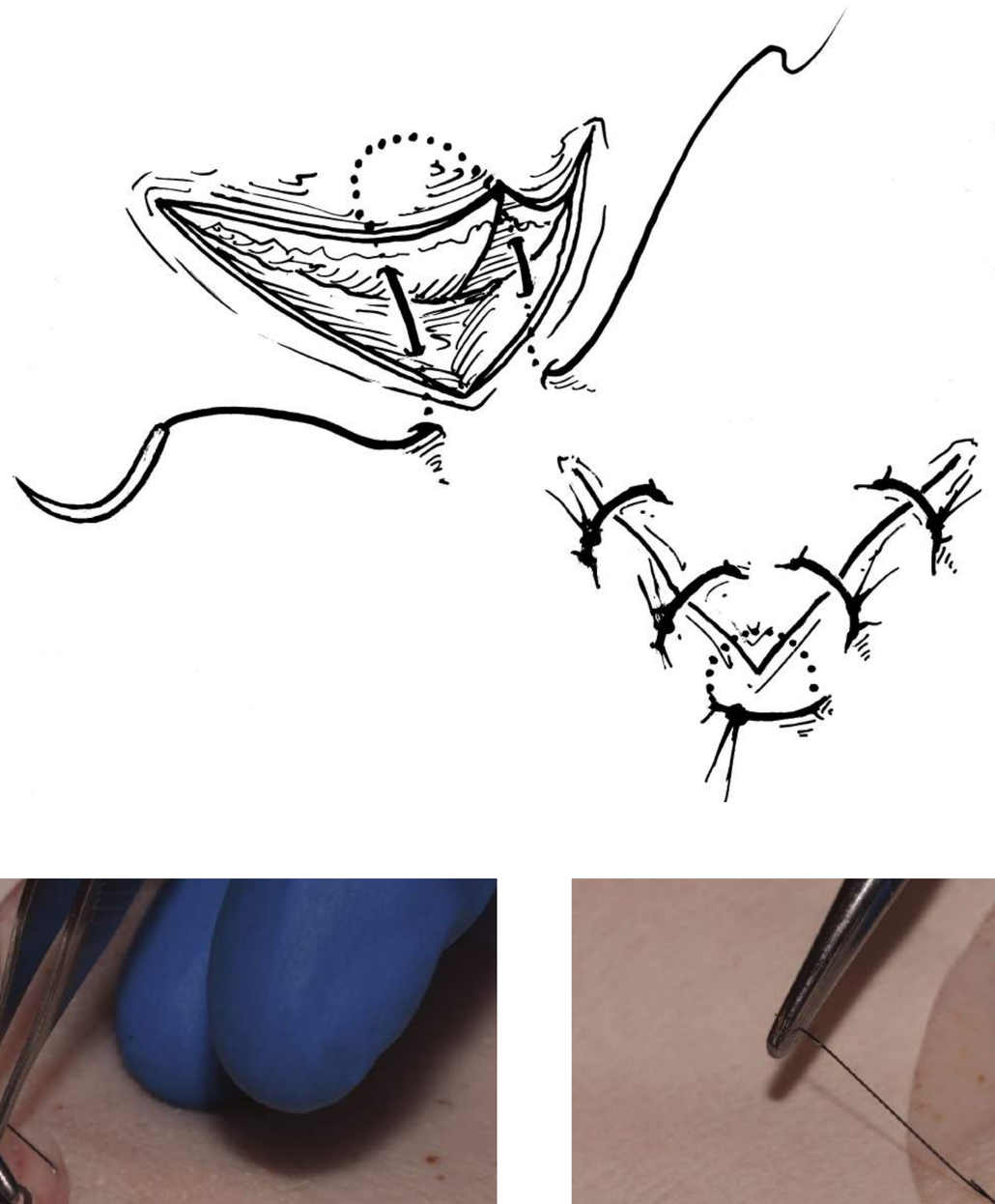
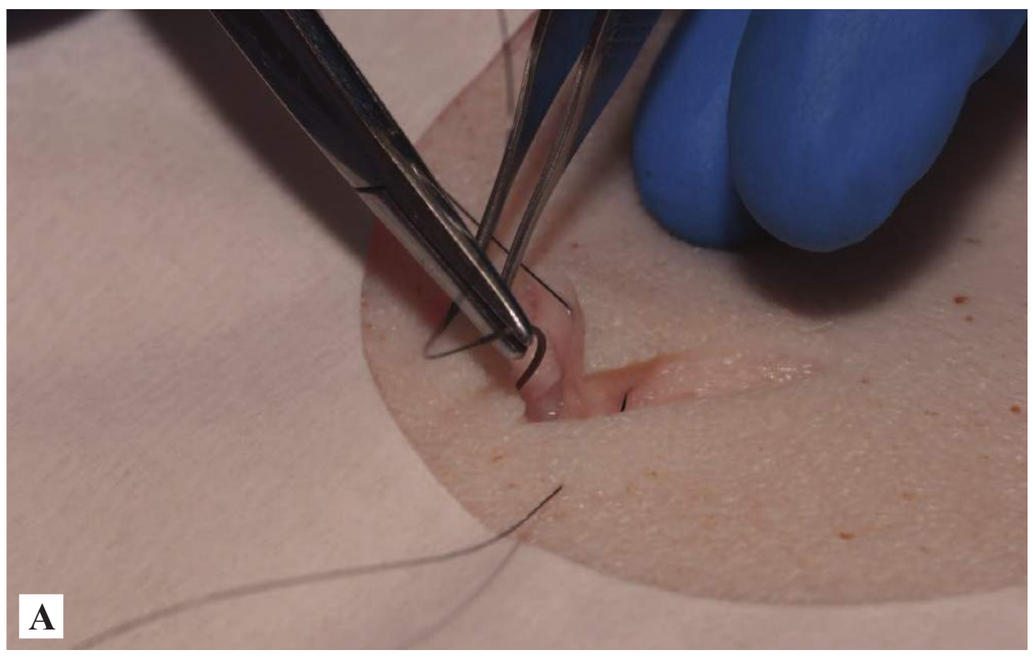
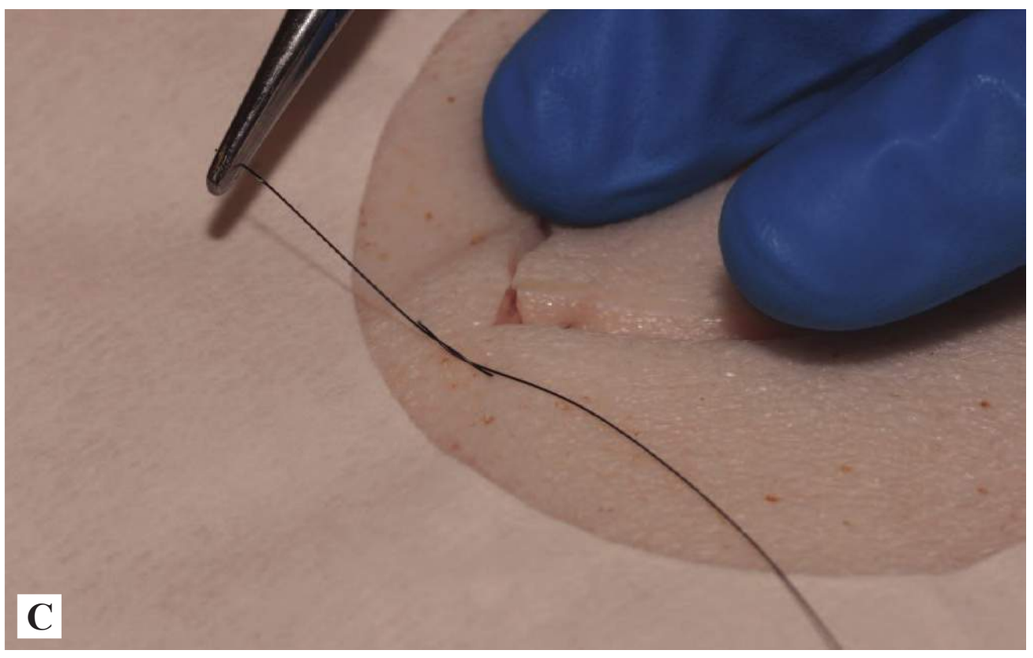
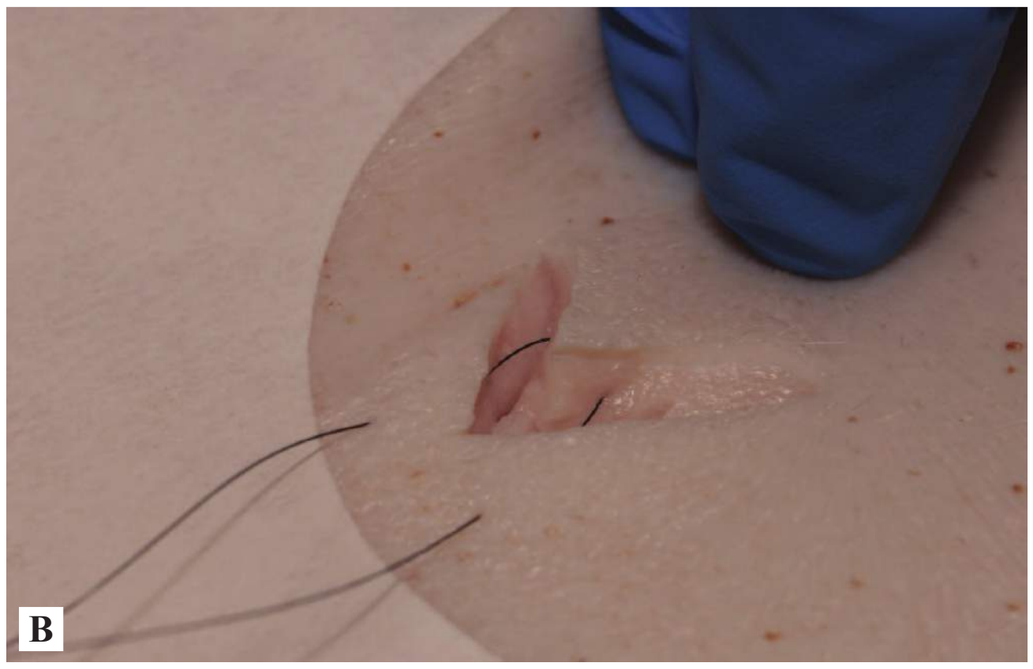


FIGURE 18.37 ■ **Corner Stitch for Stellate Wounds.** The corner stitch may also be used to close stellate lacerations.









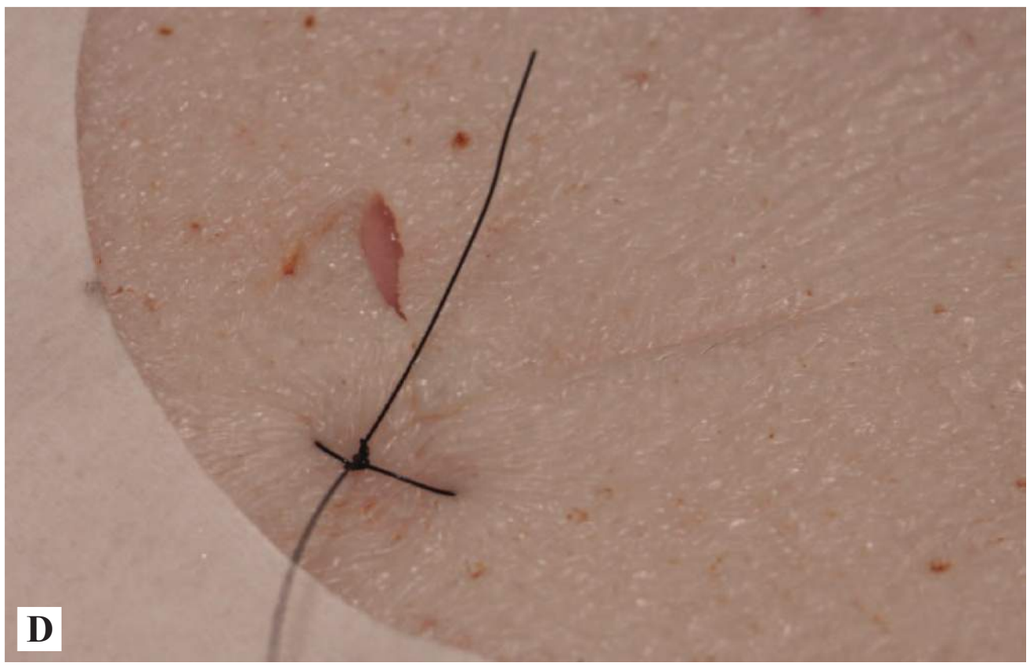


FIGURE 18.38 ■ **Corner Stitch.** Flaps generated by partial avulsion injuries must be repaired with care to avoid compromising the tenuous blood supply of the flap. The corner stitch is performed through the use of a half-buried horizontal mattress suture. The suture begins percutaneously away from the wound corner. The suture needle is then brought horizontally through the corner at the level of the dermis and back out through the epidermis at the opposite noncorner portion of the wound (**A**). This technique avoids placing suture material near the apex of the flap (**B**). Tying (**C**) results in approximation of the corner (**D**). (Photo contributor: Michael L. Juliano, MD).

Clinical Summary

Infection is suggested by pain, warmth, erythema, edema, and purulent drainage. While dehiscence can occur at any time, 7 to 10 days after repair a wound is at its weakest (this also closely coincides with suture removal). Impaired wound healing, primarily from infection, medications (especially corticosteroids), foreign bodies, advanced age, poor nutritional status, diabetes mellitus, and peripheral vascular disease, contributes to dehiscence. Elastic wound closure strips can be applied after suture removal and may reduce dehiscence.

Some degree of scarring is inevitable, but not considered a complication of wound repair. This should be discussed with the patient or caregiver. Wound myiasis is infestation by fly larvae (maggots, see Chapter 21, Tropical Medicine, Myiasis) that invade necrotic tissue.

Management and Disposition

Infections are treated with suture removal, thorough irrigation, and low threshold to explore for missed foreign bodies. A 7-day course of a first-generation cephalosporin or antistaphylococcal penicillin is appropriate; however, if methicillin-resistant Staphylococcus aureus (MRSA) is suspected, the antibiotic choice should be adjusted (eg, trimethoprim-sulfamethoxazole or other current recommendations for your area). For animal bites, other antibiotics (eg, amoxicillin/clavulanate) may be more appropriate. Sepsis, advanced infections, or infections in persons with chronic medical problems (eg, diabetes, immunocompromised) should be managed with parenteral antibiotics and possible inpatient admission.

Wound dehiscence is managed conservatively by treating the underlying causes and allowing healing via secondary intention. Dehiscence of wounds in cosmetically sensitive areas is best managed in conjunction with a consultant. Myiasis is treated with wound cleaning and irrigation.

Pearls

1. All accidental wounds are considered contaminated and treated as such. Thorough irrigation and cleansing is of paramount importance in preventing wound infection.
2. Expedient ED wound care is important since bacterial contamination increases over time.



FIGURE 18.39 ■ **Postoperative Wound Infection.** A patient with increasing pain and redness at the site of his staple closure. The area was erythematous, tender, warm, and had scant purulent drainage around some of the staples. (Photo contributor: Matthew D. Sztajnkrycer, MD, PhD.)



FIGURE 18.40 ■ **Wound Dehiscence.** After suture removal, the patient returned with dehiscence. The wound had a clean base of granulation tissue and was allowed to close by secondary intention. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 18.41 ■ **Keloid.** The degree of excessive scar bulk extending beyond the original wound margins may be dramatic and cosmetically significant. (Photo contributor: Thea James, MD.)



FIGURE 18.42 ■ **Wound Myiasis.** Necrotic tissue can be invaded by fly larvae (maggots). (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Burns can be caused by heat, electricity, chemicals, friction, or radiation. Skin barrier damage can lead to infection, fluid loss, and electrolyte abnormalities. Long-term consequences include permanent scarring, loss of sensation, and in severe cases loss of extremities due to inadequate circulation.

Burns are assessed by determining the percentage of body surface area (BSA) involved, the depth, and the area of the body involved. A common system used to estimate BSA is following the "rule of nines." This system breaks up the body into zones that each equate to 9% of BSA (see figure). Some clinicians use the palm of the patient's hand as an equivalent to 1% BSA and measure the area involved by using this method.

First-degree burns only involve the epidermal layer. They are red, painful, and heal in approximately 1 week. Second-degree burns are subdivided into two categories, superficial partial thickness and deep partial thickness. Superficial second-degree burns extend from the epidermis to the superficial dermis. Pain, skin blistering, and intact capillary refill are characteristic. Deep partial thickness burns extend into the deep (reticular) layer of the dermis and damage hair follicles, sweat glands, and sebaceous glands. Blisters may occur, the exposed dermis is pale white to yellow in color, and capillary refill is absent. The entire thickness of the skin is compromised in third-degree burns. They appear pale, feel leathery, and are painless. Fourth-degree burns extend through the layers of the skin and involve muscle or bone.

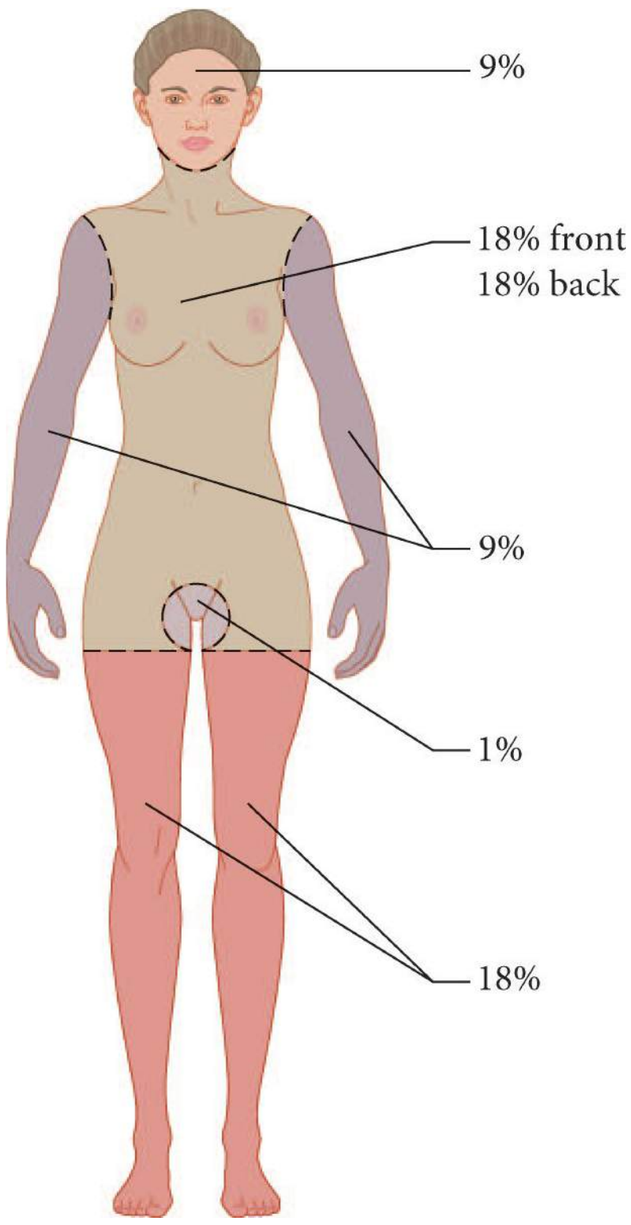


FIGURE 18.43 ■ **Rule of Nines.** Rule of nines is used to estimate the percentage of burn. (Reproduced with permission of Tintinalli JE, Stapczynski JS, Ma OJ, Cline DM, Cydulka RK, Meckler GD. Tintinalli's Emergency Medicine: A Comprehensive Study Guide. 7th ed. McGraw-Hill; 2011: Fig. 210-2, p. 1375.)



FIGURE 18.44 ■ **Second-Degree Burn.** This patient has sustained a second-degree burn to his hand from hot oil. (Photo contributor: Suzanne Dooley-Hash, MD.)



FIGURE 18.45 ■ **Facial Burn, Second Degree.** Ensuring that the patient has a protected airway in this type of injury is crucial. (Photo contributor: David Effron, MD.)

Management and Disposition

After resuscitation and stabilization, referral to a burn unit should be for partial thickness burns that involve greater than 10% BSA, third-degree burns, or involvement of the hands, feet, face, or perineum. Electrical burns, chemical burns, inhalation injuries, and patients with significant comorbidities should also be considered for a burn unit. Clinicians should cover the burned areas with a clean, dry sheet, administer aggressive pain control, and address fluid resuscitation. The Parkland formula is commonly used to estimate fluid requirements. The patient's weight in kilograms is multiplied by the percent BSA involved; this number is multiplied by 4 mL of lactated Ringer solution. Half of this amount is given during the first 8 hours from time of initial injury and the remaining amount is given over the next 16 hours. It is recommended to keep urine output approximately 0.5 to 1.0 mL/kg/h. In order to monitor for effects of cell breakdown, urinalysis, creatine kinase, and an EKG should be obtained. Circumferential burns of the extremities may compromise circulation. If compartment syndrome is suspected, an escharotomy should be considered.

For minor burns, provide pain control, cleansing of the area, and application of topical antimicrobials (eg, 1% silver sulfadiazine, bacitracin, or triple-antibiotic ointment). Dressing changes should occur daily and patients must be instructed to watch for signs of infection. Follow-up with a burn-care expert needs to be arranged within a few days of discharge for deep partial thickness or third-degree burns.

Pearls

1. Minor burns can be managed in the ED. Pain control, irrigation, antimicrobial ointments, and dressing changes are the mainstays of therapy.
2. Criteria for burn center referral should be reviewed for each patient (especially the young and elderly).



FIGURE 18.46 ■ **Extensive Burn Injury.** Burns can be of varying depths; this patient has both second- and third-degree burns. Distal pulses should be closely monitored to guarantee that there is adequate circulation. (Photo contributor: David Effron, MD.)



FIGURE 18.47 ■ **Abdominal Burn.** Mostly second-degree abdominal burn. (Photo contributor: R. Jason Thurman, MD.)

Clinical Summary

Electricity generates heat through tissue resistance or directly by the current on cells. Many factors affect injury severity: type of current (DC or AC), intensity, duration, tissue resistance, and pathway through the body. When electricity traverses the tissues, it may cause contact burns, thermal injury, muscular tetany, or severe contraction. Sudden death (asystole, respiratory arrest, ventricular fibrillation), myocardial damage, myoglobinuria, compartment syndrome, and various metabolic disorders have also been described.

High-voltage DC or AC current typically causes a single violent muscular contraction and throws the victim from the source; blunt trauma and blast injuries may occur. Low-voltage AC currents (as from a household outlet) typically cause muscular tetany, forcing the victim to continue contact with the source.

Management and Disposition

After initial stabilization, consider cervical spine immobilization, oxygen administration, cardiac monitoring, and IV crystalloid infusion. A Foley catheter will help monitor urine output and is especially important if rhabdomyolysis is suspected. Diagnostic testing to consider includes: electrocardiogram (ECG), complete blood count (CBC), urinalysis, CPK, CPK-MB, electrolytes, BUN, creatinine, and coagulation profile.

Severe or high-risk injuries should be admitted to a burn unit, or trauma center with burn consultation. Patients with minor, brief, low-intensity exposures, with a normal ECG, normal urinalysis, and no significant burns or trauma may be considered for discharge after a period of observation.

Pearls

1. Toddlers are at increased risk of labial injury from chewing on electrical cords.
2. High-risk features include high-voltage exposure (>600 V), deep burns, neurologic injury, dysrhythmias, abnormal ECG, evidence of rhabdomyolysis, suicidal intent, or significant associated trauma.



FIGURE 18.48 ■ **Electrical Burn.** This patient grabbed a high-voltage power line with his hand. Exit wounds occurred where the patient was grounded, through his feet. This patient with transthoracic injury should have cardiac monitoring. (Photo contributor: Alan B. Storrow, MD.)



FIGURE 18.49 ■ **Electrical Flash-Over Burn**. A patient struck by flash over electricity while repairing urban outdoor electric lines. (Photo contributor: Selim Suner, MD, MS.)



FIGURE 18.50 ■ **Electrical Burn.** Extensive electrical burn from a high-voltage power line. (Photo contributor: David Effron, MD.)



FIGURE 18.51 ■ **DC Thermal Ring Injury.** This middle-aged man sustained a circumferential thermal finger burn. His ring was heated when a wrench he was holding simultaneously contacted both terminals on a car battery. (Photo contributor: Aubrey Mowery, MPH, CHES.)

DECUBITUS ULCERS

Clinical Summary

Decubitus ulcers develop when soft tissue is compressed between a bony prominence and a hard external surface. Tissue compression results in decreased blood flow, tissue ischemia, and cell death. Pressure, shearing forces, friction, and excessive moisture contribute to formation. Areas commonly affected include the sacrum and heels. Immunocompromised, nursing home, neurologically impaired, and immobilized trauma patients are at high risk.

Stage I ulcers are characterized by an area of nonblanchable erythema over intact skin. Stage II appears as a shallow, open sore with a pink wound base. When the wound is full thickness with no muscle, tendon, or bone exposed, it is defined as stage III. If muscle, tendon, or bone is exposed, it is stage IV. Some wounds may have an area of black eschar over them; these wounds cannot be categorized since injury depth cannot be determined.

Management and Disposition

Prevention is key. All patients who have decreased mobility, such as trauma and nursing home patients, should have their entire skin surface regularly checked for skin breakdown and should be repositioned frequently. Backboards, c-collars, and other immobilizing devices should be removed as soon as possible.

Treatment includes pain relief, keeping the affected area clean, and keeping pressure off the area to prevent further tissue destruction. There are multiple commercial products available (hydrocolloid dressings) to use for the treatment of pressure ulcers. Frequent monitoring is required and consultation with wound care specialists or a surgeon may be necessary for advanced ulcers. The main complications of decubitus ulcer formation include infection and skin dehiscence.

Pearls

1. Decubitus ulcers are divided into four stages. If an eschar is present, the ulcer cannot be categorized.
2. Remove hard external surfaces and immobilizing devices as soon as possible.



FIGURE 18.53 ■ **Stage III Decubitus Ulcer.** Stage three decubitus ulcers extend into the subcutaneous tissue. (Photo contributor: David Effron, MD.)



FIGURE 18.52 ■ **Stage II Decubitus Ulcer.** Partial thickness loss of dermis presenting as a shallow open ulcer with a red-pink wound bed. (Photo contributor: Suzanne Dooley-Hash, MD.)

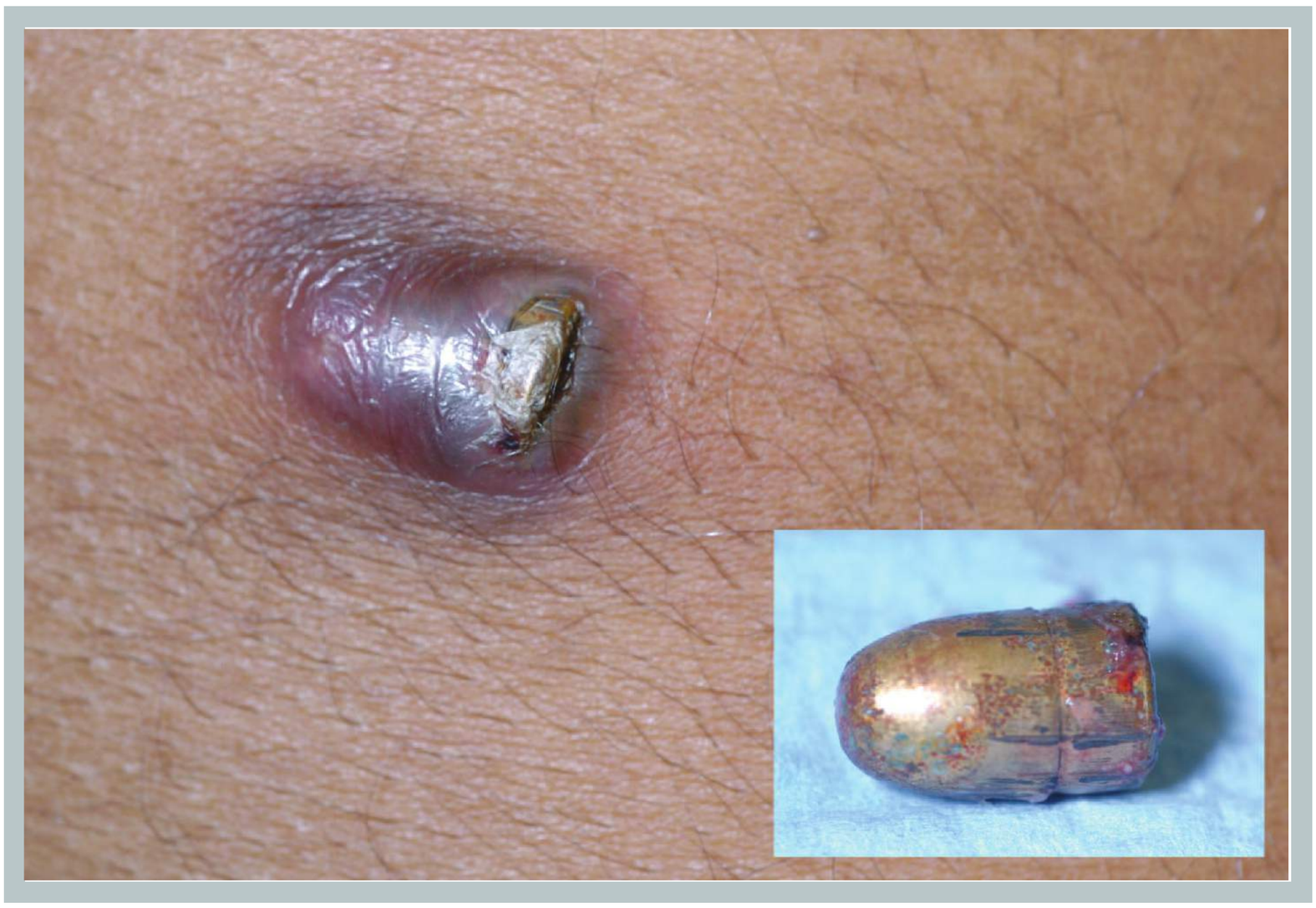


FIGURE 18.54 ■ **Stage IV Decubitus Ulcer.** Full-thickness tissue loss with exposed muscle. (Photo contributor: Lawrence B. Stack, MD.)

Chapter 19

CLINICAL FORENSIC MEDICINE

William S. Smock
Lawrence B. Stack



Skin Foreign Body. A bullet from a remote gunshot wound came to the skin surface of the right posterior chest wall. The bullet was removed (inset) at the patient's request. (Photo contributor: Lawrence B. Stack, MD.)

GUNSHOT WOUNDS

Clinical Summary

Gunshot injuries can be accurately identified and classified as entrance, atypical entrance, exit, or atypical (grazing) wounds based upon their physical characteristics. **Wounds are not classified based upon their size**. Physical findings in and around these wounds may offer evidence as to the actual mechanism of injury, supporting or refuting the initial history given to the provider. As most physical findings are transient in nature (cleaned, debrided, or eventually healed), the emergency physician must be diligent in recognizing and documenting them at the time of presentation. The physician's failure to accurately document the physical characteristics of the wounds or correctly interpret the physical findings can compromise the legal process and obstruct justice.

Entrance Wounds

Gunshot wounds of entrance are divided into four categories based on their range of fire: distant, intermediate, close, and contact. Range-of-fire is the distance from the gun's muzzle to the victim's skin or clothing.

The size of the entrance wound bears no relation to the caliber of the inflicting bullet. Entrance wounds over elastic tissue will contract around the tissue defect and have a diameter much less than the caliber of the bullet.

Distant Wounds: The distant wound is inflicted from a range sufficiently distant that the bullet is the only component expelled from the muzzle that reaches the skin. There is no visible tattooing or soot deposition associated with a distant entrance wound. As the bullet penetrates the skin, friction between it and the epithelium results in the creation of an "abrasion collar" (Fig. 19.1). The width of the abrasion collar will vary with the angle of impact. Elongated abrasion collars from projectiles that enter on an angle may produce a collar with a "comet tail" (Fig. 19.2). Most entrance wounds will have an abrasion collar; however, gunshot wounds to the palms and soles are exceptions—their entrance wounds appear slit-like. Bullets that pass through an intermediate object, a door or windshield for example, will become deformed or misshapen (Fig. 19.3). A misshapen bullet creates an irregular abrasion collar (Fig. 19.4) as compared to the smooth abrasion collar created by a nondeformed bullet (Fig. 19.1).

Intermediate-Range Wounds: Tattooing is pathognomonic for an intermediate-range gunshot wound and presents as punctate abrasions from contact with partially burned or unburned grains of gunpowder (Fig. 19.5). This tattooing cannot be wiped away. Clothing and hair, as intermediate objects, may prevent the gunpowder grains from making contact with the skin. Tattooing can, but rarely does, occur on the palms and soles owing to the thickness of their epithelium.

Tattooing has been reported with a range of fire as close as 1 cm and as far away as 4 feet. The density of the abrasions

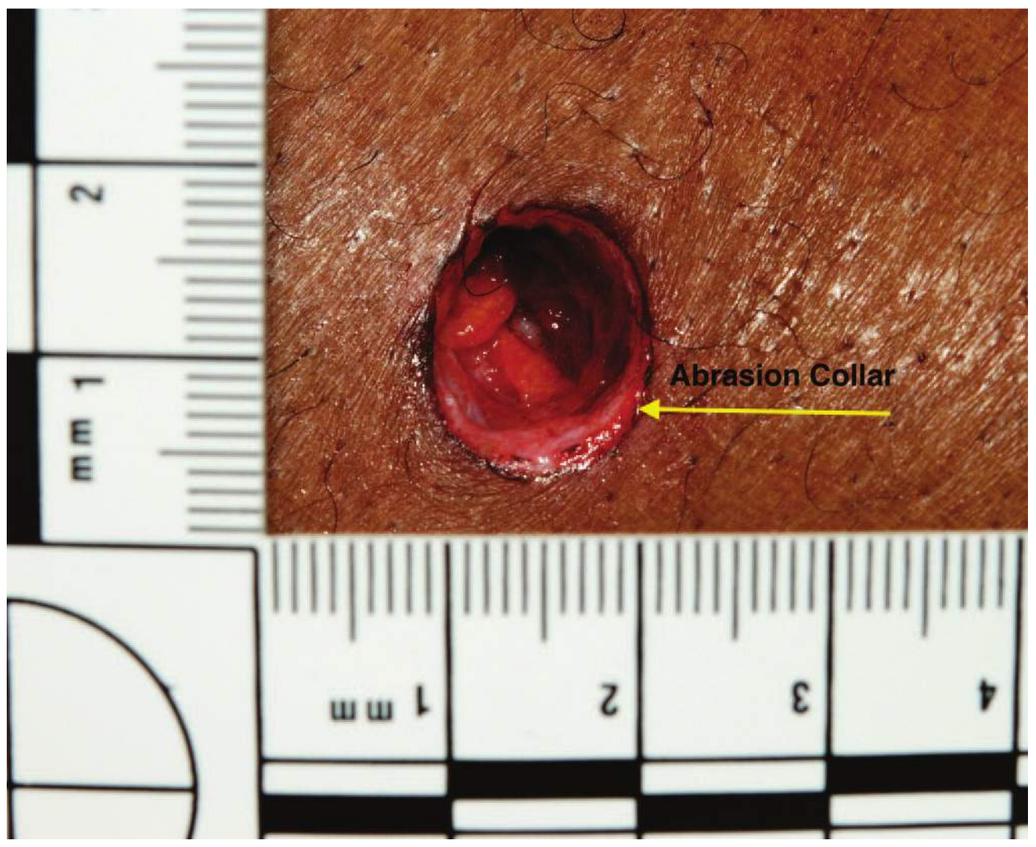


FIGURE 19.1 ■ **Distant Gunshot Wound.** An abrasion collar surrounds the wound defect and is created from the friction of a bullet passing through the skin. All entrance wounds will have an abrasion collar with the exception of entrance wounds to the palms and soles. The lack of soot, seared skin, or gunpowder tattooing confirms this is a "distant" range of fire. (Photo contributor: William S. Smock, MD.)

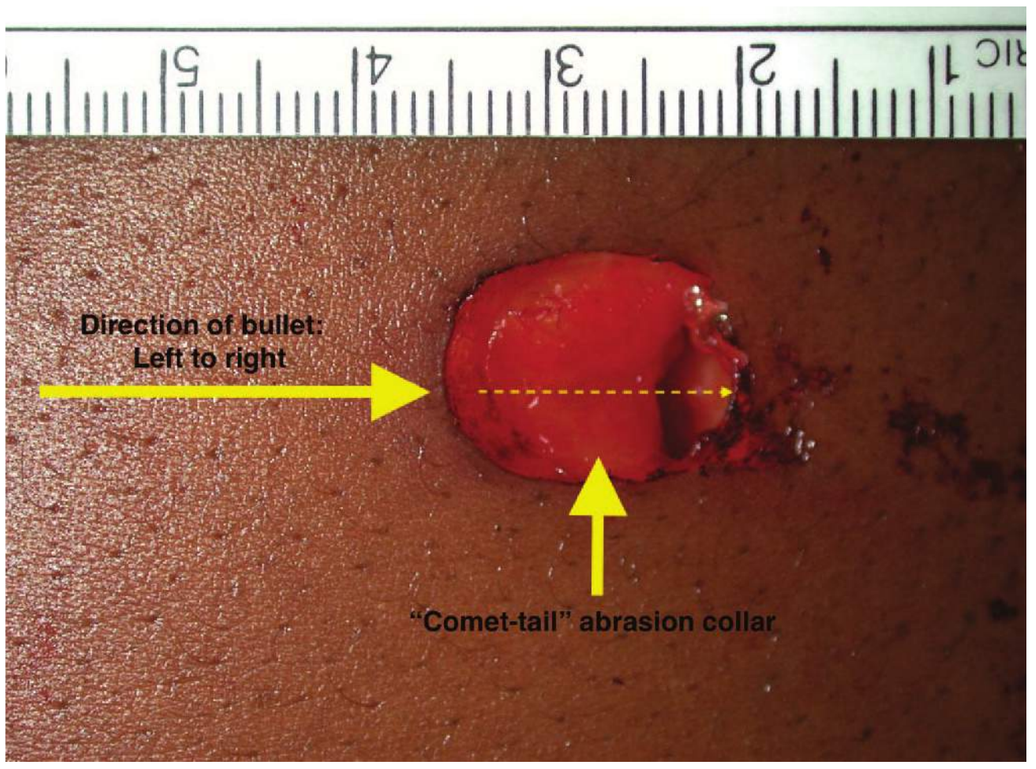


FIGURE 19.2 ■ **"Comet-Tailed" Abrasion Collar.** The "comet tail" abrasion collar located on the lateral aspect of the wound indicates that the bullet entered the wound at an angle. The "comet tail" also indicates the bullet's direction of travel: from left to right. (Photo contributor: William S. Smock, MD.)



FIGURE 19.3 ■ **Deformed Bullet.** This bullet passed through a car windshield and deformed prior to striking the suspect in the arm. The bullet's irregular shape created the atypical abrasion collar associated with the entrance wound seen in Fig. 19.4. (Photo contributor: William S. Smock, MD.)

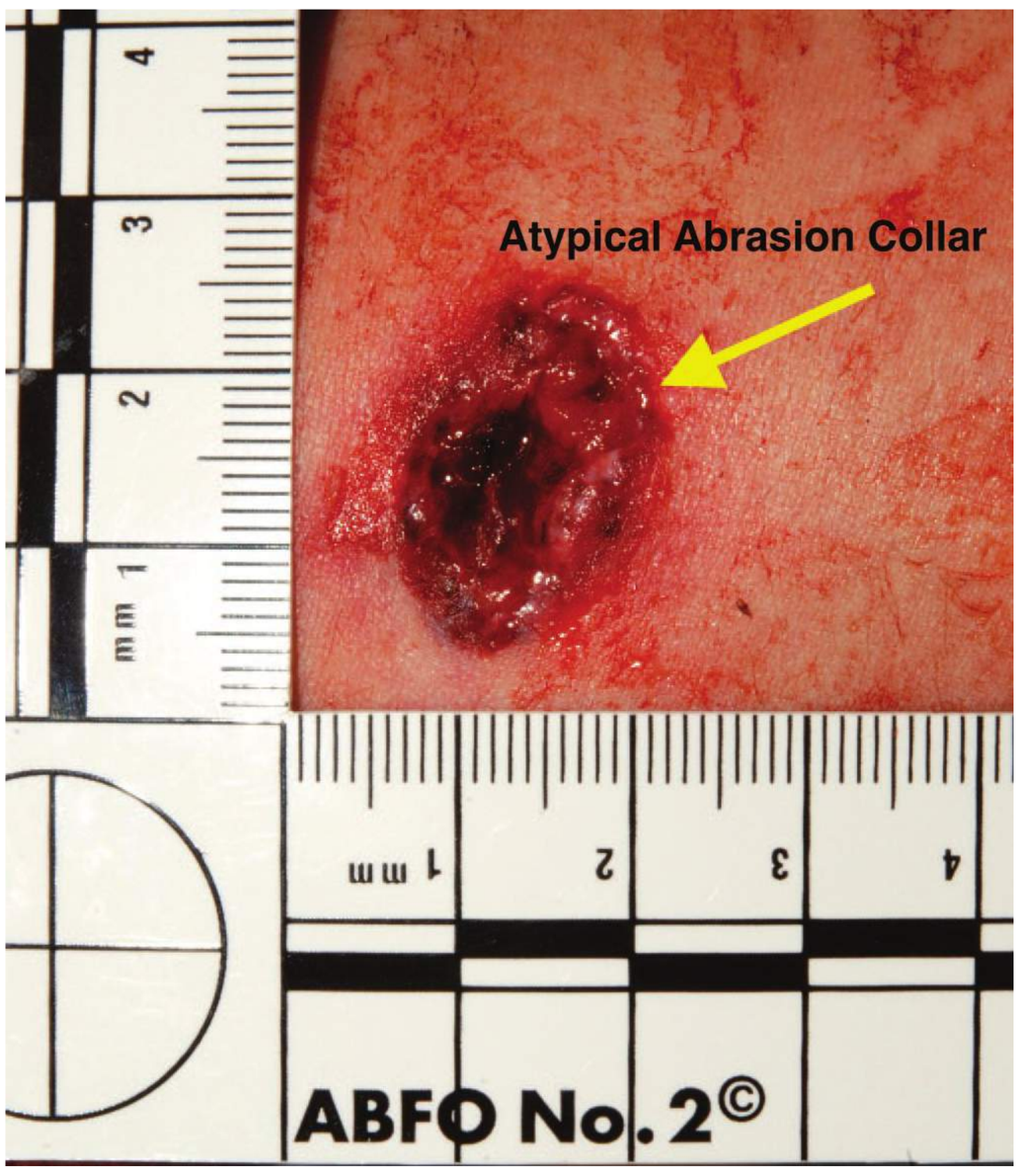


FIGURE 19.4 ■ **Atypical Abrasion Collar.** When a bullet passes through an intermediate object, ie, a door or windshield, it can become deformed or misshapen with an irregular surface. When a misshapen bullet (Fig. 19.3) passes through skin, the abrasion collar it generates appears irregular and jagged when compared to the smooth one caused by the nondeformed bullet (Figs. 19.1 and 19.2). (Photo contributor: William S. Smock, MD.)

and the associated pattern will depend upon the barrel length, muzzle-to-skin distance, type of gunpowder (ball, flattened ball, or flake), presence of intermediate objects, and caliber of the weapon. Spherical powder travels farther and has greater penetration than flattened ball or flake powder. "Pseudotattooing" (Fig. 19.6) is punctate abrasions from fragments created when bullets pass through intermediate objects like wood or glass.

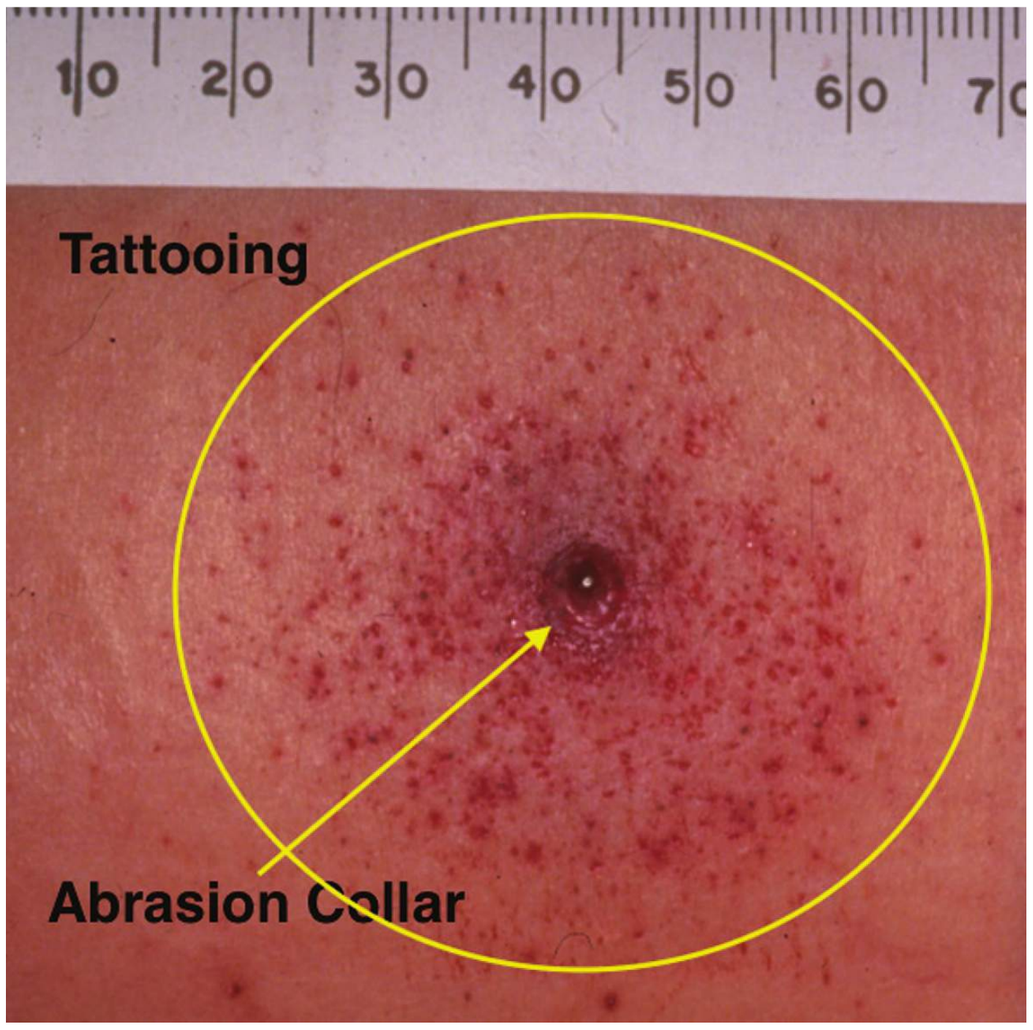


FIGURE 19.5 ■ **Intermediate-Range Gunshot Wound.** Punctate abrasions present on the skin are the result of impact from gunpowder that is either wholly or partially unburned. This phenomenon is termed tattooing or stippling. Tattooing is pathognomonic for intermediate-range (less than 48 inch) gunshot wounds. (Photo contributor: William S. Smock, MD.)



FIGURE 19.6 ■ **Pseudotattooing.** Punctate abrasions present on the forearm are the result of impacts with glass fragments. The fragments were produced from bullets penetrating the windshield. (Photo contributor: William S. Smock, MD.)

Close-Range (Near-Contact) Wounds: "Close range" is defined as the maximum range at which soot is deposited on the clothing (Fig. 19.7) or wound (Fig. 19.8) and typically is a muzzle-to-victim distance of 6 inches or less. On rare occasions, however, soot has been found on victims as far as 12 inches from the offending weapon. The concentration of soot will vary inversely with the muzzle-to-victim distance and its appearance will be affected by the type of gunpowder and ammunition used, the barrel length, the caliber, and the type of weapon.

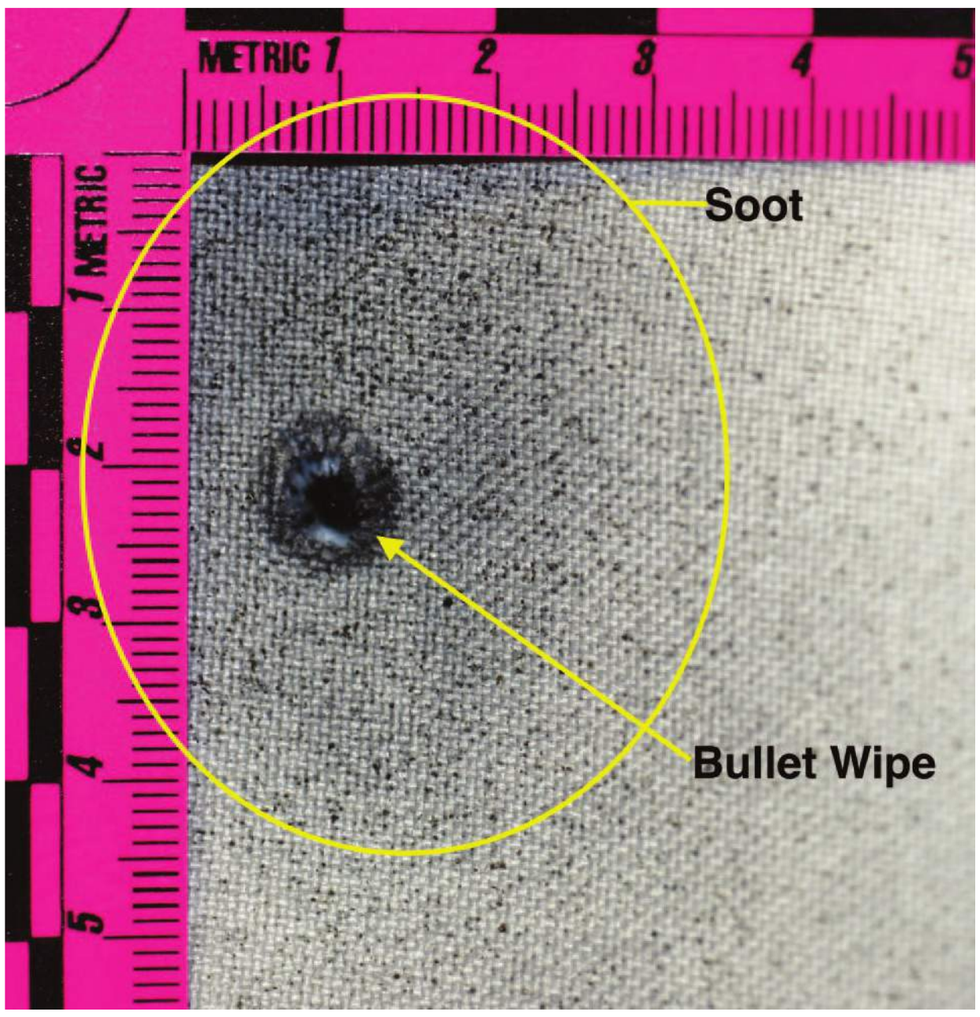


FIGURE 19.7 ■ **Soot and Bullet Wipe.** Soot is the carbonaceous residue from the burning of gunpowder. Soot is associated with close-range wounds, 6 inches or less. Bullet wipe is residue and/or lead from the surface of the bullet that is transferred to clothing or skin. Bullet wipe can be seen at any range of fire. Clothing should be collected and packaged in separate paper bags for submission to the crime laboratory. (Photo contributor: William S. Smock, MD.)

Contact Wounds: A contact wound occurs when the barrel or muzzle is in contact with the skin or clothing as the weapon is discharged. Contact wounds can be described as tight, where the muzzle is pushed hard against the skin, or loose, where the muzzle is incompletely or loosely in contact with the skin or clothing. Wounds sustained from tight contact with the barrel can vary in appearance from a small hole with seared, blackened edges (from the discharge of hot gases and an actual flame), to a gaping, stellate wound (from the expansion of the skin from gases) (Figs. 19.9 and 19.10). Large stellate wounds are often misinterpreted as exit wounds based solely upon their size and without adequate examination of the wound characteristics.

In a tight-contact wound, all materials—the bullet, gases, soot, incompletely combusted gunpowder, and metal fragments—are driven into the wound. If the wound overlies thin or bony tissue, the hot gases will cause the skin to expand to such an extent that it stretches and tears. These tears will have a triangular shape, with the base of the tear overlying the entrance wound. Larger tears are associated with ammunition of .32 caliber or greater, or magnum loads.

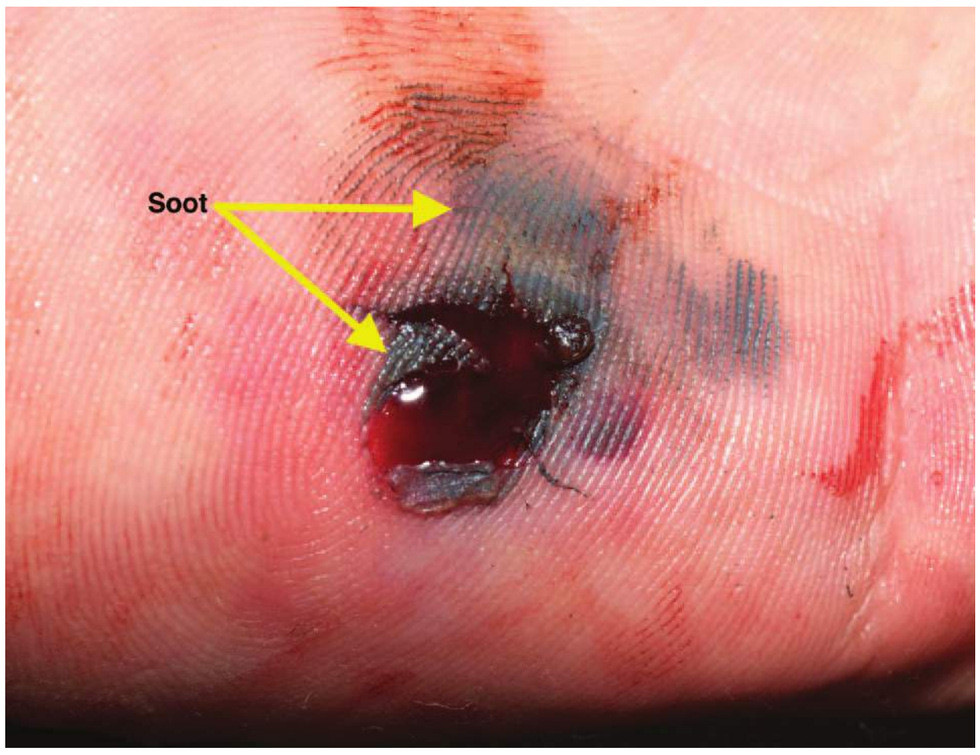


FIGURE 19.8 ■ **Close-Range Gunshot Wound.** The deposition of carbonaceous material or soot is seen on the palm from a close-range or near-contact gunshot wound. Soot is short-lived evidence and its presence surrounding a wound should always be documented. (Photo contributor: William S. Smock, MD.)

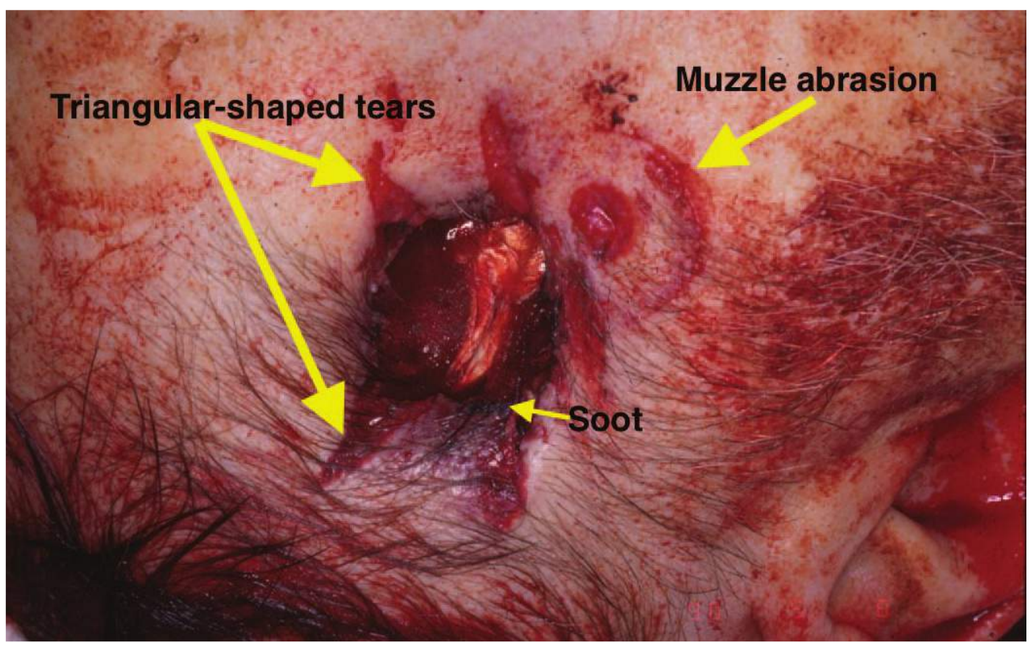


FIGURE 19.9 ■ **Contact Gunshot Wound with Muzzle Abrasion.** A contact gunshot wound to the right temple from a 9 mm semiautomatic handgun. Note the triangle-shaped tears, soot, seared wound margins and a muzzle-abrasion at the 2-o'clock position. The muzzle abrasion or muzzle imprint was the result of the injection of gases into the skin, causing a rapid and forceful expansion of the skin against the barrel. (Photo contributor: William S. Smock, MD.)



FIGURE 19.10 ■ **Contact Gunshot Wound.** A contact wound to the forehead from a .38 caliber handgun. The wound margins display triangle-shaped tears, searing, and soot deposition. (Photo contributor: William S. Smock, MD.)

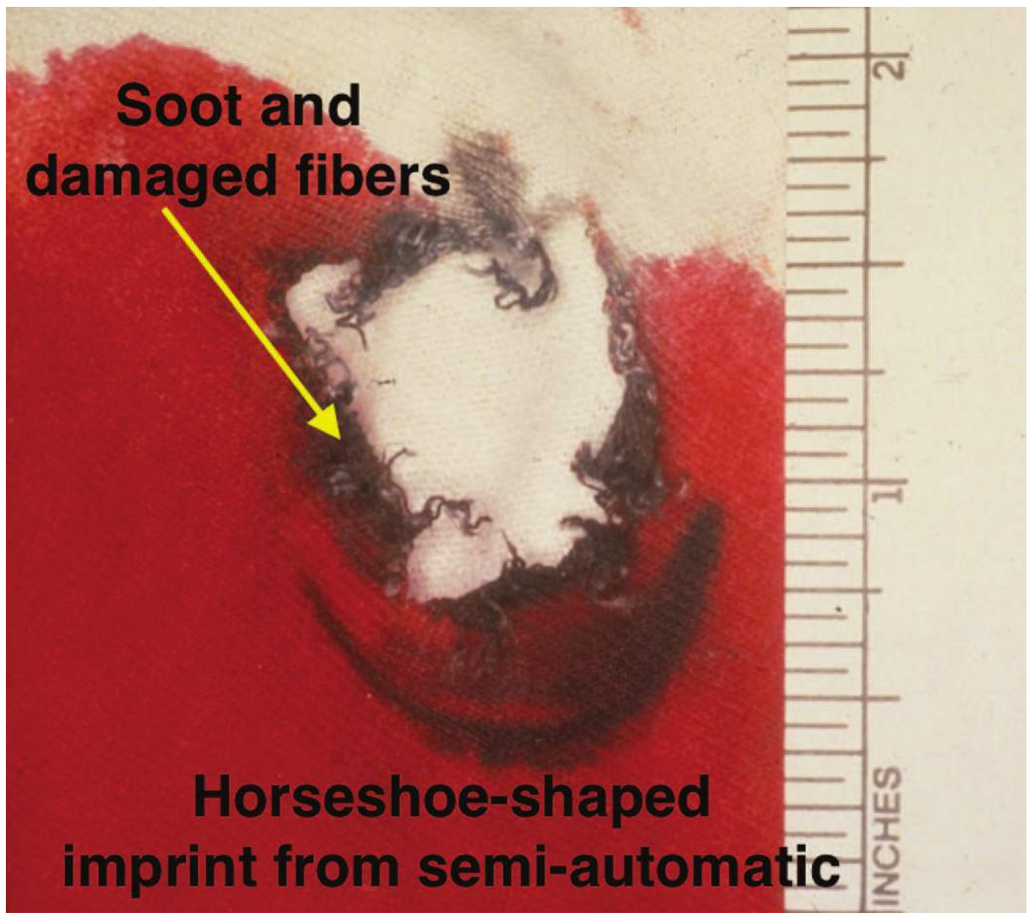


FIGURE 19.11 ■ **Muzzle Imprint, Soot, and Seared Fibers.** The clothing exhibits a "horseshoe-shaped" soot mark reflecting the outline of the frame of a semiautomatic handgun. Seared fibers are the result of flame and hot gases expelled from the barrel when it is in contact with clothing. (Photo contributor: William S. Smock, MD.)

Stellate tears are not pathognomonic for contact wounds. Tangential wounds, ricochet or tumbling bullets, and some exit wounds may also be stellate in appearance. These wounds are distinguished from tight-contact wounds by the absence of soot and powder within the wound. In some tight-contact wounds, expanding skin is forced back against the muzzle of the gun, causing a characteristic pattern contusion called a muzzle contusion (Fig. 19.9). An outline of the barrel can also be imprinted on the overlying clothing and is associated with contact wounds through clothing (Fig. 19.11). These patterns are helpful in determining the type of weapon (revolver or semiautomatic) used to inflict the injury and should be documented prior to wound debridement or surgery.

With a loose-contact wound, where the muzzle is angled or held loosely against the skin, soot and gunpowder residue will be present in and around the wound. The angle between the muzzle and skin will determine the soot pattern. A perpendicular, loose-contact or near-contact injury results in searing of the skin and deposition of the soot evenly around the wound. A tangential loose or near-contact injury produces an elongated searing pattern and deposit of soot around the wound.

"Bullet wipe" is a residue from soot, soft lead, or lubricant, which may leave a gray or black rim or streak on the skin or clothing overlying an entrance wound (Fig. 19.7). This discoloration may also be found around the abrasion collar but is usually more prominent on clothing.

Exit Wounds

Determining whether a wound is an entrance or an exit wound should be based on the physical characteristics and physical evidence associated with the wound and never upon the size of the wound. The size of the exit wounds are the result of a bullet pushing and stretching the skin from inside outward. The skin edges are generally everted, with sharp but irregular margins (Figs. 19.12-19.14). Abrasion collars, soot, searing, and tattooing are not associated with exit wounds. Soot can be seen at an atypical exit wound site if the entrance wound is close to the associated exit wound. Soot can be propelled through the wound from entrance to exit when the wound track is extremely short. If this is noted, the soot deposition will be more pronounced at the entrance and only faintly observed within the exit wound.

Exit wounds assume a variety of shapes and appearances and are not consistently larger than their corresponding entrance wounds. The size of an exit wound is determined primarily by the amount of energy transmitted to underlying tissue, bone for example, which is extruded from the wound. A bullet's size, shape, and attitude as it exits as well as postinjury tissue swelling will affect its size. A bullet's usual nose-first attitude can change upon entering the skin to a tumbling

and yawing one. A bullet with sufficient energy to exit the skin in a sideways attitude or one that has increased its surface area by mushrooming may produce an exit wound larger than its entrance wound. Energy transferred to bone, with resultant ballistic fracture, may also result in an exit wound larger than the entrance wound (Fig. 19.14). A "false abrasion collar" or "shored exit" wound may mimic an entrance wound. This occurs when the epithelium is pressed against a supporting surface such as a floor, wall, chair, firm mattress, or wallet (Figs. 19.15 and 19.16).



FIGURE 19.12 ■ **Exit Gunshot Wound.** A slit-like exit wound on the scalp from an intraoral gunshot. Exit wounds may take on a variety of appearances. (Photo contributor: William S. Smock, MD.)



FIGURE 19.13 ■ **Exit Gunshot Wound.** Stellate tears in an exit wound from a .22 caliber long rifle bullet that impacted the radius and ulna. The stellate configuration of an exit wound should not be confused with that of a contact wound. Exit wounds lack soot and seared skin. (Photo contributor: William S. Smock, MD.)

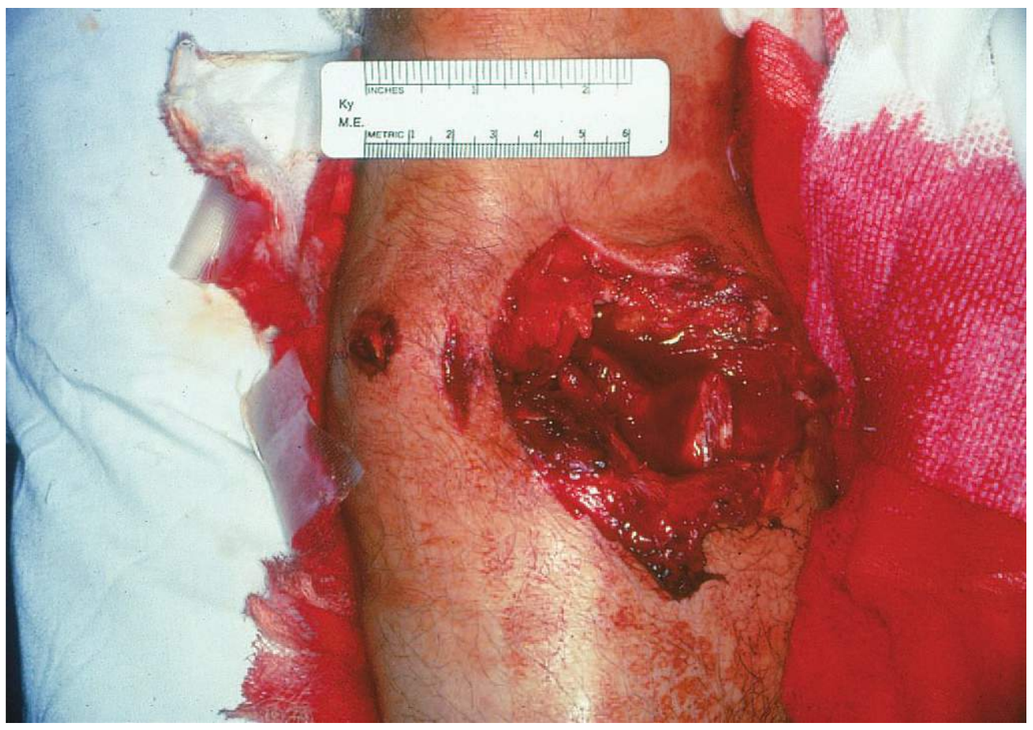


FIGURE 19.14 ■ **High-Velocity Gunshot Wound.** A perforating high-velocity gunshot wound to a lower extremity. The gaping exit wound resulted from the transfer of energy from the projectile to the tibia. The impact propelled multiple bony fragments through the skin. (Photo contributor: William S. Smock, MD.)

Graze Wounds

Graze wounds are considered atypical and result from tangential contact with a passing bullet. The direction of the bullet's path may be determined by careful wound examination. The bullet produces a trough and may cause the formation of skin tags on the lateral wound margins (Fig. 19.17). The base of these tags points toward the weapon and away from the direction of bullet travel.

Evidence Collection

Preservation of clothing, bullets, and cartridge cases is critical to the investigation of gun violence. Clothing should be place in separate paper bags and bullets in breathable containers. The microscopic marks on the exterior surface of a bullet can be used to identify the offending weapon. The use of booties on the ends of hemostats can prevent the loss of essential evidence and preserve these unique identifying microscopic marks from being altered or permanently destroyed (Fig. 19.18).

Pearls

1. Distant-range gunshot wounds are inflicted from a distance of greater than 4 feet and there is no tattooing, soot, or searing associated with the wound.
2. Intermediate-range gunshot wounds are inflicted at a distances up to 4 feet and characteristically are associated with tattooing from partially burned and unburned gunpowder impacting the skin.



FIGURE 19.15 ■ **Shored Gunshot Exit Wound.** A "shored exit" or "false abrasion collar" associated with a gunshot wound of exit. The false abrasion collar results when the skin is supported by a firm surface as the bullet exits. Shored exits occur when epithelium is pressed against a supporting surface (ie, floor, wall, chair, firm mattress, or, as in this case, wallet). (Photo contributor: William S. Smock, MD.)



FIGURE 19.16 ■ **Wallet Causing Shored Gunshot Exit Wound.** The wound seen in Fig. 19.15 occurred as a result of this wallet resisting the bullet's exit. (Photo contributor: William S. Smock, MD.)



FIGURE 19.17 ■ **Graze Gunshot Wound.** A deep graze wound from a handgun is seen. The dark wound margins are the result of drying artifact and should not be confused with the deposition of soot. (Photo contributor: Lawrence B. Stack, MD.)

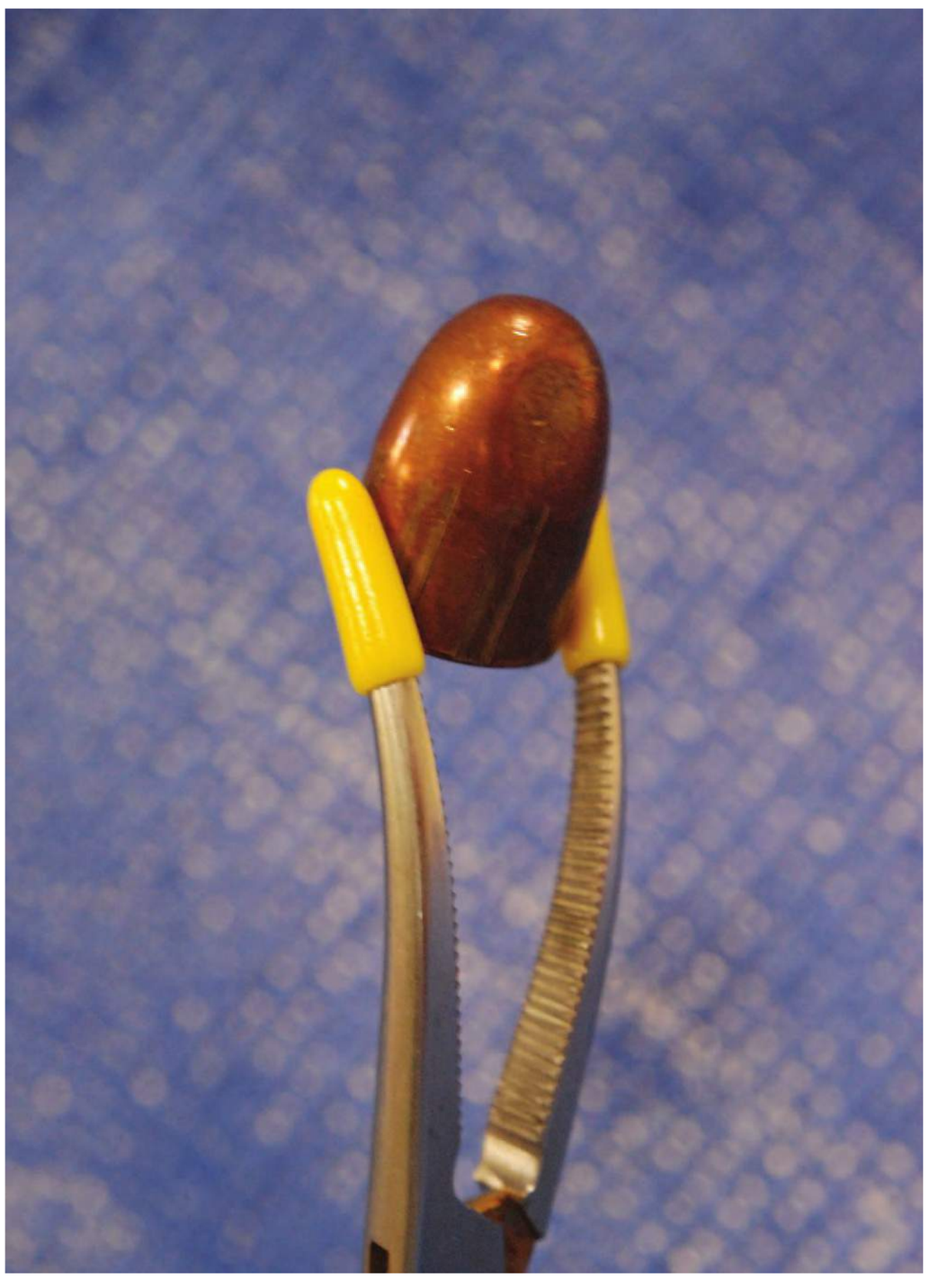


FIGURE 19.18 ■ **Suture Booties.** The use of booties on hemostats prevents the identifying microscopic marks on the bullet's surface from being damaged by handling. (Photo contributor: William S. Smock, MD.)

3. Near or close-contact gunshot wounds are defined as the maximum range at which soot is deposited on the wound or clothing and typically occur at a distance of 6 inches or less.
4. Contact gunshot wounds (barrel is in contact with the skin or clothing at time of discharge) vary in size but will include triangular tears, searing of the skin, and soot within or around the wound.
5. Abrasion collars, soot, searing, and tattooing are associated with entrance wounds.
6. Determination of whether a wound is an entrance or exit wound should **only** be based on the physical characteristics of the wound and clothing and not on the size of the wound.
7. The size of an exit wound is determined by: (1) energy transferred from the bullet to underlying tissue (bone pushed out), (2) bullet shape (mushroomed) and configuration, and (3) swelling of underlying tissue.
8. Emergency physicians should attempt to recognize, document, preserve, and collect short-lived evidence whenever the clinical situation allows.

Every "weapon" (hand, belt, hot iron, knife, electrical cord, baseball bat, tire iron) can leave a mark, design, or pattern stamped or imprinted upon or just below the level of the epithelium. The imprints of these weapons are called pattern injuries, which are considerably reproducible. These injuries can be categorized into three major classifications according to their source: sharp-force, blunt-force, and thermal-pattern injuries.

SHARP-FORCE-PATTERN INJURIES

Clinical Summary

There are two types of sharp-force injuries: incised and stabbed. The incised wound is longer than it is deep. The stab wound is defined as a puncture wound that is deeper than it is wide (Figs. 19.19-19.21). The wound margins of sharp-force injuries are clean and lack the abraded edges of lacerations from blunt forces. Forensic information can be gathered during the examination of a stab wound. Some characteristics of a knife blade, single- or double-edged, can be determined by visual inspection. Characteristics such as serrated versus sharp can be determined if the blade was drawn across the

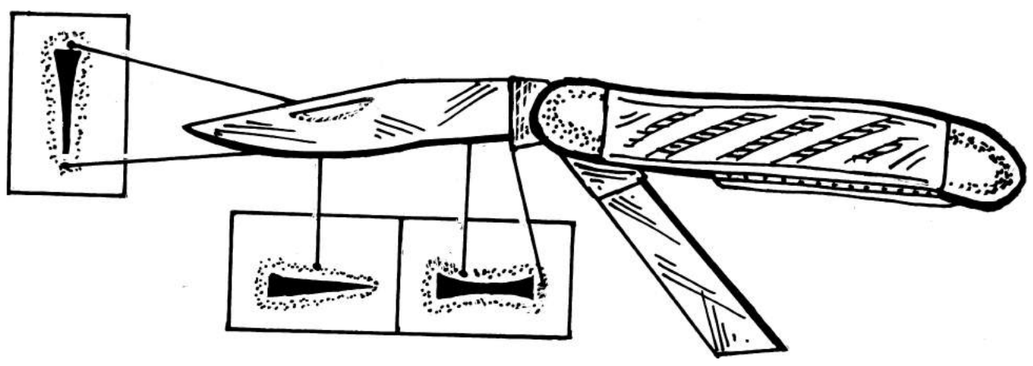


FIGURE 19.19 ■ **Stab Wound.** A stab wound from a single-edged knife blade will impart a sharp edge and a dull edge to the wound. If the blade penetrates to the proximal portion of the blade, a contusion may result from contact with the hilt of the knife.



FIGURE 19.20 ■ **Single-Edge Stab Wound with Hilt Mark.** A single-edged stab wound with a small hilt mark associated with the dull edge of the blade. (Photo contributor: William S. Smock, MD.)



FIGURE 19.21 ■ **Single-Edge Stab Wound.** A stab wound from a single-edged knife blade. The superior portion of the wound corresponds with the dull edge of the blade and the inferior with the sharp edge of the blade. (Photo contributor: William S. Smock, MD.)

skin during insertion or withdraw from the victim. Serrated blades do not always leave these characteristic marks.

When a patient presents with superficial and/or parallel incisions, the wounds must be considered intentionally self-inflicted until proven otherwise (Figs. 19.22 and 19.23). The emergency physician should obtain a more detailed history, including the number of "swipes" the alleged assailant made with the knife. Accidental, self-inflicted incised wounds on the palm or palmar aspect of the fingers are common on the hands of suspects in violent stabbings (Fig. 19.24). The suspects' hand slips down the handle and makes contact with the knife blade when the blade impacts a firm object, like bone (Fig. 19.25).

Pearls

1. Incised wounds have sharp wound margins and are longer than they are deep. Incisions result from contact with a sharp-edged implement: glass, knife blade, razor blade, or scalpel.
2. Stab wounds are puncture wounds that are deeper than they are long.
3. Knife-blade characteristics (single or dual edged, serrated or smooth) can frequently be determined by a visual inspection of the wound.
4. Superficial and/or parallel incisions should be assumed to be self-inflicted.



FIGURE 19.23 ■ **Self-Inflicted Incised Wounds.** Examination of the "victim's" chest reveals multiple superficial incisions. The patient initially claimed she was "assaulted" but later admitted that the wounds were self-inflicted. (Photo contributor: William S. Smock, MD.)

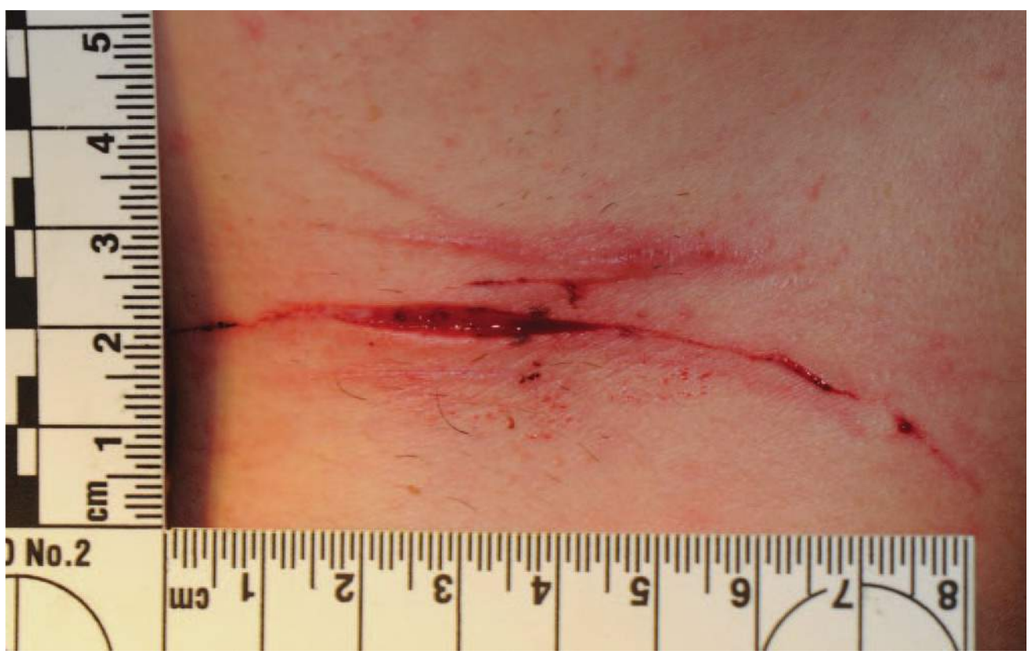


FIGURE 19.22 ■ **Self-Inflicted Incised Wounds.** Superficial and/or parallel incisions are pathognomonic for intentional self-inflicted wounds. This patient has acute and healed self-inflicted incisions in the same location on the anterior lateral aspect of her neck. (Photo contributor: William S. Smock, MD.)



FIGURE 19.24 ■ **Incised Wounds on the Palm.** Suspects in stabbings sometimes have incised wounds on their own palm or the palmar surfaces of their fingers. (Photo contributor: William S. Smock, MD.)



FIGURE 19.25 ■ **Hand Slippage on Knife.** When the knife blade suddenly stops (for example if the blade hits bone) the hand can slip down the handle and make contact with the blade incising wounds on the assailant's palm (Fig. 19.24). (Photo contributor: William S. Smock, MD.)

Clinical Summary

The most common blunt force injury is the contusion (Fig. 19.26). The pattern contusion is one that helps identify the causative weapon. A blow from a linear object leaves a contusion that is characterized by a set of parallel lines separated by an area of central clearing (Fig. 19.27). The blood underlying the striking object is forcibly displaced to the sides, which accounts for the pattern's appearance. Pattern injuries that an emergency physician should recognize include those caused by the hand (slap marks [Fig. 19.27], fingertip contusions [Fig. 19.28], grab marks, ligature marks [Fig. 19.30], fingernail abrasions), solid objects (baseball bat, tire iron, 2 by 4, belt, shoe, comb), and bite marks.

Other manifestations of blunt-force trauma to the skin are the abrasion and the laceration. A weapon with a unique shape or configuration may stamp a mirror image of itself on the skin (Fig. 19.29) and provide the emergency physician with additional information on the mechanism of the injury. The color of a contusion cannot be used to accurately date its age.

Pearls

1. A contusion is the most common blunt-force injury pattern in interpersonal violence.



FIGURE 19.26 ■ **Periorbital Contusion.** A contusion, without a discernable pattern, is the result of some type of blunt-force trauma. Victims of interpersonal violence frequently present with nonspecific, nonpattern contusions. A 24-hour-old cigarette burn is located on the victim's right upper lid. This victim was held against her will for 48 hours and repeatedly beaten about the face. (Photo contributor: William S. Smock, MD.)



FIGURE 19.27 ■ **Slap-Mark Pattern Contusions.** This victim of a "fall from the couch" has a pattern injury present on the right cheek. The pattern of central clearing surrounded by linear parallel contusions is pathognomonic for impact with a rounded linear object, in this case the extended fingers of a hand. (Photo contributor: William S. Smock, MD.)

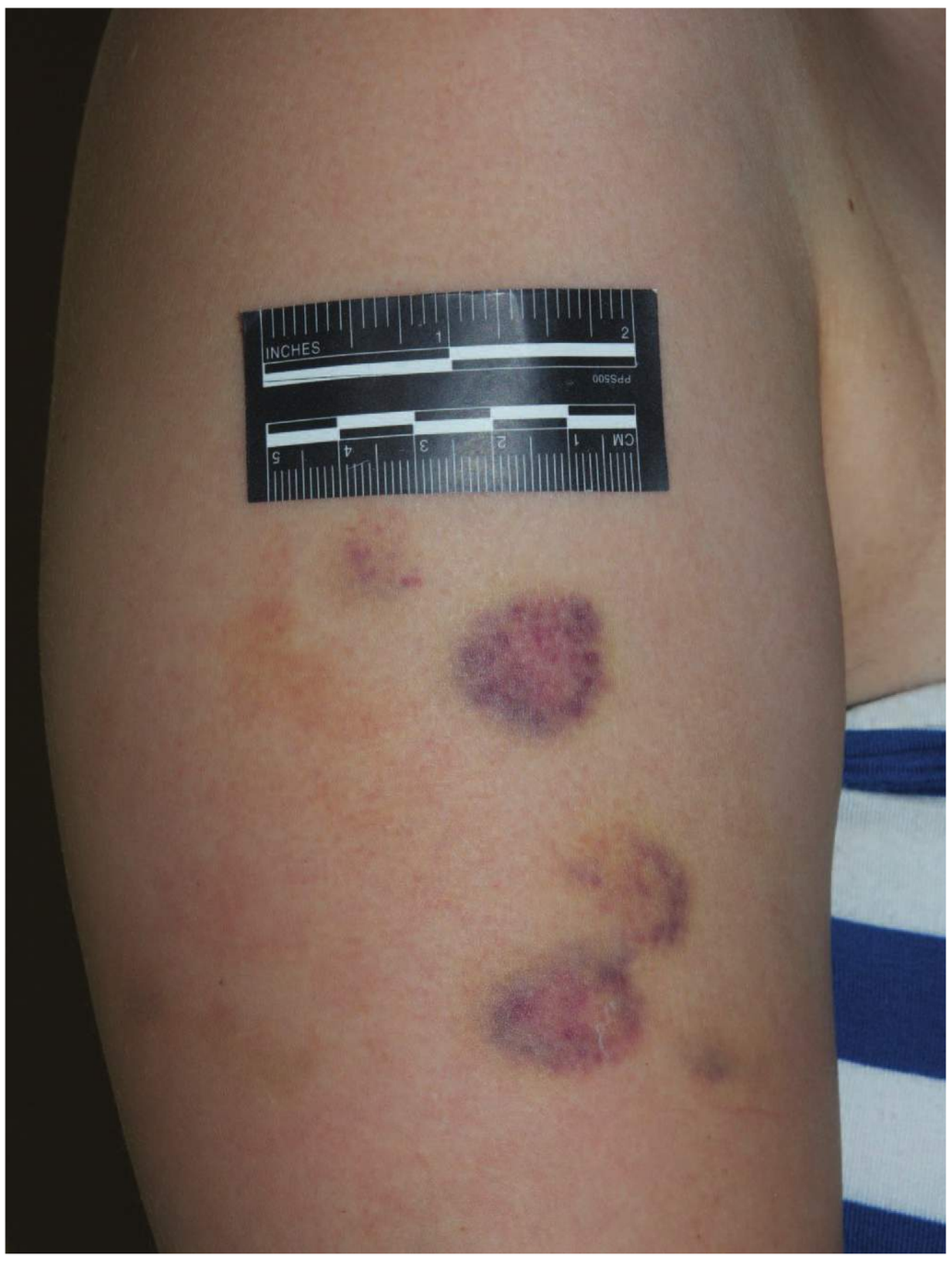


FIGURE 19.28 ■ **Fingertip Contusion Pattern.** This patient exhibits fingertip contusions. The emergency physician should never attempt to date the age of a contusion based upon its color. (Photo contributor: William S. Smock, MD.)

2. Blood underlying the force of the contusion is displaced to either side of the object, causing a pattern contusion in the shape of that object. This pattern is recognized by the central clearing surrounded by linear parallel contusions.
3. Emergency physicians must be able to recognize the pattern injuries caused by the hand (Fig. 19.27), solid objects, and bites.
4. Emergency physicians should not attempt to date the age of a contusion based upon its color or stage of resolution.



FIGURE 19.29 ■ **Tire Mark.** This tire mark, a mirror image of the tire tread, was the only evidence that connected this pedestrian with the vehicle involved in the hit-and-run incident. (Photo contributor: William S. Smock, MD.)

Clinical Summary

Manual strangulation is one of the most lethal forms of intimate partner violence and demonstrates the abuser's power and control over the victim. The brain will suffer an anoxic injury when the flow of oxygenated blood is stopped by occlusion of the carotid arteries. Strangulation victims report visual and auditory changes just prior to a loss of consciousness. The loss of bladder control indicates a deep anoxic insult to the brain of at least 15 seconds in duration. The loss of bowel control occurs with carotid artery occlusions of greater than 30 seconds. The jugular veins can be occluded with 4 psi, the carotids with 11 psi, and the trachea with 34 psi. Occlusion of venous return can cause capillary rupture from increased pressure and the development of petechial hemorrhage (Figs. 19.32-19.34). Petechial hemorrhages can develop in any vascularized tissue, including the brain. The most sensitive areas for petechial development include the conjunctiva, sclera, and intraoral mucosa. The use of an oral-pharyngeal scope (Fig. 19.35) often reveals vocal cord injury and the presence of petechial hemorrhage in the posterior pharynx. With simultaneous occlusion of both the arterial and venous systems, it is possible to induce fatal and nonfatal strangulation events without petechial hemorrhage and evidence of external trauma.



FIGURE 19.31 ■ **Strangulation Ligature Mark.** Ligature marks from the wires of her husband's "ear buds." The victim sustained neurological compromise from the compression of her carotids. (Photo contributor: William S. Smock, MD.)



FIGURE 19.32 ■ **Petechial Hemorrhage.** Extensive facial petechial was found above the area of neck compression in a nonfatal strangulation victim. (Photo contributor: William S. Smock, MD.)



FIGURE 19.30 ■ **Strangulation Ligature Belt Marks.** Ligature marks from a belt, with petechial hemorrhage above the marks, supported the mentally challenged patient's story of being "choked" by her caretaker. The majority of fatal strangulations do not exhibit external evidence of trauma. (Photo contributor: William S. Smock, MD.)

Pearls

1. Strangulation is associated with a 700% increased risk of interpersonal homicide.
2. Application of a ligature (Figs. 19.30-19.31) will leave a characteristic mark due to pressure applied over a small surface area.
3. Strangulation-induced petechial hemorrhage, from the rupture of venous vessels, may be found in the conjunctiva (Fig. 19.32), the soft palate, the posterior pharynx (Fig. 19.33), and above and below the area where pressure was applied (Fig. 19.34). Petechial hemorrhage is NOT required to prove strangulation. The majority of fatal strangulations exhibit no external evidence of trauma.
4. A victim of strangulation can be rendered unconscious in less than 8 seconds with bilateral occlusion of the carotid arteries. The carotids will be occluded with 11 pounds of pressure. Obtaining a history of neurological changes—loss of hearing, vision, consciousness, and bowel or bladder control—confirms neurologic injury and an anoxic insult to the brain.



FIGURE 19.33 ■ **Subconjunctival and Scleral Petechial Hemorrhage.** Increased venous pressure from the occlusion of the jugular veins during manual strangulation results in ruptured capillaries. (Photo contributor: William S. Smock, MD.)

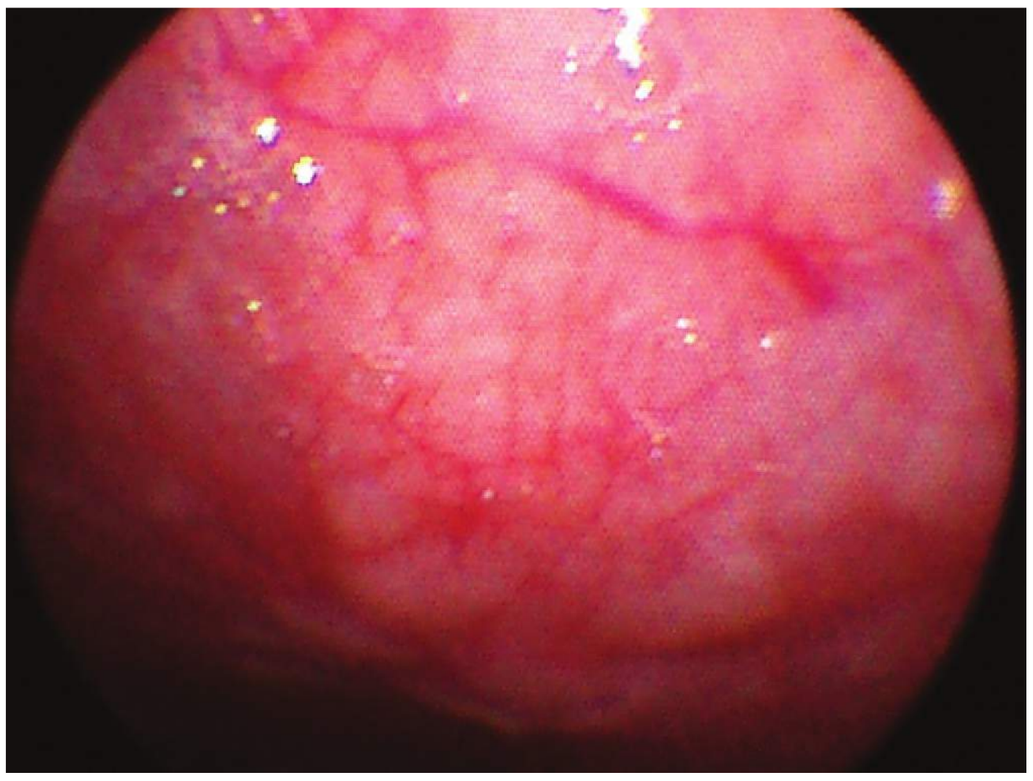


FIGURE 19.34 ■ **Petechial Hemorrhage in Posterior Pharynx.** Petechial hemorrhage in the posterior pharynx is a common finding in victims of strangulation. (Photo contributor: William S. Smock, MD.)



FIGURE 19.35 ■ **Oral-Laryngeal Scope.** An oral-laryngeal scope, equipped with a camera, is an excellent tool to document the extent of petechial hemorrhage in the pharynx and vocal cord injury in victims of strangulation. (Photo contributor: William S. Smock, MD.)

THERMAL-PATTERN INJURIES

Clinical Summary

Thermal pattern injuries are commonly seen in cases of abuse and assault especially when the victims are children and the elderly. A detailed history of the incident should include the position of the patient relative to the thermal source. This will help determine whether the injury was inflicted or accidental. Thermal injuries characterized by a distinct pattern that frequently present to emergency departments are those caused by cigarettes (Fig. 19.26), flatirons (Fig. 19.37), curling irons (Fig. 19.38), and scalding hot water (Fig 19.40). Water burns will be of two natures: immersion and splash. A sharp or clear line of demarcation between burned and unburned tissue characterizes an immersion or dipping burn. In contrast, splash burns are characterized by an irregular or undulating line or by isolated areas of thermal injury, usually round or oval in shape, caused by droplets of hot liquid. The severity of the scald injury depends upon two variables: the length of time that the skin was in contact with the offending substance and the temperature of the substance itself. Tap or faucet water causes full-thickness thermal damage in 1 second at 70°C (158°F), and 180 seconds at 48.9°C (120°F). Law-enforcement agencies should routinely measure the household's or institution's water temperature in any investigation involving a scald injury of a child, a developmentally delayed person, or an elderly one. The water temperature will give authorities an estimate of the duration of the insult and therefore the degree of malice of intention on the part of the assailant.

Pearls

1. A thermal-pattern injury is a common form of abuse seen in infants, institutionalized patients, and the elderly.
2. Emergency physicians must recognize thermal-pattern injuries of abuse.



FIGURE 19.37 ■ **Clothes-Iron Thermal Injury Pattern.** An iron was the weapon used to inflict this thermal injury. The areas of sparing are associated with its steam holes. (Photo contributor: William S. Smock, MD.)



FIGURE 19.36 ■ **Cigarette Thermal Injury Pattern.** A 24-hour-old cigarette burn is located on this victim's right upper lid. This victim was held against her will for 48 hours and repeatedly beaten about the face. (Photo contributor: William S. Smock, MD.)



FIGURE 19.38 ■ **Curling Iron Thermal Injury Pattern.** Curling irons can reach more than 200°F. This temperature can cause a full-thickness burn in less than 1 second. When this burn to the anterior surface of the leg was inflicted, someone's thumb was on the spring lever—evidence of intent (Fig. 19.39). (Photo contributor: William S. Smock, MD.)



FIGURE 19.39 ■ **Curling Iron.** The V-shaped configuration of the pattern injury (Fig. 19.38) indicated that the assailant's thumb was on the spring-loaded lever when the burn was inflicted. This was evidence of intent. The victim's aunt confessed to inflicting the burn. (Photo contributor: William S. Smock, MD.)



FIGURE 19.40 ■ **Immersion-Line Thermal Injury Pattern.** A characteristic "immersion line" is seen in a thermal-pattern injury. The line of demarcation is associated with the depth of the immersion. (Photo contributor: William S. Smock, MD.)

Chapter 20

HIV CONDITIONS

Stephen P. Raffanti
Anna K. Person



Kaposi Sarcoma. Multiple KS lesions surrounding the nipple of this patient with advanced HIV. (Photo contributor: Seth W. Wright, MD.)

The authors would like to acknowledge the special contributions of J. Michael Ballister, MD, Roderick Morrison, MD, Shane Cline, MD, and Michael Krentz, MD, for their excellent work on prior editions of this chapter.

PRIMARY HIV INFECTION

Clinical Summary

Establishing a definitive diagnosis in the earliest stage of HIV infection can have significant impact on the patient's outcome and may serve a public health function as well. Patients are highly infectious during acute seroconversion and may be unaware that they are infected. It is believed that a significant number of the new cases of HIV infection occurring in the United States are acquired from acutely infected individuals. Identifying acute seroconverters could offer the opportunity to discuss transmission reduction behavior. Clinical illness accompanies primary HIV infection in approximately two-thirds of patients. The usual time from HIV exposure to the development of symptoms is approximately 10 to 20 days, with average symptom duration of 1.5 to 2 weeks. The most common symptoms following seroconversion mimic a typical viral syndrome and may include mucocutaneous lesions and a generalized maculopapular rash located over the face, neck, and trunk. The rash is seen in over 50% of persons with symptomatic primary HIV infection. The lesions are typically small, well circumscribed, erythematous, nonpruritic, and nontender. Less frequently, patients may demonstrate neurologic signs and symptoms consistent with meningoencephalitis, myelopathy, and peripheral neuropathy. If obtained, laboratory studies may show lymphopenia and thrombocytopenia.

Management and Disposition

Although rapid HIV testing has become feasible in most emergency department (ED) settings, the diagnosis of acute infection cannot rely solely on rapid antibody-based tests. If the possibility of acute seroconversion is entertained, then a discussion should take place with the patient regarding level of risk behavior. If acute HIV is suspected, HIV-1 ribonucleic acid (RNA) quantitative polymerase chain reaction (PCR) is the appropriate diagnostic test. Patients who are acutely seroconverting should have extremely high levels of HIV RNA. Importantly, a negative HIV antibody test does not rule out HIV infection in these cases. During the "window period" of acute HIV infection, antibodies against the HIV virus have not yet formed and may not be detected for 8 to 10 weeks. If acute HIV is suspected or confirmed, patients should be educated about disease transmission and referred for prompt follow-up and further outpatient testing and evaluation.

Pearls

1. Maintain a high degree of clinical suspicion for acute primary HIV infection, especially when sexually active patients or patients who use intravenous drugs present with mononucleosis-like symptoms, unexplained rash, mucocutaneous ulcers, lymphadenopathy, or aseptic meningitis.
2. Obtain an HIV RNA quantitative PCR (and not just an HIV antibody test) if acute HIV is suspected.
3. Ensure proper follow-up for patients in whom the diagnosis of acute HIV infection is entertained, and counsel patients regarding potential risk of transmission.



FIGURE 20.1 ■ **Primary HIV Infection.** A maculopapular rash is seen in over half of persons with symptomatic acute HIV infection. This less typical papular/vesicular rash was present in a patient with primary HIV infection. (Photo contributor: Gregory K. Robbins, MD, MPH.)

IMMUNE RECONSTITUTION INFLAMMATORY SYNDROME (IRIS)

Clinical Summary

Effective antiretroviral therapy establishes virologic suppression and can allow the patient's immune system to rebuild to a near-normal competency. In some patients who develop a rapid immune response after a very advanced degree of immune suppression, the immune reconstitution inflammatory syndrome (IRIS) can develop. In these patients, it is thought that previously subclinical infections manifest with the development of a more robust immune status. IRIS is most often seen in patients who have recently started antiretroviral therapy (usually within 60 days of starting therapy) with an initial CD4 count below 100 cells/mm^3. Most common infections that can "trigger" IRIS include mycobacterium (tuberculosis [TB] and mycobacterium avium complex [MAC]), cytomegalovirus (CMV), cryptococcal disease, and histoplasmosis. Patients typically present with fever, malaise, lymphadenopathy, and symptoms associated with the active opportunistic infection.

Management and Disposition

The key for the ED physician is to identify those patients who have recently started antiretroviral therapy and may be presenting with IRIS-related findings. Appropriate evaluation and management depends on the underlying opportunistic infection. Patients will often require admission to the hospital and consultation with an infectious diseases specialist.

Pearl

1. HIV-infected patients who are currently taking antiretroviral therapy and present with a febrile illness should be questioned regarding the duration of antiretroviral therapy and pretreatment CD4 count. Patients with advanced disease who have recently started antiretroviral therapy may be presenting with signs and symptoms of IRIS-related disease.



FIGURE 20.2 ■ **IRIS.** This patient presented with fever and diffuse lymphadenopathy secondary to MAC infection. The patient had no manifestations of his disseminated disease until being started on antiretroviral therapy. (Photo contributor: Stephen P. Raffanti, MD.)

ORAL HAIRY LEUKOPLAKIA

Clinical Summary

Oral hairy leukoplakia (OHL) is a disease of the lingual squamous epithelium caused by the Epstein-Barr virus (EBV). OHL generally affects the lateral portion of the tongue, although the floor of the mouth, palate, or buccal mucosa may also be involved. The lesions are white corrugated plaques that, unlike Candida, cannot be scraped from the surface to which they adhere. Most often OHL is asymptomatic, although occasionally this condition can be painful. Diagnosis is usually clinical, though definitive diagnosis can be made by biopsy characteristically revealing acanthosis and parakeratosis.



FIGURE 20.3 ■ **Oral Hairy Leukoplakia.** Typical-appearing lesions on side of tongue in this patient with HIV. (Photo contributor: Robert Brandt, MD.)

Management and Disposition

Patients who are known to be HIV-seropositive can be educated about the disease and reassured. OHL is not considered to be a premalignant lesion. If the patient is not known to be infected with HIV, primary care provider referral and HIV screening, if available, should be offered.



FIGURE 20.4 ■ **Oral Hairy Leukoplakia.** Exudate does not scrap off the tongue in oral hairy leukoplakia, differentiating it clinically from oral thrush. (Photo contributor: Kevin J. Knoop, MD, MS.)

Pearls

1. Oral candidiasis can be distinguished from OHL by utilizing a swab in an attempt to remove the exudate characteristic of thrush and by observing pseudohyphal elements microscopically with candida. OHL cannot be scraped off and usually involves the lateral aspect of the tongue.
2. OHL is fairly specific for HIV infection as it is rarely observed in patients with other immunodeficiencies. If OHL is identified in a patient not known to be HIV infected, risk factors and HIV screening options should be discussed.

Clinical Summary

Oral candida infections are seen in over half of all HIV patients, with the severity of infection often correlating with the degree of immunosuppression. Oral candidiasis can occur at all stages of HIV disease. The usual causative agent is Candida albicans, but other Candida species have been isolated. Oral candidiasis, or "thrush," can be classified as pseudomembranous, angular, or erythematous. Pseudomembranous candidiasis can be diagnosed by identifying removable whitish plaques on the tongue, uvula, and buccal mucosa. Erythematous or atrophic candidiasis appears as smooth red patches along the soft and hard palate. Although isolated oral candidiasis is not an AIDS-defining illness (though esophageal candidiasis is), oral candidiasis is an indication for pneumocystis prophylaxis regardless of CD4+ cell count.

Vaginal candidiasis is common in HIV positive patients and can cause a severe whitish discharge with vulvar erythema.

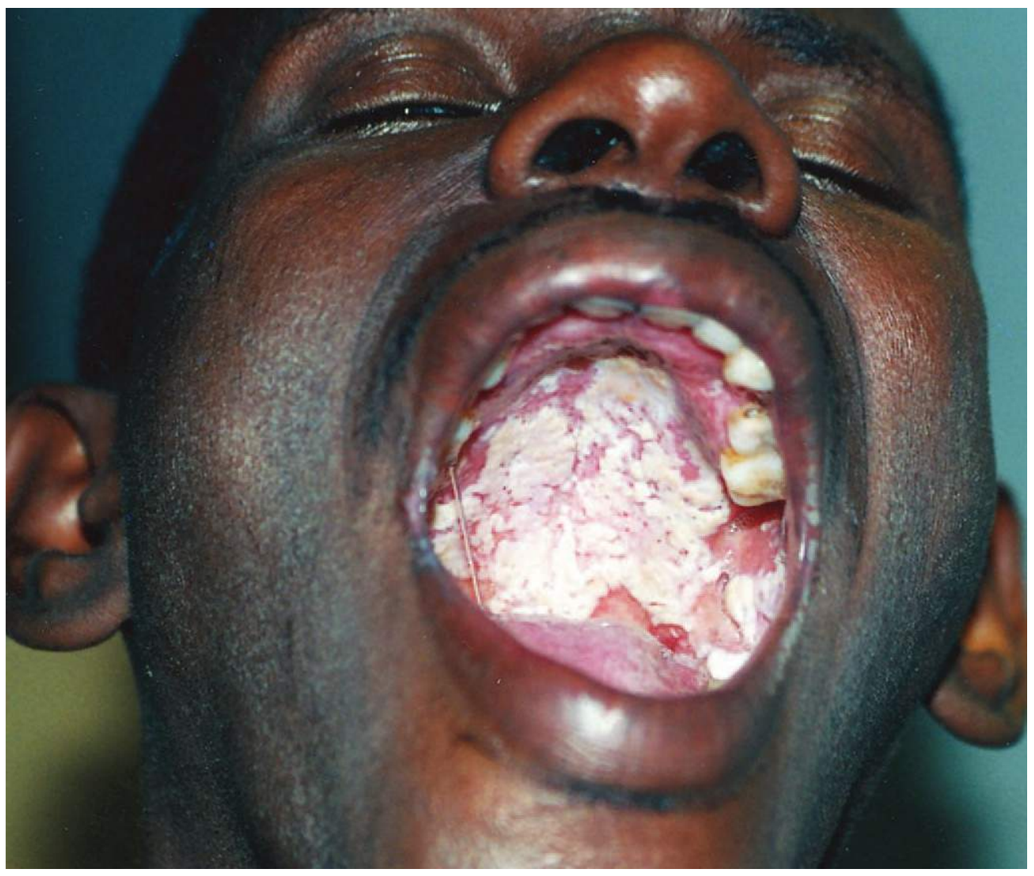


FIGURE 20.5 ■ **Oral Candidiasis.** Removable whitish plaques on the palate are seen in this HIV patient with pseudomembranous candidiasis. (Photo contributor: Thea James, MD.)

Management and Disposition

Patients presenting to the ED for dysphagia or odynophagia thought to be due to candidiasis should be evaluated for dehydration and may require inpatient admission. Most cases of minimally symptomatic oral candidiasis respond well to nystatin suspension or clotrimazole troches. More symptomatic oral or esophageal candidiasis usually responds to fluconazole. Rare cases of resistant candidiasis may require intravenous echinocandin therapy.

Pearls

1. Symptomatic oral and esophageal candidiasis is still a common ED presentation in HIV patients who are not receiving or are not compliant with effective antiretroviral therapy.
2. The diagnosis of oral candidiasis in a patient not already diagnosed with HIV infection should lead to a discussion of risk factors and should prompt HIV screening.
3. Mildly symptomatic oral, esophageal, or vaginal candidiasis usually responds to a one-time oral dose of fluconazole. Close clinical follow-up should be arranged.



FIGURE 20.6 ■ **Esophageal Candidiasis.** Endoscopy demonstrating esophageal candidiasis in this HIV patient. (Photo contributor: Edward C. Oldfield III, MD.)



FIGURE 20.7 ■ **Oral Thrush.** Typical plaque lesions are seen on the palate and uvula of this HIV patient. (Photo contributor: Seth W. Wright, MD.)

Clinical Summary

Kaposi sarcoma (KS) is a low-grade vascular tumor associated with human herpesvirus 8 (HHV-8). Since the introduction of highly active antiretroviral therapy (HAART), the incidence of KS in HIV-infected persons has significantly declined. Skin involvement is characteristic but extracutaneous spread of KS is common, particularly to the oral cavity, gastrointestinal (GI) tract, and respiratory tract. The skin lesions appear most often on the lower extremities, face (especially the nose), oral mucosa, and genitalia. Most commonly, the lesions are papular, ranging in size from several millimeters to centimeters in diameter. Less commonly, the lesions may be plaque-like, especially on the soles of the feet, or exophytic and fungating with breakdown of overlying skin. Papular lesions may resemble lesions associated with bacillary angiomatosis or herpesvirus infections.

Pulmonary involvement can occur in AIDS-related KS. Affected persons may present with shortness of breath, fever, cough, hemoptysis, or chest pain or as an asymptomatic finding on chest x-ray. Diagnosis can be confirmed via bronchoscopy.

Management and Disposition

All HIV-infected patients with KS should receive antiretroviral therapy, which is generally effective in treating most cutaneous lesions. Systemic chemotherapy and newer immunomodulator therapy (interleukin-12, angiogenesis inhibitors) are reserved for more extensive disease including patients with visceral involvement.

Pearls

1. HIV patients who present with new papular lesions should be referred for dermatologic evaluation and biopsy.
2. A careful examination of the skin and oral cavity is appropriate in HIV-infected patients presenting with GI or respiratory complaints.
3. Half of patients with oral involvement have other GI tract lesions as well. Hemoccult testing of stool is a good preliminary screen.



FIGURE 20.8 ■ **Kaposi Sarcoma.** A single violaceous patch is seen on the face of an HIV-positive patient. (Photo contributor: George Turiansky, MD.)



FIGURE 20.9 ■ **Cutaneous Kaposi Sarcoma.** Characteristic non-raised violaceous Kaposi sarcoma lesions on the arm of this Caucasian patient with HIV. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)



FIGURE 20.10 ■ **Kaposi Sarcoma.** Multiple black KS lesions, as typically seen in darker skinned patients. (Photo contributor: Seth W. Wright, MD.)



FIGURE 20.11 ■ **Kaposi Sarcoma.** Multiple KS lesions surrounding the nipple of this patient with advanced HIV. (Photo contributor: Seth W. Wright, MD.)



FIGURE 20.12 ■ **Oral Kaposi Sarcoma—Tongue.** Raised palpable erythematous Kaposi sarcoma on the tongue of this HIV patient. (Photo contributor: Seth W. Wright, MD.)

Clinical Summary

Toxoplasma gondii is a widespread intracellular protozoan parasite with a definitive host stage in cats. Immunocompetent persons are usually asymptomatic, but immunocompromised persons (especially HIV-positive persons with <100 CD4 cells/μL) are susceptible to reactivation of latent disease with considerable morbidity and mortality.

Patients with central nervous system (CNS) toxoplasmosis most often present with symptoms consistent with intracranial mass lesions (headache, focal neurologic deficit, nausea/emesis, and/or seizure) or, less likely, encephalitis (fever, confusion, altered mental status). Contrasted computed tomography (CT) of the head typically reveals ring-enhancing lesions with a predilection for the basal ganglia. Magnetic resonance imaging (MRI) of the head is more sensitive than CT for identifying these lesions. Serological tests for T gondii antibodies (IgG) are usually positive. Other causes of mass lesions in the brain in HIV-infected individuals include CNS lymphoma, cryptococcoma, tuberculoma, and brain abscess.

Ocular toxoplasmosis is a less frequent complication of HIV disease. Patients typically present with eye pain and decreased visual acuity. Retinitis may be diagnosed by ophthalmoscopic evaluation revealing characteristic exudates and hemorrhage. Toxoplasmosis retinitis appears as raised yellow-white, cottony lesions in a nonvascular distribution, unlike the edematous perivascular exudates of CMV retinitis.

Management and Disposition

There are two combination regimens considered to be first choice for the treatment of toxoplasmosis. The most commonly used regimen is pyrimethamine and sulfadiazine for 6 weeks or until neurological findings have resolved; however, for those with sulfa allergies, pyrimethamine and clindamycin is an alternative. If the patient presents with seizures, loading doses of antiepileptics such as fosphenytoin or levatiracetam should be administered along with benzodiazepines. All patients with acute symptomatic CNS toxoplasmosis infection should be admitted for treatment.

In cases of suspected ocular toxoplasmosis, a complete eye examination including slit-lamp examination and measurements of intraocular pressure should be performed before consulting ophthalmology.

Pearls

1. HIV-infected patients presenting with focal neurologic findings or seizures should be evaluated for meningitis and space-occupying lesions in the brain. AIDS-specific differential includes toxoplasmic encephalitis, CNS lymphoma, and cryptococcal disease.
2. All HIV-infected patients should have a CNS space–occupying lesion excluded by imaging prior to lumbar puncture (LP).
3. Initial evaluation of the HIV-infected patient with possible CNS lesions should include CT or MRI imaging, LP, and triage management for bacterial meningitis. Further workup will require admission and infectious diseases consultation.



FIGURE 20.13 ■ Toxoplasma gondii **Infection.** Contrast head CT showing typical multiple ring-enhancing lesions seen in T gondii CNS infection. (Photo contributor: Edward C. Oldfield III, MD.)

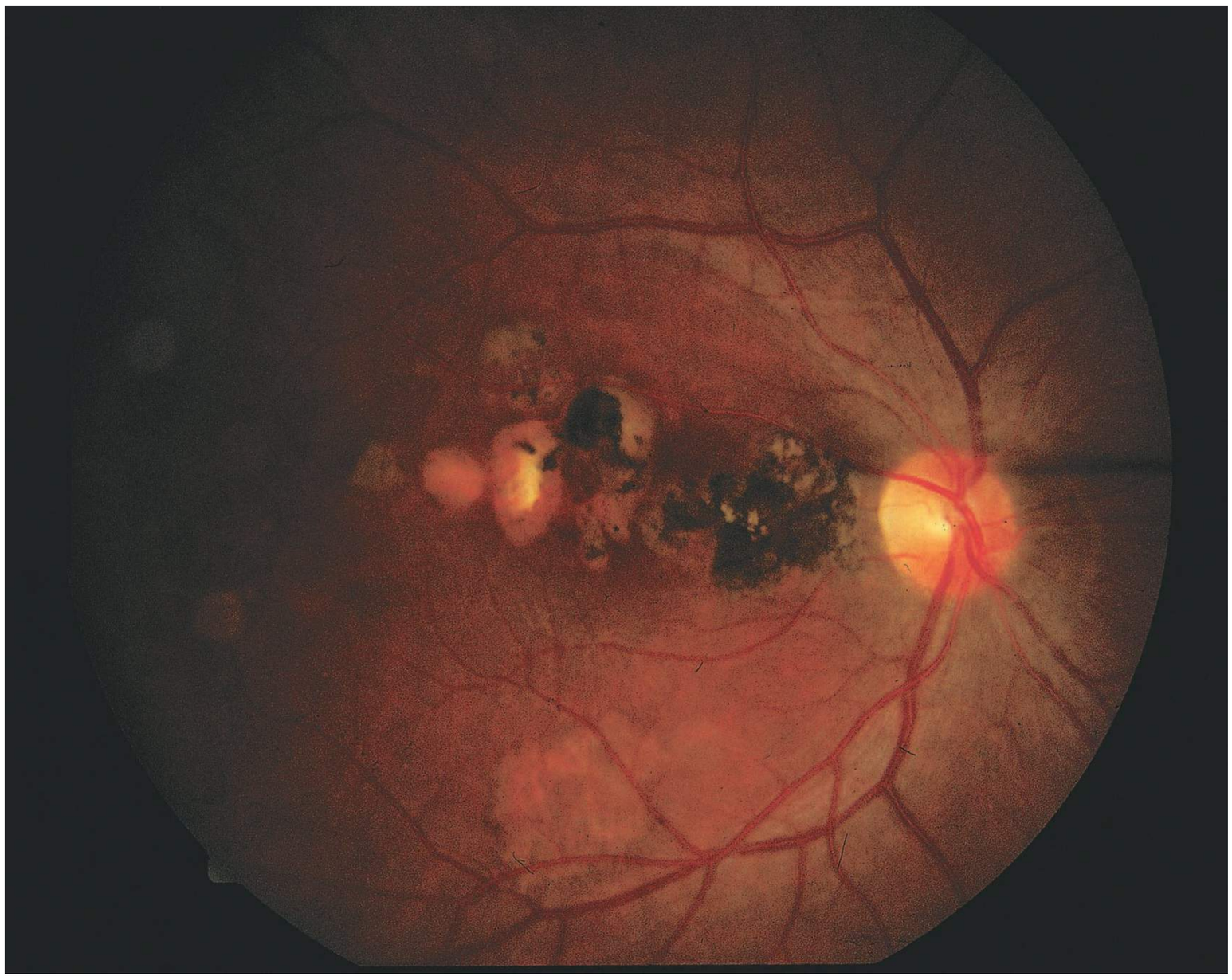


FIGURE 20.14 ■ **Toxoplasmosis Retinitis.** Ocular toxoplasmosis is a common complication of HIV disease. The lesion is a focal destructive chorioretinitis which leaves well-defined, heavily pigmented scars, especially in the macular area. (Photo contributor: Department of Ophthalmology, Naval Medical Center, San Diego, CA.)

Clinical Summary

Pneumocystis jirovecii (formerly carinii) pneumonia (PCP) is the most common opportunistic infection in HIV-infected patients. Clinical suspicion for PCP pneumonia in any HIV patient presenting with complaints of dyspnea and nonproductive cough should remain high. Presentations can be indolent, acute, or subacute, with associated symptoms including fever, fatigue, anorexia, weight loss, and chest pain. The complete blood count (CBC) is usually normal except for lymphopenia, while serum lactate dehydrogenase (LDH) is often elevated. Arterial blood gases (ABGs) most often reveal a respiratory alkalosis, Po_2 of 70 mm Hg or less, and an increased A-a gradient of 35 mm Hg or more. Radiographic chest findings are variable with diffuse interstitial alveolar infiltrates being common.

Management and Disposition

The initial treatment of PCP is often based on a presumptive diagnosis pending definitive results, usually a stained bronchial lavage sample obtained through bronchoscopy. It is essential that any HIV-infected patient who may have a CD4 count below 200 cells/mm^3 or CD4 cell percent less than 14 presenting with respiratory signs or symptoms be evaluated for PCP. Initial evaluation should include history of medications, especially adherence with prophylactic medications, clinical exam, chest x-ray, and room air ABG. Trimethoprim-sulfamethoxazole, orally or intravenously, is the first-line standard treatment. Clindamycin-primaquine, pentamidine, trimetrexate-leucovorin, atovaquone, and trimethoprim-dapsone are less effective alternatives. Treatment with corticosteroids should be initiated if the Po_2 is less than 70 or the A-a gradient is greater than 35. Any patient treated with steroids for presumptive PCP must have a definitive diagnosis made before discharge. Other pathogens that can cause pneumonia in HIV-infected patients may initially respond to steroid therapy.

Pearls

1. Include PCP in the differential diagnosis of any HIV patient who presents with a persistent fever or respiratory complaint.
2. Patients with significant dyspnea should have a room air ABG obtained. Oxygen saturation measures are not adequate to evaluate neither the oxygenation level nor the need for adjunct steroid therapy in a patient with suspected PCP.
3. The classic clinical presentation of PCP is a quiet patient with an increased respiratory rate, poor air movement, and a nonproductive cough on deep inspiration.
4. Over 30% of patients with PCP will have a normal chest x-ray. CT scanning of the chest is much more sensitive to reveal the classic interstitial ground glass pattern.

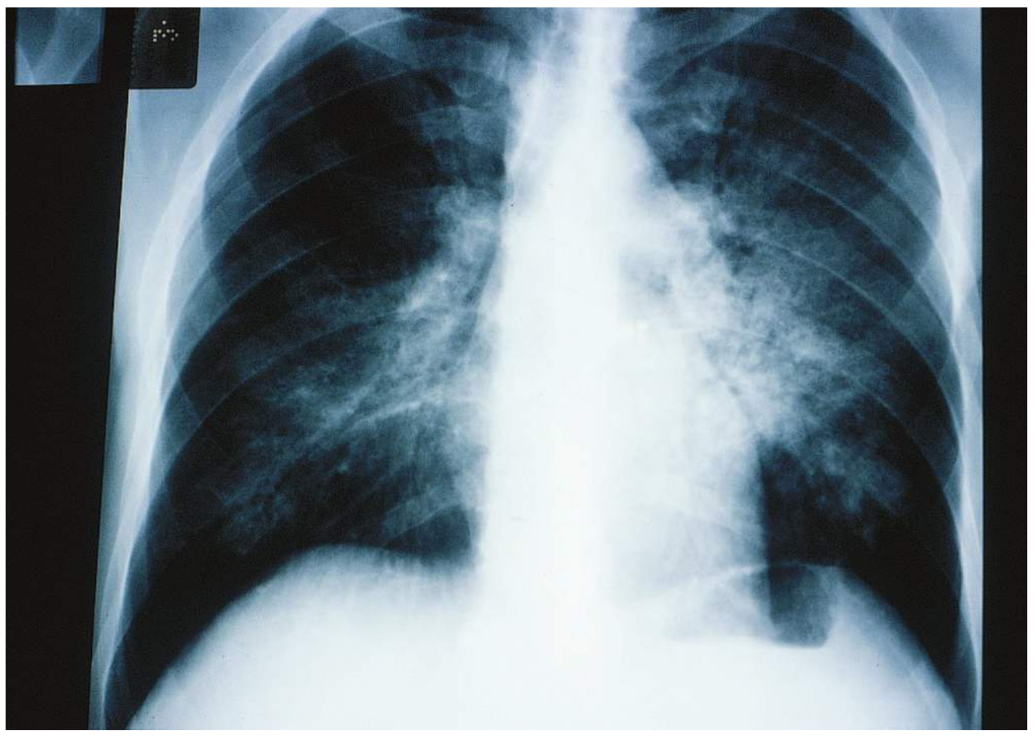


FIGURE 20.15 ■ Pneumocystis jirovecii **Pneumonia (PCP).** Chest radiograph showing diffuse interstitial alveolar infiltrates of PCP. (Photo contributor: Edward C. Oldfield III, MD.)

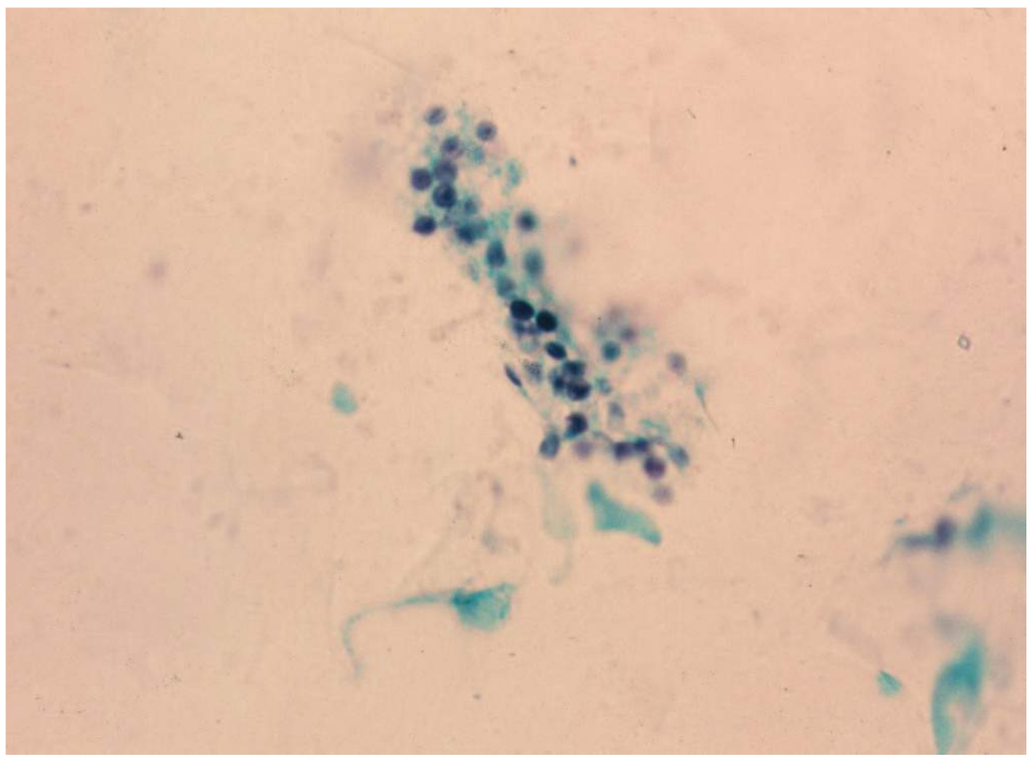


FIGURE 20.16 ■ **PCP on Silver Stain.** Pneumocystis jirovecii seen on methenamine-silver stain of a bronchoalveolar lavage sample from an HIV patient with PCP. (Photo contributor: Lawrence B. Stack, MD.)

CRYPTOCOCCAL INFECTIONS

Clinical Summary

Cryptococcus neoformans is the most common cause of meningitis in patients with AIDS. Cryptococcal meningoencephalitis typically manifests itself in patients whose CD4 cell counts are less than 50/μL. The onset tends to be insidious with fairly nonspecific symptoms such as fever, nausea, and headache. Symptoms may be present for several weeks. Seizures or focal neurological presentations are rare and neck stiffness and/or photophobia are usually absent. Diagnosis is usually made on examination of cerebrospinal fluid (CSF). Opening pressures may be quite elevated on LP, and CSF values usually reveal a normal CSF glucose concentration, a mildly elevated CSF protein concentration, and a CSF leukocyte count of less than 20/mL. India ink staining shows the organisms directly with an approximate sensitivity of 70%, while CSF cryptococcal latex antigen testing has a sensitivity approaching 90%. Fungal CSF cultures should also be sent. Cutaneous manifestations are seen in disseminated disease.

Management and Disposition

Treatment should begin for presumptive meningitis pending completion of CSF studies. Most often, the decision to treat for cryptococcal meningitis will be based on the results of CSF studies, along with consultation with infectious disease specialists. Cryptococcus can also cause pneumonia and skin lesions. Treatment regimens are usually based on amphotericin preparations followed by fluconazole.

Pearls

1. Perform the LP only after CT with contrast rules out space-occupying lesions, and do so with the patient in a lateral decubitus position so as to obtain an accurate opening pressure.
2. "False-positive" India ink stains can occur with other encapsulated organisms such as Klebsiella pneumoniae, Rhodotorula, Candida, and Proteus species.



FIGURE 20.17 ■ **Cryptococcal Infection.** Cryptococcal skin lesions in disseminated form. Note that the umbilicated centers give a similar appearance to that of molluscum contagiosum. (Photo contributor: Briana Hill, MD.)



FIGURE 20.18 ■ **Cryptococcal Infection.** Cutaneous cryptococcal lesions are often clinically difficult to distinguish from other cutaneous eruptions. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)



FIGURE 20.19 ■ **Cryptococcal Infection.** This patient had extensive cutaneous involvement with disseminated cryptococcal infection. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

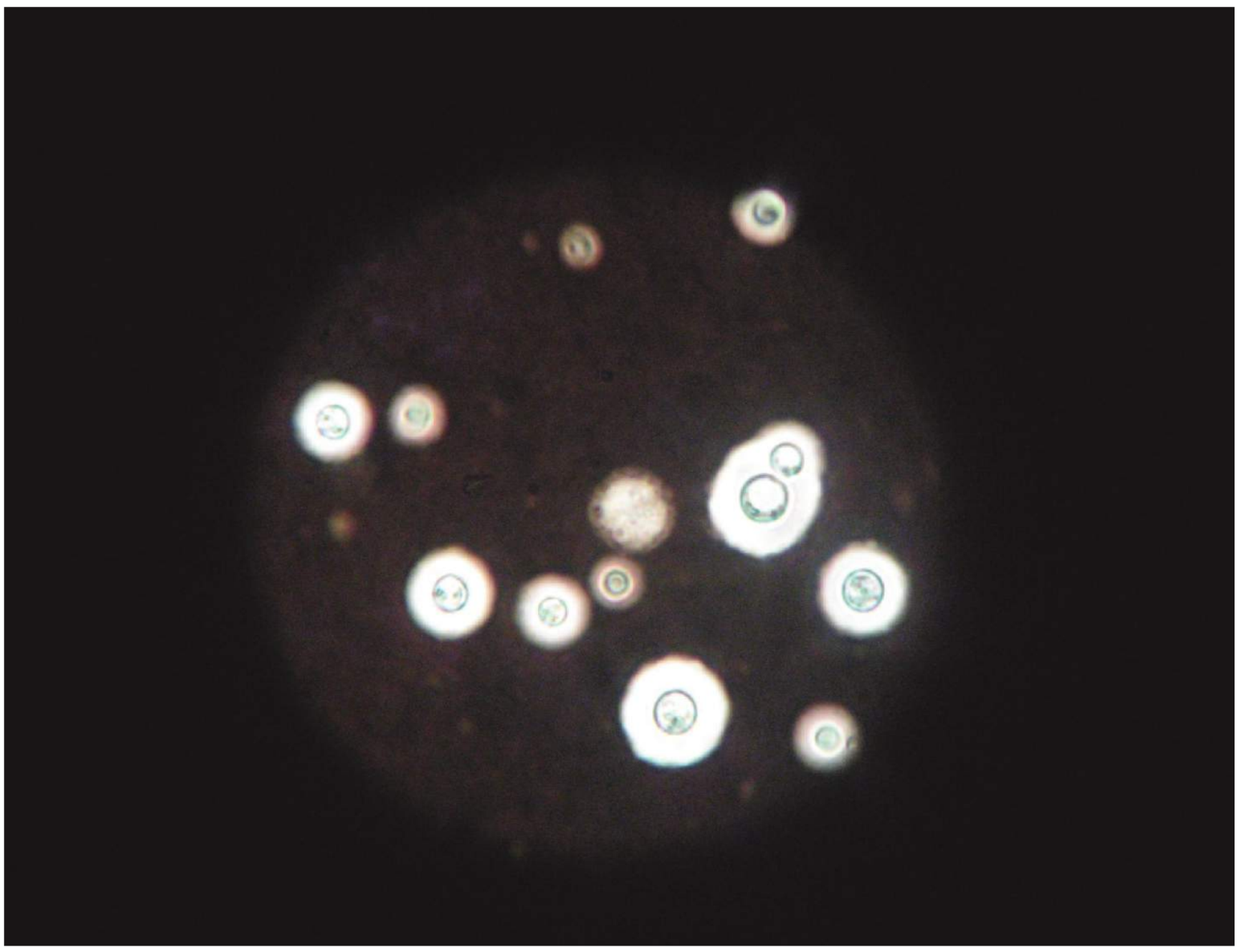


FIGURE 20.20 ■ **India Ink Preparation.** Cryptococcus neoformans seen as encapsulated yeast on India ink preparation of the CSF from an HIV patient with cryptococcal meningitis. (Photo contributor: Seth W. Wright, MD.)

HISTOPLAMOSIS

Clinical Summary

Disseminated histoplasmosis most commonly occurs in immunocompromised patients living in the Ohio and Mississippi River Valleys. HIV-infected patients usually have a CD4 count <200 cells/mm^3 and present with fever, weight loss, malaise, and pulmonary symptoms such as cough and dyspnea. Pulmonary symptoms predominate and in severe cases patients may present with acute respiratory distress syndrome. Patients may present with a diffuse papular rash in disseminated disease. Mediastinitis is a rare but serious complication of histoplasmosis. Urine and serum antigen testing carries a high sensitivity, but bronchoalveolar lavage may be needed to make the diagnosis. Serum LDH and alkaline phosphatase levels may be markedly elevated as well.

Management and Disposition

Disseminated histoplasmosis should be considered in any patient with moderate to advanced HIV infection and constitutional symptoms. Acutely ill patients with fever, weight loss, and functional decline should be admitted for evaluation treatment. Some patients with less severe disease may be treated with oral itraconazole but should be referred for evaluation and a definitive diagnosis.

Pearls

1. Consider disseminated histoplasmosis in any patient with moderate to advanced HIV infection and constitutional symptoms, rash, and appropriate geographic exposure.



FIGURE 20.21 ■ **Disseminated Histoplasmosis.** Skin lesions indicative of cutaneous involvement in an AIDS patient with disseminated histoplasmosis. (Photo contributor: Stephen P. Raffanti, MD.)



FIGURE 20.22 ■ **Pulmonary Histoplasmosis.** Chest x-ray demonstrating severe miliary histoplasmosis in a patient with AIDS. The patient presented with fever, cough, and respiratory distress. (Photo contributor: Jake Block, MD.)

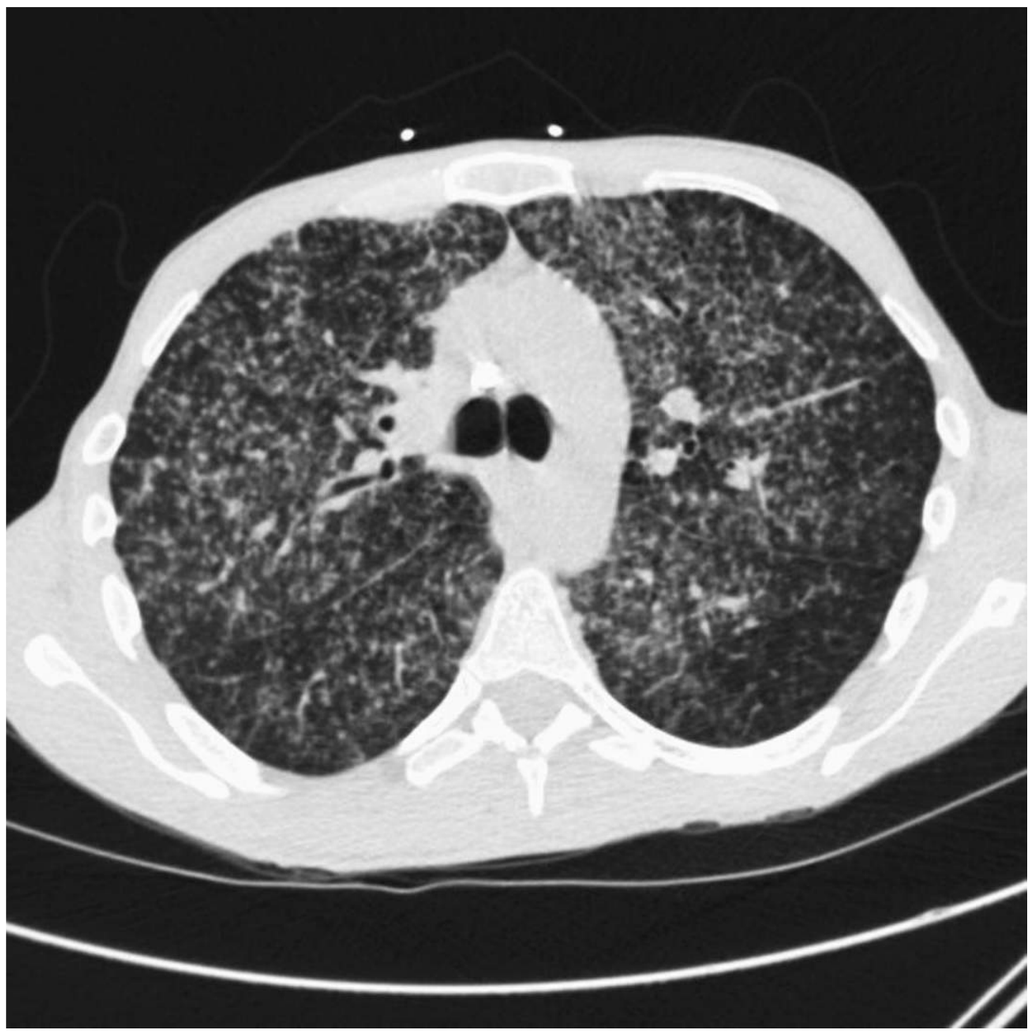


FIGURE 20.23 ■ **Pulmonary Histoplasmosis.** CT scan from the same patient in Fig. 20.22. (Photo contributor: Jake Block, MD.)

Clinical Summary

Cytomegalovirus (CMV) can be a cause of considerable morbidity in severely immunocompromised HIV-infected individuals. The major problems encountered are, in order of frequency, retinitis, colitis, esophageal ulceration, encephalitis, and pneumonitis.

CMV retinitis is the most common cause of blindness and eye disease in patients with AIDS. Patients typically present with loss of vision, floaters, scotoma, or visual field loss. Fundoscopic examination reveals exudates and hemorrhages, which follow the vasculature of the retina, giving it the typical "pizza pie" or "cottage cheese and ketchup" appearance.

CMV colitis is an uncommon but serious complication of HIV. The usual presenting features include generalized abdominal pain, diarrhea which may be bloody, and a low-grade fever. Loops of dilated large bowel may be seen on abdominal imaging, but definitive diagnosis is made with mucosal biopsy revealing characteristic "owls eye" inclusion bodies.

Management and Disposition

Treatment for CMV retinitis can be sight saving and should be started as soon as the diagnosis is considered. Emergent ophthalmologic consultation is needed. Treatment can be initiated with intravenous, intravitreal, or oral ganciclovir formulations. Relapse is common with CMV retinitis and if left untreated may progress to complete blindness.

Pearls

1. Ocular complaints in HIV patients require a comprehensive eye examination along with ophthalmology involvement if lesions are observed or complaints are significant.
2. Patients with CMV esophagitis often complain of odynophagia, but rarely complain of dysphagia, which may help clinically differentiate between CMV and candidal esophagitis.

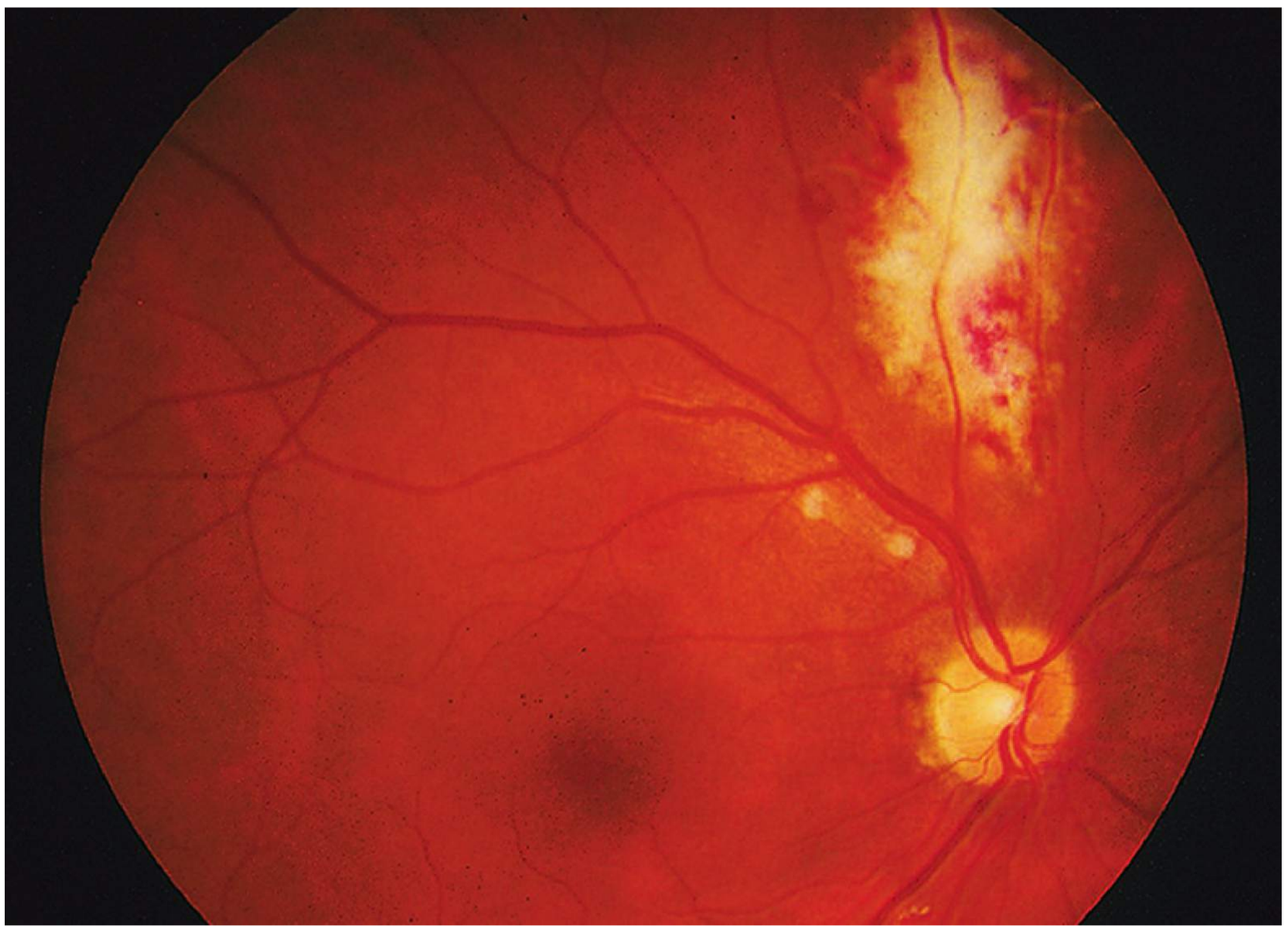


FIGURE 20.24 ■ **Cytomegalovirus Retinitis.** Funduscopic examination shows exudates and hemorrhages ("cottage cheese and ketchup" appearance) seen with CMV retinitis. (Photo contributor: Edward C. Oldfield III, MD.)

Clinical Summary

Varicella zoster virus (VZV) infection presents as shingles in HIV-infected patients at a greater frequency and severity than the general population. Despite the high incidence of shingles in this population, severe disseminated disease is rare.

The diagnosis of herpes zoster in the ED is often made clinically, by either recognizing the pain syndrome or visualizing the rash. Patients can present with a well-defined area of hyperesthesia for days prior to an outbreak of the characteristic lesions. Immunocompromised patients may demonstrate multidermatomal distribution or scattered vesicles at a distant site. Approximately 20% of patients will have systemic symptoms such as malaise, fever, headache, or fatigue. Culture, serologic testing, or PCR confirms the diagnosis. A Tzanck test can be obtained by scraping the base of a lesion in an attempt to demonstrate multinucleated giant cells.

Management and Disposition

Three antiviral drugs are used to treat herpes zoster infections: acyclovir, famciclovir, and valacyclovir. Patients with severe disseminated disease or ophthalmic zoster should receive intravenous acyclovir (10-12.5 mg/kg IV q 8 h for 10-14 days). Treatment of zoster ophthalmicus should include topical antibiotics and immediate ophthalmology consultation. Providing treatment of the disease within 72 hours of rash onset will result in a more rapid resolution of cutaneous lesions and decrease viral shedding, but will not change the incidence of postherpetic neuralgia. Immunocompromised patients should not be placed on glucocorticoids. Narcotics, capsaicin cream, and tricyclic antidepressants can be used for pain control.

Pearls

1. Worsening cases of herpes zoster, complicated herpes zoster, or ophthalmic zoster all require intravenous acyclovir and admission.
2. Herpes zoster encephalitis is rare, but can occur months after the cutaneous phase and can be difficult to diagnose. Common presenting symptoms include mental status changes, headache, fever, photophobia, vomiting, or even focal neurological deficits.
3. Patients presenting with localized hyperesthesia may have a zoster-related prodrome and treatment should be initiated if clinical suspicion is high.
4. The presence of vesicles on the tip of the nose (Hutchinson sign) indicates involvement of the nasociliary branch of cranial nerve V and is associated with a higher risk of ocular involvement.



FIGURE 20.25 ■ **HZV (Shingles).** A painful eruption of many tiny vesicles on an erythematous base in this patient with HIV. (Photo contributor: Jeffery Gibson, MD.)



FIGURE 20.26 ■ **HZV—Multiple Dermatomes.** Severe painful shingles (vesicles and bullae) spanning multiple cutaneous dermatomes in this HIV patient. (Photo contributor: John O'Mara, MD.)



FIGURE 20.27 ■ **Disseminated Herpes Zoster Infection.** Vesicles are seen over the entire face, representing disseminated HZV infection (multiple dermatomal distributions). (Photo contributor: Department of Dermatology, National Naval Medical Center, Bethesda, MD.)



FIGURE 20.28 ■ **Ophthalmic Herpes Zoster.** This patient has vesicles in the ophthalmic division of cranial nerve V bilaterally. (Photo contributor: Daniel L. Savitt, MD.)

EOSINOPHILIC FOLLICULITIS

Clinical Summary

Eosinophilic folliculitis, commonly seen in advanced HIV patients, is a pruritic skin eruption usually involving the face, neck, trunk, and extremities manifesting in the form of pustules and papules. The skin lesions usually start as small pustular groups that later coalesce to create irregular erosions and plaques with central hyperpigmentation. The associated pruritus is often so intense that the lesions become excoriated. Most patients with this condition will demonstrate CD4 cell counts of less than 250/μL. The etiology of eosinophilic folliculitis is unknown, but is commonly thought of as an inflammatory process associated with immune dysfunction. It has become less common with the advent of HAART.

Management and Disposition

The diagnosis requires dermatology consultation and referral for biopsy. Antihistamines, potent topical steroids, itraconazole, topical permethrin, retinoids, metronidazole, dapsone, and ultraviolet B phototherapy have all shown variable levels of efficacy.

Pearls

1. The severe pruritus associated with this condition helps distinguish it from bacterial folliculitis. Symptomatic treatment for pruritus is necessary, and the disease generally improves with effective antiretroviral therapy.
2. Dermatological referral is warranted prior to initiating definitive therapy.
3. Patients with eosinophilic folliculitis may present with prurigo nodularis and lichen simplex chronicus as a result of severe itching and rubbing.
4. Secondary syphilis should be considered in all HIV-infected patients presenting with rash.



FIGURE 20.29 ■ **Eosinophilic Folliculitis.** The rash of eosinophilic folliculitis consists of small groups of pustules and vesicles, as seen on the face of this patient. (Photo contributor: Department of Dermatology, National Naval Medical Center, Bethesda, MD.)



FIGURE 20.30 ■ **Eosinophilic Folliculitis.** A magnified view showing pustules at the base of each hair follicle. (Photo contributor: Department of Dermatology, National Naval Medical Center, Bethesda, MD.)

Clinical Summary

Infection with herpes simplex virus (HSV) is extremely common in HIV-infected patients and may present with oral, genital, anal, esophageal, or ophthalmologic involvement. The hallmark for most clinical presentations of HSV outbreaks is an inflammatory cutaneous eruption, with or without vesicles and pain. HSV esophagitis is seen in immunocompromised HIV-infected patients and presents as dysphagia and odynophagia with or without oral lesions. Idiopathic apthous ulcerations in HIV-infected patients are indistinguishable from HSV lesions. Perirectal lesions are often erythematous, ulcerative, and extremely tender, with a predilection for the gluteal cleft. Perirectal HSV may also be associated with proctitis and anal fissures.

Ocular HSV may be demonstrated by observing dendritic lesions after fluorescein staining. HSV is associated with a syndrome of acute retinal necrosis characterized by pain, keratitis, and iritis that may lead to retinal detachment. The diagnosis is usually made clinically but can be confirmed by a Tzanck test, biopsy, or culture.

Management and Disposition

Multiple oral treatment regimens are available to treat HSV. Duration of treatment is usually increased for immunocompromised patients. Ocular HSV requires prompt ophthalmology consultation.

Pearls

1. Anticipate involvement of multiple sites in HIV patients; presentations of HSV may be atypical compared to HSV in immunocompetent individuals.
2. Suspect HSV in any HIV patient with a chronic painful ulcerative lesion.
3. Perirectal HSV outbreaks may be missed on a superficial history and physical examination. Ask if it is painful to wipe after stool and examine the perianal area. Do not assume hemorrhoids in HIV-infected patients at risk for HSV.

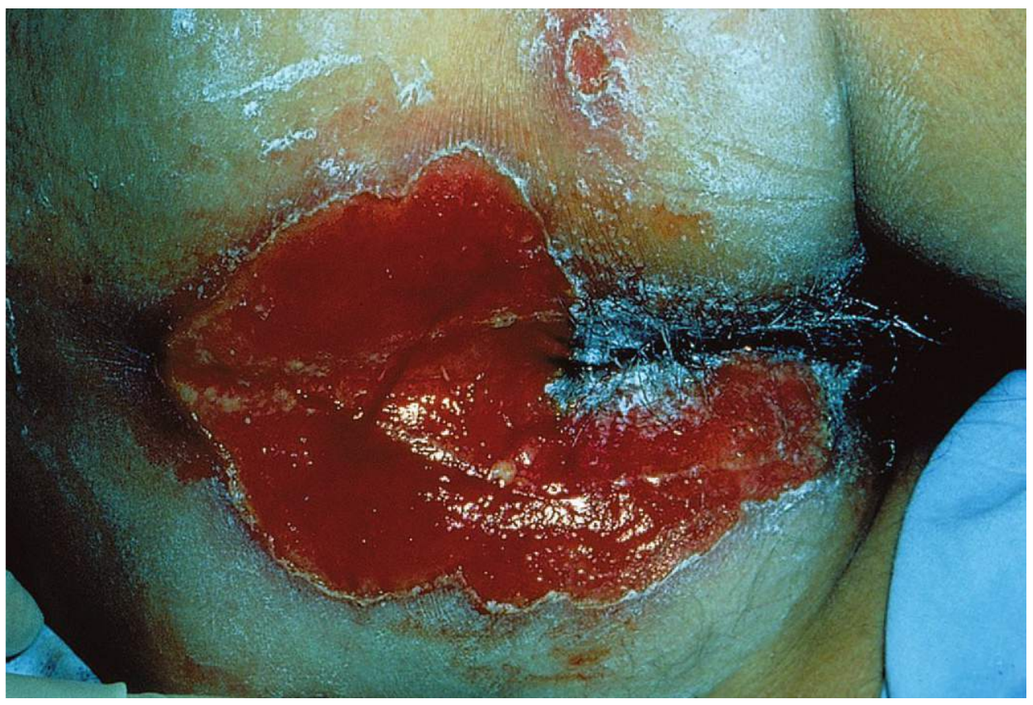


FIGURE 20.31 ■ **Herpes Simplex Virus in an HIV Patient.** Severe, recurrent perirectal HSV lesions in an HIV patient. (Photo contributor: Briana Hill, MD.)



FIGURE 20.32 ■ **Severe HSV on Genitalia.** Severe ulcerative HSV lesions on the penis and scrotum of an HIV patient. (Photo contributor: John O'Mara, MD.)

Clinical Summary

Human scabies, caused by the Sarcoptes scabiei mite, is one of the most common contagious dermatoses. In HIV patients, this organism can cause "crusted scabies," also known as Norwegian scabies, which denotes an overwhelming scabies infestation. In typical scabies, the mites cause extremely pruritic burrows, vesicles, and papules in a characteristic distribution involving the finger webs, sides of the hands and feet, breasts, waist, and groin. Pruritus is most intense at night. In contrast, crusted scabies typically affects the hands and the feet with asymptomatic crusting and does not cause significant pruritus. Transmission through infected linens or clothing is common in cases of Norwegian scabies. Risk factors include poor hygiene, crowding, and exposure to pets.

Most often the diagnosis of scabies is made clinically, with evidence of burrows and severe pruritus in a characteristic distribution. Definitive diagnosis is made from examination of shavings from the lesions. Placing mineral oil over a suspected lesion and then shaving it with a number 15 blade can demonstrate the mites, which are usually 0.3 to 0.4 mm in length.

Management and Disposition

Topical permethrin 5% cream has a low toxicity and is the treatment of choice for scabies. Patients are instructed to apply the cream from the neck down and leave it on for 8 to 14 hours before removal. One application is usually sufficient for treatment in nonimmunocompromised patients. The patient's clothes and bedding should be washed in hot water. Antihistamines should be prescribed to alleviate the pruritus. Often the pruritus persists after treatment as a hypersensitivity reaction to the mites and their feces and eggs. Any complicating secondary skin infection should be treated with appropriate systemic antibiotics. Localized infections can be treated with topical steroids.

For HIV patients with Norwegian scabies, permethrin should be applied to the face, scalp, behind the ears, and from the neck downward. Repeat treatments may be needed. For severe or refractory cases, oral ivermectin (200 mg/kg for one dose) can be tried with the exception of pregnant or lactating women.



FIGURE 20.33 ■ **Scabies.** Typical scabies rash showing unroofed papules secondary to scratching. Several small burrows are also seen. (Photo contributor: George Turiansky, MD.)



FIGURE 20.34 ■ **Norwegian Scabies.** Overwhelming scabies infestation causing "crusted scabies" in this patient with HIV. Hyperinfestations involve thousands to millions of mites. (Photo contributor: Francisco Bravo Puccio, MD, with permission from The Gorgas Course in Clinical Tropical Medicine, Universidad Peruana Cayetano Heredia, Lima, Peru.)

Pearls

1. Often secondary staphylococcal infections complicate the diagnosis, including impetigo, ecthyma, paronychia and furunculosis.
2. Oral antibiotics are often indicated for Norwegian scabies because of skin breakdown.
3. Close contacts of infected patients should be treated simultaneously. Inform patients that although the scabicide will kill the mites, itching may last for weeks. Patients often seek repeat treatments and inappropriately receive additional scabicides, which can cause contact dermatitis.



FIGURE 20.35 ■ **Norwegian Scabies.** Crusted lesions with hyperkeratotic scales on this patient's foot. Millions of mites were seen under microscopy in scrapings of the lesions. (Photo contributor: Larry Mellick, MD.)

MOLLUSCUM CONTAGIOSUM

Clinical Summary

Molluscum contagiosum is caused by a poxvirus that presents flesh-colored, dome-shaped lesions on affected individuals. Usually, molluscum contagiosum is an asymptomatic benign disease, but may become severe in HIV patients. Typical presentations consist of groups of 2 to 20 small, discrete, shiny lesions with central umbilication.

Management and Disposition

If the diagnosis is suspected in the ED, the patient should be reassured and referred to a dermatologist. Usually self-limited with spontaneous remission, treatment outside of genital lesions is for cosmetic reasons only.

Pearl

1. Clinically, it may be difficult to distinguish between cutaneous Cryptococcus and molluscum contagiosum. Dermatology or infectious disease consultation and biopsy may be required if the patient is clinically ill.

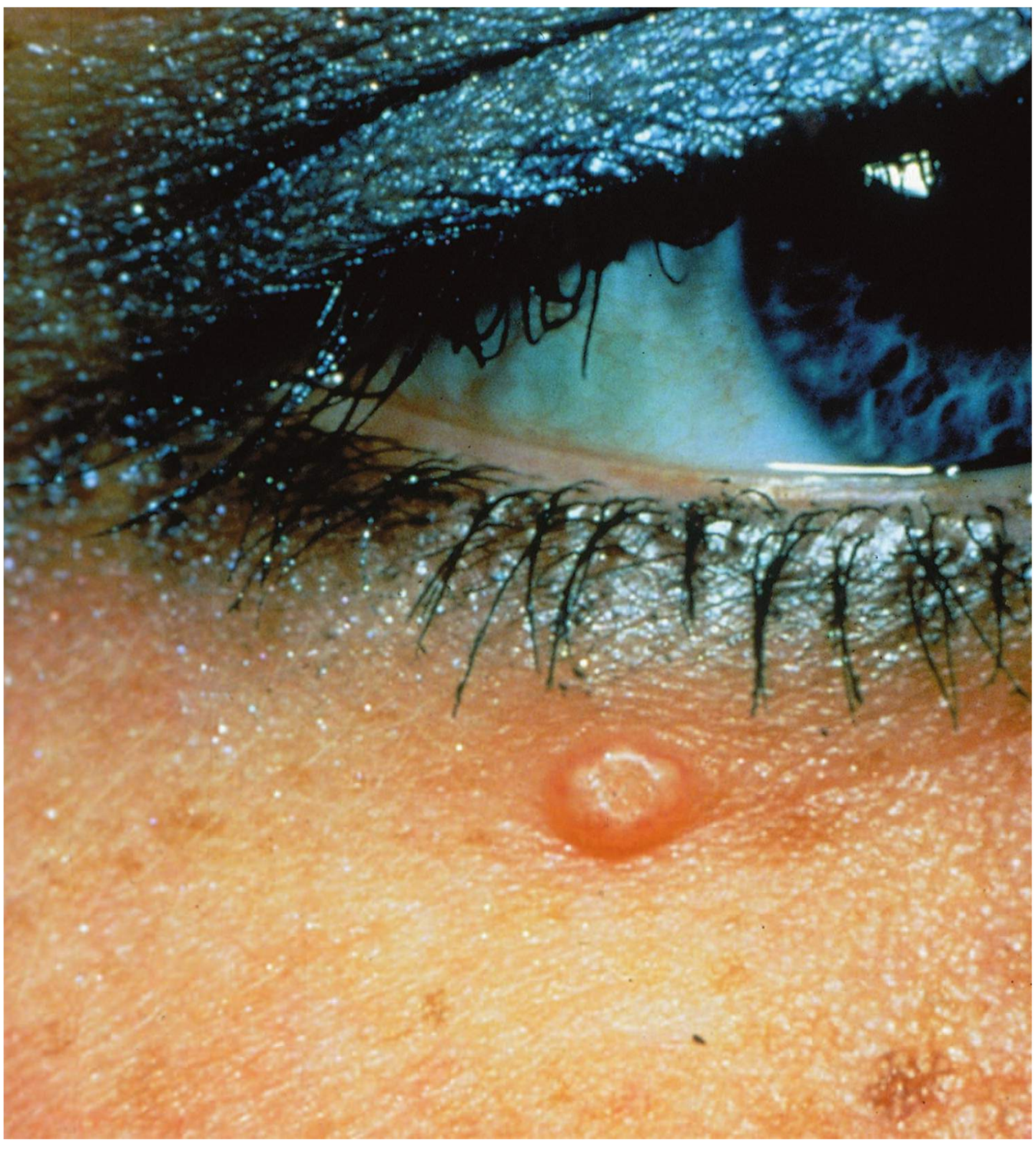


FIGURE 20.36 ■ **Molluscum Contagiosum.** Facial rash with ocular involvement is a common site of infection in HIV patients. Note the central umbilication. (Photo contributor: Department of Dermatology, National Naval Medical Center, Bethesda, MD.)

Clinical Summary

Dermatophyte infections are common superficial fungal infections involving the scalp (tinea capitis), body (tinea corporis), feet (tinea pedis), crural fold (tinea cruris), and nails (onychomycosis). Infection is very common in healthy individuals, but can be varied and atypical in patients with HIV. HIV patients also can demonstrate a much more severe and extensive form of disease. Three different dermatophytes are the usual source of infection: epidermophyton, trichophyton, and microsporum.

Tinea capitis is seen as an enlarging scaling patch on the scalp that may progress to a kerion. The initial lesion can be overlooked until alopecia is present. Untreated permanent scarring can occur. Tinea pedis is the most common dermatophyte infection seen in practice. Usually self-limited, intermittent, recurrent, and intensely pruritic, these lesions can often be secondarily infected due to mechanical irritation. Tinea corporis is the most common fungal infection seen in HIV patients. Usually, it begins as an intensely pruritic, oval scaling erythematous lesion that spreads centrifugally and can merge with other lesions. Tinea cruris is an infection involving the crural fold, much more common in men than women. It usually begins as a macular patch on the inner thigh, opposite the scrotal sac.

Management and Disposition

Tinea capitis responds to griseofulvin; however, other oral agents that can be effective include terbinafine, itraconazole, or fluconazole. Treatment usually is carried out for at least 3 weeks, with topical treatments ineffective. Tinea pedis can usually be treated with a topical antifungal cream for 4 weeks. Often patients have tried outpatient, over-the-counter antifungal medications without success. Extensive treatment can be initiated with griseofulvin, terbinafine, or itraconazole orally. Tinea corporis often responds to daily application of topical antifungal medications; however, systemic oral agents described above are also effective. Tinea cruris usually responds to topical antifungal medications.

Pearls

1. Confirming the diagnosis with a KOH preparation is important before initiating therapy, especially in diffuse disease. Often HIV patients suffer from a variety of skin infections that mimic dermatophyte lesions (eczema).
2. Athletes are especially at risk when close skin-to-skin contact occurs (wrestling, football). Oral treatment in these cases is preferred, with restriction in sports for 2 weeks or until lesions heal.
3. Some dermatophyte infections fluoresce under the Wood lamp examination.



FIGURE 20.37 ■ **Tinea Infection.** Tinea infection involving the scalp, neck, and upper back of this HIV-infected patient. (Photo contributor: Seth W. Wright, MD.)



FIGURE 20.38 ■ **Tinea Corporis.** Tinea infection involving the lower back and buttocks of the patient in Fig. 20.37. (Photo contributor: Seth W. Wright, MD.)

THROMBOCYTOPENIA

Clinical Summary

Thrombocytopenia occurs in 40% to 70% of all HIV patients. It can occur independently at all stages of HIV infection and may be encountered as the initial presentation of disease. HIV-associated anemia and granulocytopenia can occur concomitantly as course of the HIV infection worsens, with thrombocytopenia seen in 30% of patients with CD4 counts less than 200/μL. HIV patients with thrombocytopenia often present to the ED with bleeding (especially from the oral mucosa), ecchymosis, and petechiae. Secondary causes of thrombocytopenia are generally seen as the result of opportunistic infections, malignancy, or medications.

Management and Disposition

The emergency physician's efforts are initially focused on stabilization of the patient with two large intravenous lines, type and cross-match, and crystalloid infusion if significant bleeding has occurred. Because of the complexity of the differential diagnosis and potentially complicated treatment of HIV thrombocytopenia, an infectious disease specialist should be consulted early. In most cases, HIV patients with platelet count greater than 50,000 can be managed conservatively with spontaneous remission of approximately 20%. Zidovudine can increase platelet counts up to twofold in over 50% of patients. If the platelet count is less than 20,000, many infectious disease specialists recommend γ-globulin infusion and parenteral steroids. Other possible treatments include dapsone, danazol, interferon-α vincristine, anti-D immunoglobulin, splenic irradiation, and splenectomy. Even if the patient is to be managed conservatively, bone marrow analysis should be performed to rule out other causes of thrombocytopenia.

Pearls

1. Perform thorough skin and oral examinations in all patients with HIV looking for manifestations of thrombocytopenia.
2. Take a careful drug history and consider other infectious etiologies before assuming the thrombocytopenia is directly secondary to HIV infection.
3. Spontaneous bleeding is rare unless the platelet count is less than 10,000.



FIGURE 20.39 ■ **Thrombocytopenia.** Ecchymosis in an HIV patient with thrombocytopenia. (Photo contributor: Edward C. Oldfield III, MD.)



FIGURE 20.40 ■ **HIV-Associated Thrombocytopenia.** A large spontaneous subconjunctival hemorrhage is noted in this patient with HIV and severe thrombocytopenia. (Photo contributor: John O'Mara, MD.)

Clinical Summary

HIV patients have a 5 to 20 times higher rate of drug reactions than non-HIV patients. Up to 5% of ED visits by HIV patients are due to complications of therapy. Many of these manifest dermatologically, in order of decreasing frequency: (1) exanthems, (2) urticaria/angioedema, (3) fixed drug reactions, (4) erythema multiforme, and (5) photosensitivity reactions. The most common medications associated with rashes are antivirals, antibiotics, and antifungals.

Management and Disposition

The emergency physician may need to consult an infectious disease specialist, a pharmacist, or a dermatologist to help clarify the existence of a drug reaction. Clues besides recent initiation of a new drug are eosinophilia greater than 1000 or elevated liver function tests. Individual treatment varies depending on the situation. The offending agent should be discontinued. Antihistamines and steroids are indicated in certain situations.

Pearls

1. Ask about alternative and nonprescription medicines.
2. Half of all HIV patients will react adversely to sulfa drugs.
3. Beware of serious drug reactions such as Stevens-Johnson syndrome and toxic epidermal necrolysis (TEN).



FIGURE 20.42 ■ **Efavirenz Rash.** Drug exanthem typical of efavirenz. (Photo contributor: Stephen Burdette, MD.)



FIGURE 20.43 ■ **Efavirenz Rash.** Close-up image of drug exanthem caused by efavirenz in an HIV patient. (Photo contributor: Stephen Burdette, MD).



FIGURE 20.41 ■ **Drug Reaction.** Exanthematous drug reaction in an HIV patient. (Photo contributor: Kenneth Skahan, MD.)



FIGURE 20.44 ■ **Nevirapine Rash.** Drug exanthema caused by nevirapine in this HIV patient. (Photo contributor: Seth W. Wright, MD.)

ACUTE NECROTIZING ULCERATIVE GINGIVITIS

Clinical Summary

Acute necrotizing ulcerative gingivitis (ANUG), also known as Vincent angina or trench mouth, is commonly seen in HIV patients. It is a distinct and rapidly progressive ulceration typically starting at the tip of the interdental papilla, spreading along the gingival margins, eventually destroying the periodontal tissue. The triad associated with ANUG is oral pain, halitosis, and ulcerations. Other signs and symptoms include "metallic taste," "wooden teeth" sensation, tooth mobility, fever, adenopathy, and malnutrition. The cause of this aggressive, destructive process in HIV patients is a polymicrobial infection by oral anaerobes (Treponema, Selenomonas, Fusobacterium, Porphyromonas, Prevotella). ANUG represents a spectrum of disease from mild ulcerations to severe cellulitis and spread of the infection to the soft tissues, cheeks, lips, and bones.

Management and Disposition

ANUG is most frequently seen in four population groups: (1) HIV patients, (2) malnourished children, (3) young adults who are under a great deal of stress, and (4) polysubstance abusers. The first steps for the emergency physician are to eliminate other potentially more serious life-threatening infections and to address hydration status. Treatment includes (1) eliminating contributing factors (stress, poor nutrition, poor sleep, alcohol, and tobacco use), (2) chlorhexidine rinses twice a day, (3) surgical debridement by an oral surgeon if needed, and (4) oral penicillin and metronidazole.

Pearls

1. Beware of Ludwig angina (brawny submandibular induration and tongue elevation) associated with severe progression of ANUG as acute airway compromise is possible.
2. Noma (cancrum oris, gangrenous stomatitis), a rare disease of childhood associated with malnutrition, is characterized by an anaerobic destructive infectious process of the orofacial tissues that can clinically resemble ANUG.



FIGURE 20.45 ■ **Acute Necrotizing Ulcerative Gingivitis (ANUG).** ANUG (Vincent angina or "trench mouth") caused by spirochetal and fusiform bacteria in an HIV patient. Note the punched-out ulcerations of the interdental papillae, which are pathognomonic. (Photo contributor: Department of Dermatology, National Naval Medical Center, Bethesda, MD.)

Chapter 21

TROPICAL MEDICINE

Seth W. Wright
Andrew N. Pfeffer

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Uganda. (Photo contributor: Seth W. Wright, MD.)

The authors acknowledge Meg Jack for portions of this chapter written for the third edition.

FREE-LIVING AMEBA INFECTION

Clinical Summary

Free-living amebas, usually harmless protozoan residents of soil and water, can cause three distinct, occasionally devastating, human illnesses. Primary amebic meningoencephalitis (PAM) is a disease of the previously healthy and is caused by Naegleria fowleri. Granulomatous amebic encephalitis (GAE) is caused by Acanthamoeba species or Balamuthia mandrillaris, and occurs in both healthy and immunocompromised persons. In wealthier countries, contact lens users may suffer from chronic amebic keratitis, also caused by Acanthamoeba. While these diseases are found worldwide, they are more common in tropical and subtropical regions.

PAM is devastating, usually fatal, and found mostly in children or young adults with a history of recent freshwater exposure. The organism enters through the nose and penetrates the cribriform plate to the subarachnoid space and brain. The acute illness is indistinguishable from bacterial meningitis. Patients with GAE often present with an initial focus of infection in the skin or respiratory tract followed by neurologic changes reflective of extensive brain involvement.

Management and Disposition

The mainstay of management is consideration of these uncommon diseases. PAM is almost always fatal, but one survivor was successfully treated with amphotericin B, miconazole, and rifampin. Isolated cutaneous disease from Acanthamoeba and B mandrillaris can be cured, but brain involvement is fatal and often diagnosed at autopsy. Patients with suspected amebic keratitis should have immediate ophthalmologic referral.

Pearls

1. Lack of response to usual antimicrobials in a patient with severe meningitis symptoms should lead to the suspicion of PAM, particularly with recent freshwater exposure.
2. Global warming might increase the rate of N fowleri infection as the organism thrives in freshwater over 30°C.
3. Fatal cases of PAM have been reported following tap water nasal irrigation using a "neti pot" (a device used to clean nasal passages). Use of sterile or previously boiled water when irrigating eliminates the risk.
4. Consider Acanthamoeba infection in all contact lens wearers with a corneal infection. Early amebic keratitis can mimic the dendritic pattern of herpes simplex infection.
5. Immunocompromised patients with space-occupying lesions should have GAE on the differential.

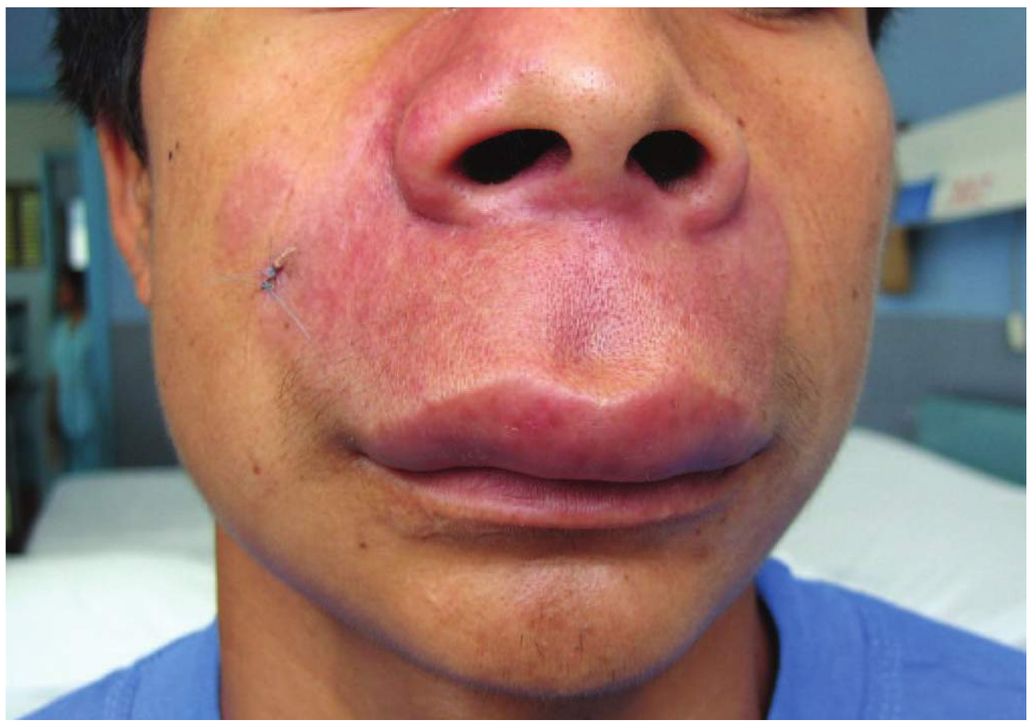


FIGURE 21.1 ■ **Ameba.** Twenty-one-year-old patient from South America with 1 year of symptoms from Balamuthia mandrillaris. The primary site often involves the mid-face and oral cavity. This patient did not have intracranial involvement. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

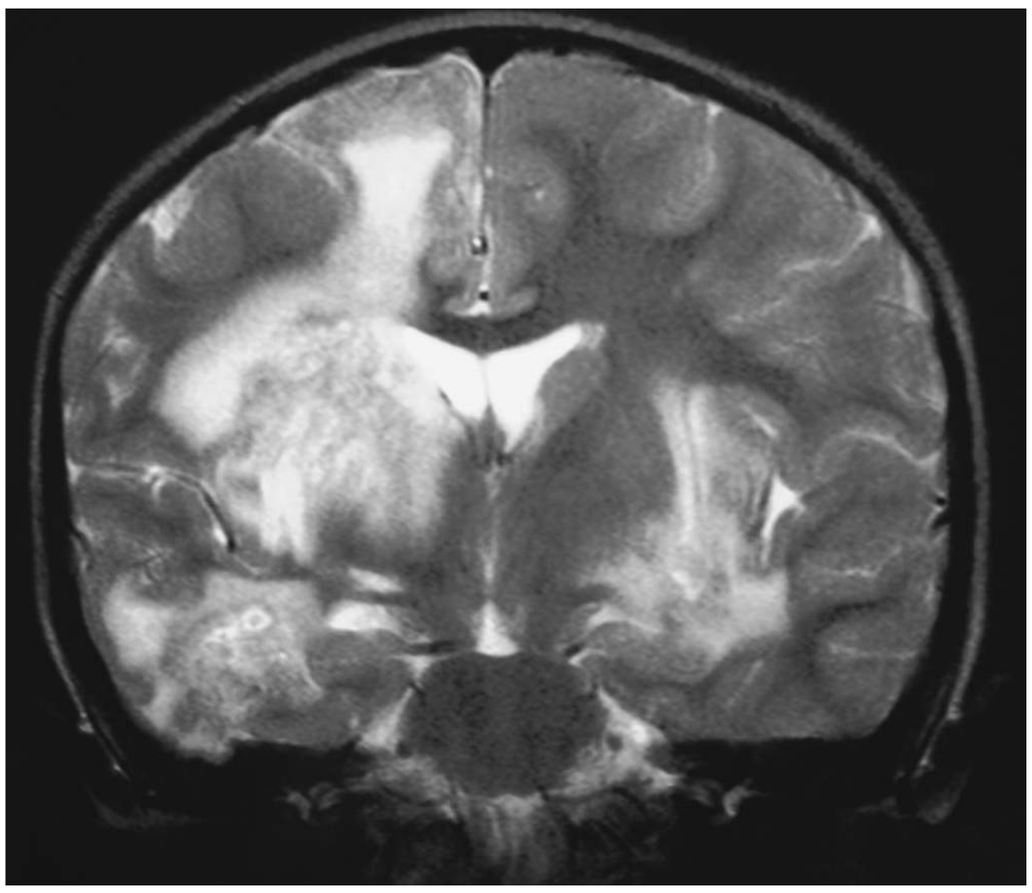


FIGURE 21.2 ■ **Ameba Imaging.** MRI showing extensive cerebral involvement in a fatal case of Balamuthia mandrillaris. (Photo contributors: Rob Greidanus, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

Clinical Summary

Anemia in the tropics is a common medical condition resulting from various nutritional deficiencies, infections, parasites, and inherited disorders. Its prevalence and causes differ significantly from developed countries, and is often multifactorial.

Overall, iron deficiency is the most common cause of anemia worldwide. Similar to deficiencies of folic acid and vitamin B_{12}, it results from diets rich in carbohydrates, but poor in meats, proteins, and vegetables. Most patients with AIDS in developing countries are anemic, with more severe cases seen in those coinfected with tuberculosis. Malaria causes hemolysis and hypersplenism, and should always be considered in anemic patients. Hookworm infection is another common cause of anemia, particularly in children and pregnant women. Other parasitic illnesses causing anemia include visceral leishmaniasis (kala-azar) and African trypanosomiasis (sleeping sickness). Hemoglobinopathies, including sickle cell anemia and thalassemia, are common in Africa, the Middle East, the Americas, and South Asia. Severe hemolytic anemia can occur in patients with G6PD deficiency.

Management and Disposition

Treatment depends on the suspected cause(s). Iron and folate supplementation can lead to a rapid and dramatic increase in red cell counts in patients with nutritional deficiencies. A low threshold for malaria diagnosis and treatment is important. Empiric treatment for hookworm is advisable. Red blood cell transfusion is rarely needed in stable patients, even those with extremely low counts.

Pearls

1. Administration of the antimalarial agent primaquine to patients with G6PD deficiency can lead to fatal hemolysis.
2. Thrombocytopenia in conjunction with anemia is highly suggestive of malaria in endemic areas.
3. Anemia in pregnancy should be screened for and aggressively treated as this improves both maternal and fetal outcomes.

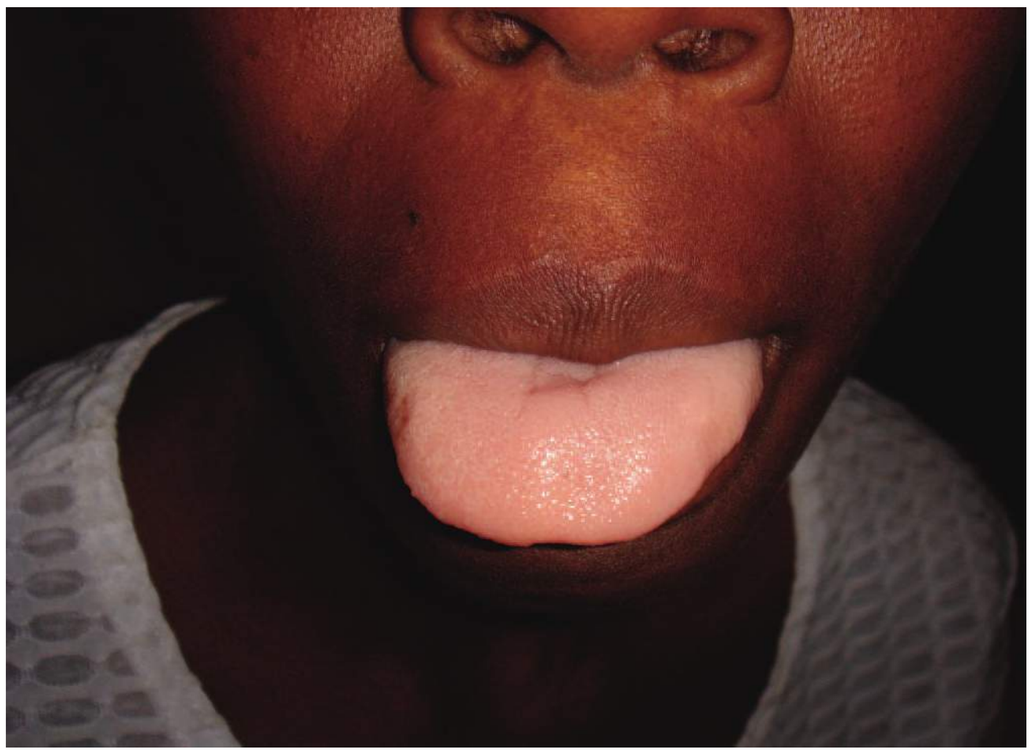


FIGURE 21.4 ■ **Anemia.** Pale tongue in a severely anemic Haitian woman. Pale mucus membranes, palms, and soles are often good indicators of anemia, particularly in dark-skinned individuals. (Photo contributor: Andreas Fischer, RN.)



FIGURE 21.3 ■ **Anemia.** Conjunctival pallor in a Haitian woman with complaints of weakness and a Hgb of 2.2. (Photo contributor: Andreas Fischer, RN.)



FIGURE 21.5 ■ **Anemia in AIDS.** Gums of Ugandan patient with end-stage AIDS and Hgb of 3.0. (Photo contributor: Seth W. Wright, MD.)

ANTHRAX

Clinical Summary

Anthrax, a worldwide zoonotic infection caused by Bacillus anthracis, was the first definitively recognized bacterial pathogen in human history. Most cases are sporadic, although occasional large outbreaks have been reported in Africa. Control of the disease in livestock has made endemic anthrax uncommon in developed countries, but it has taken on increased importance due to its use as a bioterrorism agent.

Cutaneous anthrax is the most common form, accounting for 95%. Infection occurs after spore exposure, with the initial manifestation of a small, painless, pruritic papule. The papule develops a large, central vesicle, followed by the classic necrotic ulcer with a black, depressed eschar. Local edema, regional lymphadenopathy, and systemic symptoms can occur. About 20% will progress to systemic bacteremia. Inhalational anthrax occurs after aerosolized spore exposure, and typically follows a biphasic course: (1) nonspecific upper respiratory infection–like symptoms and (2) then, in 1 to 4 days, fulminant bacteremia with fever, respiratory distress, hemorrhagic mediastinitis, and shock. Gastrointestinal (GI) anthrax is rare, affects the oropharynx or alimentary tract, and occurs after contaminated meat ingestion. It causes a nonspecific gastroenteritis, necrotic GI tract ulcers, and may progress to shock or death 2 to 5 days after symptom onset.

Management and Disposition

Uncomplicated endemic cutaneous anthrax is typically treated with oral or intramuscular penicillin. Doxycycline, erythromycin, and ciprofloxacin are alternatives. High-dose intravenous (IV) penicillin is used in cutaneous disease with systemic findings, and for inhalational or GI disease.

Patients with suspected bioterrorism-related anthrax require hospital admission with immediate public health notification. They should be decontaminated with removal (and sealed storage) of all clothing and cleansing with soap and water. The Centers for Disease Control and Prevention (CDC) guidelines recommend ciprofloxacin or doxycycline as first-line therapy. Inhalational and other severe forms are treated with additional multidrug antimicrobial therapy. Glucocorticoid therapy is controversial, but might be beneficial in meningitis or cutaneous disease with extensive head and neck edema.

Pearls

1. All forms can progress to hemorrhagic meningitis, which can mimic a traumatic tap on lumbar puncture.
2. Cutaneous and inhalational anthrax have been reported from use of imported animal hide drums.
3. Injection anthrax is rare and recently found among heroin users in Great Britain and Germany. Symptoms include abscess, cellulitis, necrotizing fasciitis, and sepsis.
4. The length of antibiotic therapy is usually 7 to 10 days, but postexposure prophylaxis involves 60 days, including vaccine administration.
5. The classic pathologic finding of inhalational anthrax is hemorrhagic mediastinitis, demonstrated as a widened mediastinum on chest radiography.



FIGURE 21.6 ■ **Anthrax.** A black eschar with a central hemorrhagic ulceration on the thumb associated with massive edema on the hand. (Used with permission from Wolff K, Johnson RA, Suurmond D. Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology. 5th ed. New York, NY: McGraw-Hill; 2005: p. 631.)







FIGURE 21.7 ■ **Anthrax Progression.** (**A**) Initial facial cutaneous anthrax with early lesion development and massive facial edema. (**B**) Interval development of septicemia. The lesion is developing a more typical dark coloration. (**C**) The patient following resolution of sepsis. (Photo contributor: Seth W. Wright, MD.)

ASCARIASIS

Clinical Summary

Ascaris lumbricoides is the most common human intestinal roundworm infection. The parasites are found worldwide and are highly endemic where sanitation and hygiene are poor; they commonly cause infection in regions where human feces are used as fertilizer. Adult worms live in the small intestine and produce enormous numbers of eggs, which are excreted in the feces. Passed eggs require at least several weeks in warm, moist soil before embryonating into an infective egg. Ingested eggs hatch in the jejunum, migrate through the intestinal wall into the bloodstream, and are transported to the lungs. Larval worms burrow through the alveolar walls, ascend up through the trachea, and are swallowed back into the small intestine where they develop into adults.

Most infections are asymptomatic. Patients may present for care if they pass a worm in their stool. A heavy worm burden may lead to abdominal pain, pancreatic/biliary disease, or intestinal obstruction. Failure to thrive and decreased cognitive development are seen in heavily infected children. Migrating worms may cause biliary obstruction, appendicitis, or liver abscesses.

Diagnosis is from identification of a passed worm or examination of stool for eggs.

Management and Disposition

Outpatient treatment with mebendazole or albendazole is usually effective but only on worms in the adult intestinal stage. Thus, it is recommended that a stool examination be done at 2 to 3 months and retreatment initiated if positive for eggs.

Pearls

1. Marked peripheral eosinophilia can be seen during the migratory stage.
2. While in the lungs, migrating worms can cause an eosinophilic pneumonitis with asthma-like symptoms, or "Löffler syndrome."
3. Ascaris worms occasionally migrate from the anus, mouth, or nose following antihelmintic treatment.
4. Ascaris is the most common cause of acute abdominal surgical emergencies in some highly endemic countries.



FIGURE 21.8 ■ **Ascariasis.** Worms spontaneously extruding from the anus of a boy with a high fever. Worms often migrate in patients with fever. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.9 ■ **Ascariasis.** Adult ascaris worm after passage from the anus. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.10 ■ **Ascariasis.** Distended abdomen of a Haitian girl with a large ascaris worm burden. (Photo contributor: Seth W. Wright, MD.)

Clinical Summary

American trypanosomiasis, or Chagas disease, is a zoonotic protozoal infection caused by Trypanosoma cruzi. Chagas is spread by triatomine insects with a large variety of mammals serving as natural reservoirs. Disease is limited to the western hemisphere and is most common in areas of substandard housing as the vector insects proliferate in adobe, unfinished brick walls, and in thatched roofs. Chagas is considered the most important protozoal disease in the Americas, surpassing malaria in morbidity and mortality. It is estimated that 10 million people are infected, including 300,000 immigrants within the United States.

Chagas is usually acquired during childhood and persists for life. Infection occurs when the insect defecates while taking a blood meal and the infectious stool is rubbed into the bite wound or a mucus membrane (often the conjunctiva). Acutely, infection is often asymptomatic but can present with a skin lesion at the infection site (chagoma), fever, adenopathy, myocarditis, and hepatosplenomegaly. The organism can be isolated during the indeterminate phase (years or even lifespan). Chronically, about 30% develop a cardiomyopathy with progressive biventricular heart failure. Severe arrhythmias, including complete heart block, are common. Approximately 10% of patients will develop digestive complications due to loss of lumen wall neurons, including megaesophagus and megacolon.

During acute infection, trypanosomes are seen on the blood smear and polymerase chain reaction (PCR) is useful for diagnosis, but serology is typically negative. Serological assays are used to diagnose chronic disease. Blood culture and xenodiagnosis can also be used, but lack sensitivity.

Management and Disposition

Patients presenting with acute symptoms are treated with benznidazole or nifurtimox. Patients with chronic disease should be worked up with an electrocardiogram (ECG) and echocardiography, and referred as indicated. Antitrypanosomal treatment is warranted in the indeterminate phase of chronic infection and in those with early stage cardiomyopathies.

Pearls

1. Chagas is the most common cause of cardiomyopathy in many Latin American countries. The disease should also be considered in patients with complete heart block or other arrhythmias.
2. Chagas is surprisingly common in wild and domestic mammals in the Southern and Southwest United States. The prevalence in Tennessee dogs is 6.4%, and is higher in wild animals such as raccoons, skunks, and rodents.
3. Human infection in the United States is rare since the vector insects are uncommon inside modern housing, but occasional cases have been reported in those without a history of travel.
4. Universal screening of blood for Chagas is now routine in the United States.



FIGURE 21.11 ■ **Chagas Disease.** A child with unilateral periorbital swelling from local inflammation at the site of a Chagas organism inoculation (Romana's sign). (Photo contributor: Jorge Kleisinger, MD, and Ana Rosa de Benedetto).

Clinical Summary

Cholera is a severe diarrheal disease caused by Vibrio cholerae, a gram-negative bacterium, and is spread via the fecal-oral route. It is associated with poor hygiene and overcrowding, as well as the resultant contamination of food and water. Endemic disease is present in many areas of the world with occasional epidemics. Massive outbreaks in previously disease-free areas can occur following disasters or breakdowns in public health measures, such as seen in Haiti following the 2010 earthquake.

Cholera is characterized by massive, watery, gray, and painless diarrhea. The stool resembles "rice water" without blood or pus. Patients may have associated vomiting. Renal failure, hypotension, and circulatory collapse can occur within hours of diarrhea onset. Mortality rates as high as 20% to 50% among severe cases can be seen if adequate rehydration is not available. Death rates of less than 2% are seen with good case management.



FIGURE 21.12 ■ **Cholera.** Severely dehydrated child during an outbreak in Uganda. Lethargy and sunken eyes are typical findings. IV fluids are typically reserved for severe cases. The child is on a typical cholera cot. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.13 ■ **Dehydration in Cholera.** "Tenting" of the skin of a dehydrated cholera patient. The presence of tenting implies moderate to severe dehydration. (Photo contributor: Seth W. Wright, MD.)

Management and Disposition

The mainstay of treatment is hydration. Oral rehydration solutions are adequate for mild and moderate dehydration. Patients with severe dehydration are treated with IV lactated Ringer solution and oral rehydration. Per World Health Organization (WHO) guidelines, adult patients may require 10 to 15 L in the first 24 hours. Monitoring of electrolytes and renal function is ideal, but often not available in areas struck by cholera epidemics. Hypokalemia is common. Antibiotics are not required for recovery as the illness is self-limiting, but doxycycline may reduce the volume of diarrhea and shorten the duration of illness.

Pearls

1. Cholera, plague, and yellow fever are the three diseases internationally notifiable to WHO.
2. John Snow's removal of the Broad Street water pump handle during the 1854 cholera epidemic in London is considered to be the beginning of modern field epidemiology.
3. Rapid cholera test kits are available and are essential for early confirmation of disease in the first suspected cases.
4. An oral cholera vaccine is available, but only confers short-term immunity and is not available in the United States.



FIGURE 21.14 ■ **Rice Water Stool.** Typical "rice water" stool from cholera patient. Patients with cholera often lose a liter or more of watery stool an hour. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.15 ■ **Cholera Cot.** Patient on cholera cot. Use of the cholera cot allows for monitoring of fluid output and allows for easier waste control in patients too weak to leave the bed. (Photo contributor: Seth W. Wright, MD.)

CUTANEOUS LARVA MIGRANS

Clinical Summary

Cutaneous larva migrans (CLM), also known as "creeping eruption," is the most common dermatologic problem following a trip to the tropics. CLM is a worldwide, parasitic infection commonly seen in warm, tropical environments; it is uncommon in developed countries due to shoe wearing habits and routine deworming of pets.

CLM is most commonly caused by dog and cat hookworms, Ancylostoma caninum and Ancylostoma braziliense; humans are an accidental host. The infected animal passes eggs in its feces where they hatch, molt, and feed on soil bacteria. Upon contact with a human host, the larval worm penetrates the skin and attempts dermal migration. The worm remains under the skin since it lacks collagenase and cannot penetrate deeper layers. The larvae are unable to complete their life cycle, and, if left untreated, are trapped in the epidermis and will die in 2 to 8 weeks.

CLM is commonly seen on the lower extremities of travelers who walk barefoot on beaches with contaminated sandy soil. Symptoms are manifested by an erythematous tract with a distinctive serpiginous dermal pattern. The tract is markedly pruritic and may feel like a thread on palpation.

Management and Disposition

Treatment of CLM consists of mebendazole, albendazole, ivermectin, or topical application of thiabendazole. Antipruritics may also help.

Pearls

1. The lesions advance a few millimeters to several centimeters daily as the larva migrates.
2. Excoriation and secondary infections frequently occur. This can make the distinctive wandering rash more difficult to visualize.
3. Biopsy is usually not helpful for diagnosis since the organism lies 1 to 2 cm away from the leading edge of the eruption.



FIGURE 21.16 ■ **Cutaneous Larva Migrans.** A serpiginous, linear, raise, tunnel-like erythematous lesion outlining the path of migration in the larva. Upon palpation, it feels like a thread within the superficial layers of the skin. (Photo contributor: Janet Rohde.)



FIGURE 21.17 ■ **Cutaneous Larva Migrans.** CLM with secondary infection on the foot of a toddler from Zambia. (Photo contributor: Meg Jack, MD.)

Clinical Summary

The larval form of the pork tapeworm Taenia solium causes cysticercosis and affects 50 million people worldwide. Disease in developed countries is usually due to immigration or foreign travel. Taeniasis (intestinal tapeworm) is acquired from ingesting cysts from undercooked pork. Eggs passed in the stool from these carriers are highly infectious and may survive in the environment for months. Humans acquire cysticercosis from the ingestion of these eggs/larva, usually through unhygienic food preparation. Once the eggs are consumed, they hatch, penetrate the bowel wall, and travel to the subcutaneous tissue, skeletal muscle, and brain, though they may involve any organ. Two distinct types of diseases exist: neurocysticercosis (which can be parenchymal or extraparenchymal) and extraneural cysticercosis. Symptoms are dependent on the affected organ system; significant morbidity is associated with ocular, cardiac, and neurologic involvement. The larval worm will eventually die and leave calcified lesions.

Intestinal taeniasis is diagnosed through egg identification on stool exam or by passage of an intact worm or worm segment. Computed tomography (CT) and magnetic resonance imaging (MRI) have facilitated recognition of neurocysticercosis with visualization of a contrast-enhancing ring lesion. Diagnosis may also be made by biopsy, serum, or cerebrospinal fluid (CSF) antibody testing. Neurocysticercosis should be strongly considered in endemic regions or in immigrants with new onset seizures.

Management and Disposition

Seizures are treated with standard medications. Calcified lesions do not require specific anticysticercal therapy. Viable cysts can be treated with albendazole or praziquantel, but should be done with expert consultation. Inpatient management is advisable if viable cysts are to be treated as inflammatory reactions may occur. Corticosteroids are recommended in this situation. Surgical intervention may be indicated for obstructing neurologic lesions or intraocular lesions.

Pearls

1. Infection is most common in rural areas of developing countries where pigs are allowed to roam freely and ingest infected human feces. This completes the cycle and propagates infection.
2. Cysticercosis and taeniasis are rare in predominantly Muslim countries where eating pork is uncommon; however, cysticercosis is still possible due to ingestion of contaminated nonpork food products.

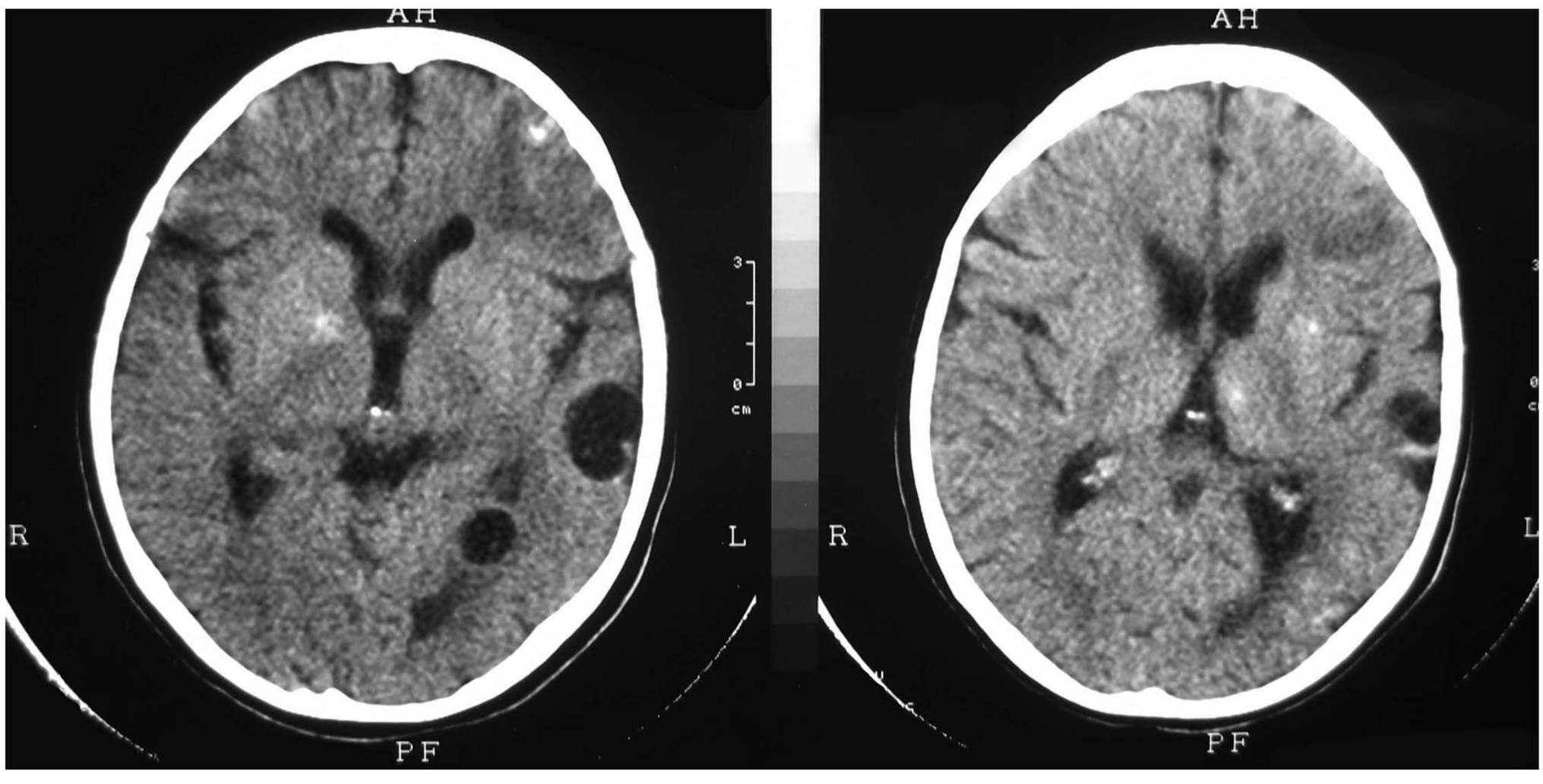


FIGURE 21.18 ■ **Neurocysticercosis.** Noncontrast head CT showing new (cystic) and old (calcified) lesions of neurocysticercosis. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

3. The adult tapeworm can attain a length of 20 feet or more, and can live up to 20 years in the intestine.
4. Most people with an intestinal pork tapeworm do not have cysticercosis.
5. The only pathognomonic CT or MRI finding of neurocysticercosis is a 2- to 4-mm bright nodule within the cyst ("cyst with dot sign") that represents a solid larval tapeworm.

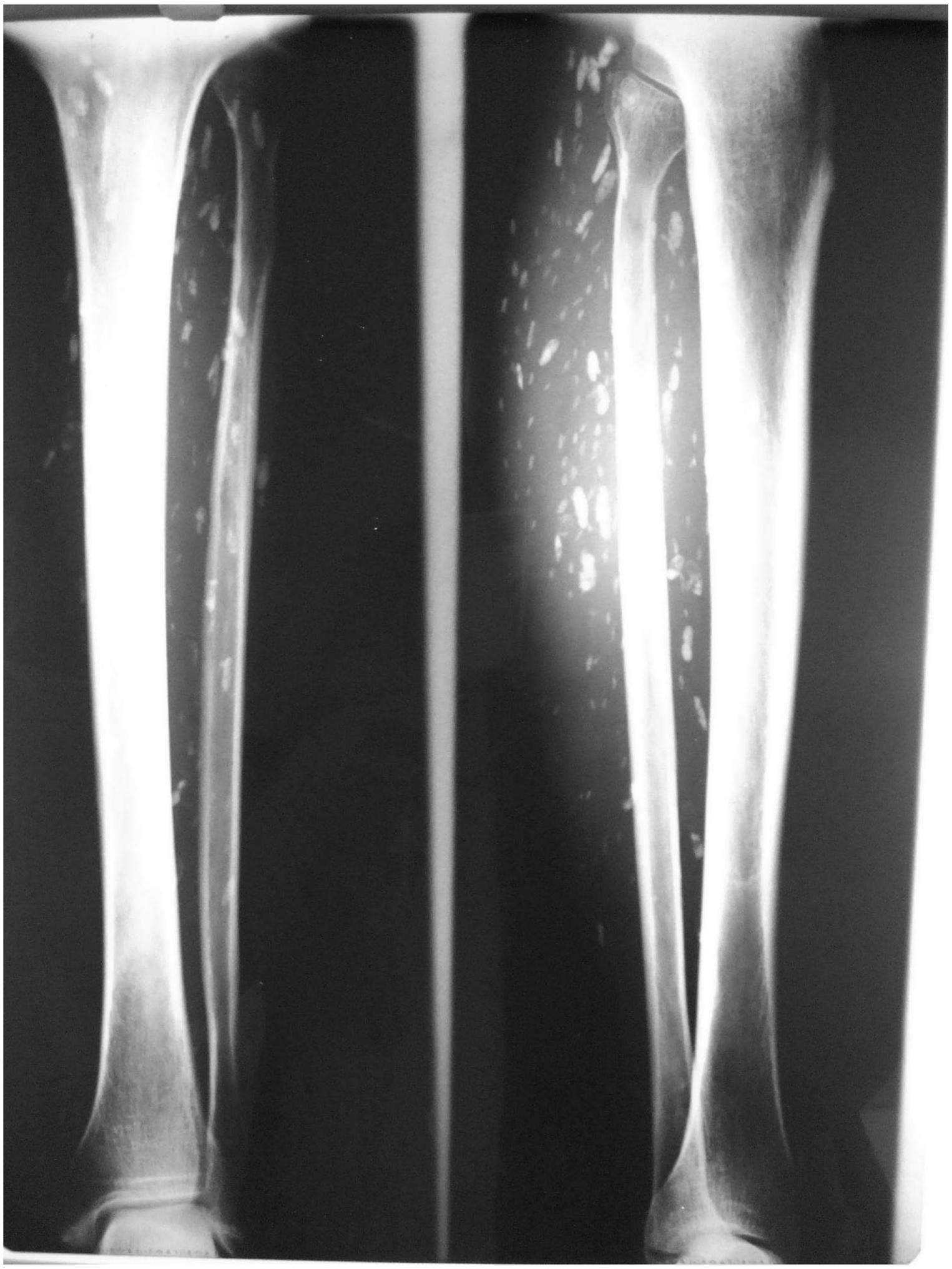


FIGURE 21.19 ■ **Cysticercosis Cysts.** Multiple calcified soft tissue cysts noted as an incidental finding on a lower leg radiograph in a patient with disseminated cysticercosis. The "cigar-shaped" calcifications are highly suggestive of cysticercosis. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

Clinical Summary

Dengue fever, also known as "breakbone fever," is a mosquito-borne viral disease. A flavivirus, dengue is a rapidly emerging illness due to reintroduction of Aedes species mosquitoes into areas of previous eradication. It is now distributed throughout tropical and subtropical regions, and widespread epidemics have occurred in the Caribbean and Southeast Asia, among other world regions. Dengue is one of the most common causes of fever in returning travelers.

The incubation period is usually 3 to 7 days, with symptom resolution within 10 days. Infection ranges from subclinical symptoms to a hemorrhagic state with possible shock. Most commonly, it is characterized by fever, severe myalgias, retro-orbital pain, and headache. A rash, seen in approximately half, is usually maculopapular, but may be mottled, flushed, or petechial. The recently revised classification of dengue severity consists of dengue without warning signs, dengue with warning signs, and severe dengue based upon the degree of plasma leakage, spontaneous bleeding, organ impairment, and shock.

There are four serotypes; exposure to one serotype provides lifelong immunity to that type. Previous infection with one serotype may predispose an individual to a more severe infection with another serotype. Thus, severe dengue is more common in indigenous individuals than the previously unexposed traveler. The diagnosis is primarily clinical. The presence of thrombocytopenia, leukopenia, and hemoconcentration is suggestive, and confirmatory tests with PCR or immunoglobulin M antibodies via enzyme-linked immunosorbent assay (ELISA) are available.

Management and Disposition

Supportive therapy is the only treatment available, with a focus on fluid repletion. Patients with mild illness are treated as outpatients. Admission of those with severe illness, hemorrhagic manifestations, shock, or an uncertain diagnosis is warranted.

Pearls

1. Consider dengue fever in recently returned travelers with fever, headache, and myalgias, particularly travelers from the Caribbean or Southeast Asia.
2. The fever tends to be high and may suddenly resolve and then return, also known as a "saddle back" fever pattern.

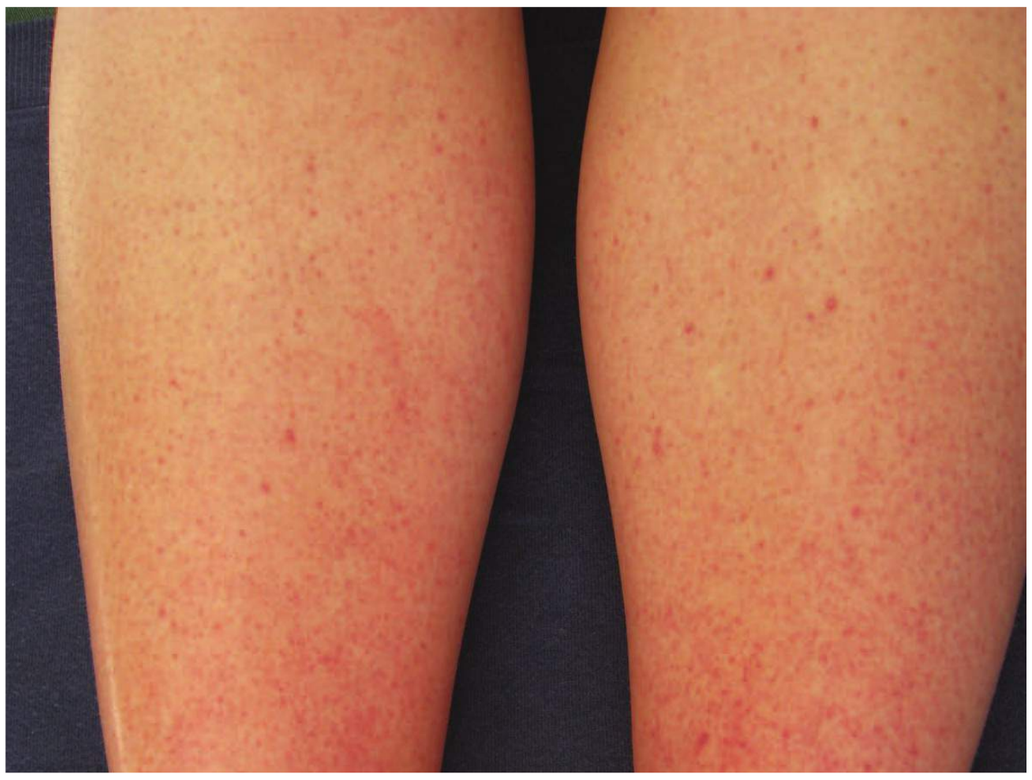


FIGURE 21.20 ■ **Dengue Rash.** Maculopapular rash from dengue on legs of an American medical worker during an outbreak in Guyana. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.21 ■ **Dengue Petechiae.** Measurement of petechiae following a tourniquet test in a patient with dengue. Twenty or more petechiae in the template area are suggestive of dengue diagnosis. (Photo contributor: WHO/TDR.)

3. A positive "tourniquet test" is suggestive of dengue. A blood pressure cuff is applied and inflated to a point between the systolic and diastolic pressures for 5 minutes. The test is positive if there are more than 20 petechiae per square inch.
4. Occasional cases of dengue occur in South Texas and an outbreak recently occurred in Key West, Florida.



FIGURE 21.22 ■ **Dengue Rash.** A hemorrhagic bullae on the arm of a patient with dengue hemorrhagic fever. (Photo contributor: WHO/TDR.)

Clinical Summary

Dracunculiasis is a debilitating roundworm infection caused by Dracunculus medinensis, commonly known as the Guinea worm. Dracunculiasis was historically widespread throughout equatorial Africa, the Middle East, and South Asia but the disease is now isolated to limited areas of Africa due to intensive eradication efforts. Dracunculiasis has a complex life cycle beginning when infected water fleas, or cyclops, are ingested while drinking from unclean water sources. Following ingestion, the larval worm penetrates through the stomach and intestine and migrates to the subcutaneous tissue, where it matures and reproduces. A painful wound develops, usually on the lower extremity, when the female emerges to release larvae. Immersion in water relieves the pain, allowing the worm to release larvae and complete the life cycle.

Emergence of the worm leads to a painful wound that is often accompanied by fever, nausea, vomiting, edema, intense pruritus, ulceration, and eosinophilia, all of which can last for months. The wounds often become secondarily infected, leading to significant disability. Drinking boiled or filtered water prevents dracunculiasis.

Management and Disposition

The worm is carefully extracted, usually around a stick or rolled gauze, in order to avoid retained worm products and the resultant inflammatory reaction. This process can take days to weeks. Daily immersion in water, with proper disposal to avoid larval spread, helps facilitate worm removal. Surgical excision is possible.

Pearls

1. The Guinea worm is the largest tissue parasite of humans. While very thin (2 mm in diameter), a full-grown worm is about a meter in length.
2. Dracunculiasis is rarely fatal, but the wound can cause significant morbidity and economic loss in areas with high infection rates.
3. It is predicted that dracunculiasis will be the first parasitic disease to be fully eradicated, largely through patient education and use of filtered water.



FIGURE 21.23 ■ **Dracunculiasis.** Gradual extraction of a Guinea worm using rolled gauze in Ghana. (Photo contributor: The Carter Center/Louise Gubb.)

ELEPHANTIASIS

Clinical Summary

Elephantiasis affects more than 120 million people worldwide with over 40 million severely disfigured. It is not a specific disease, but rather a syndrome caused by chronic obstruction of lymphatics. The most common cause is lymphatic filariasis, which is caused by the thread-like worms Wuchereria bancrofti, Brugia malayi, and Brugia timori. The infection is transmitted to humans by mosquitoes. Adult worms lodge in the lymphatics, disrupting the fluid balance between tissues and blood vessels, causing lymphedema of the extremities, breast, or genitourinary (GU) system. The infection is generally acquired in childhood, although clinical manifestations may take years to develop. Adult worms live 4 to 6 years, producing millions of blood circulating microfilariae. Acute symptoms of lymphadenopathy and dermal inflammation may precede and later accompany chronic swelling. With persistent infection and inflammation, the skin develops a hyperkeratotic, pebbly, or warty appearance that may become ulcerated and darkened. Bacterial and fungal superinfections contribute to morbidity.

Mosquito nets and insect repellants are the main means of prevention of lymphatic filariasis. Parasites may be detected microscopically in the blood, but the nocturnal periodicity makes identification challenging. Eosinophilia is common, but is nonspecific. Availability of a rapid card test that identifies circulating antigens has overcome this problem and is available in some endemic areas.

Management and Disposition

Treatment of lymphatic filariasis depends upon the presence or absence of other filarial organisms and includes various combinations of albendazole, ivermectin, and diethylcarbamazine. Cleansing of the affected areas and topical antibiotics aid in thwarting secondary disease. Local massage and elevation of the extremity improve lymphatic flow.

Pearls

1. Elephantiasis bears a heavy social burden due to physical limitations, disfigurement, sexual disability, and social stigmatization. Affected patients may be shunned by their families, are often unwed, and unable to work.



FIGURE 21.24 ■ **Elephantiasis.** Unilateral lymphatic filariasis. Note diffuse edema with early evidence of chronic skin changes. (Photo contributor: Larry Stack, MD.)



FIGURE 21.25 ■ **Elephantiasis.** Late-stage bilateral elephantiasis with chronic nodular skin changes. (Photo contributor: Seth W. Wright, MD.)

2. Testicular hydrocele is the most common manifestation of chronic W bancrofti infection for males in endemic areas.
3. Symptomatic filariasis is occasionally seen in travelers, even in those with only short-term stays in endemic regions.
4. W bancrofti occasionally causes an acute asthma-like condition known as tropical pulmonary eosinophilia.
5. Podoconiosis is a noninfectious cause of elephantiasis that occurs in people who walk barefoot in areas with large amounts of volcanic ash. Kaposi sarcoma is another emerging cause of nonfilarial elephantiasis in sub-Saharan Africa.



FIGURE 21.27 ■ **Elephantiasis.** Nonfilarial elephantiasis due to extensive Kaposi sarcoma in a 26-year-old Ugandan woman with AIDS. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.26 ■ **Elephantiasis.** Large hydrocele from lymphatic filariasis in a man from Haiti. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.28 ■ **Elephantiasis.** Bilateral disfigurement from lymphatic filariasis in an elderly Haitian man. (Photo contributor: WHO/TDR.)

EPIDEMIC MENINGITIS

Clinical Summary

Meningococcal disease in developed countries usually consists of sporadic cases and small outbreaks. In contrast, massive epidemics of serogroup A or C meningococcal meningitis occur in tropical countries, most notably in sub-Saharan Africa. These seasonal outbreaks tend to occur during the dry season along a wide swath of equatorial Africa known as the "meningitis belt."

The clinical presentation of epidemic meningitis is the same as in developed countries. Patients will typically present with fever, headache, nausea/vomiting, photophobia, and neck stiffness. Coma and death typically ensue if not treated. Diagnosis is with a spinal tap, although patients in resource-limited countries are often treated based on clinical grounds during a known outbreak. Mortality rates of less than 10% are obtainable. Large-scale vaccination programs are effective in decreasing spread of the disease within the affected areas and in adjacent population centers.

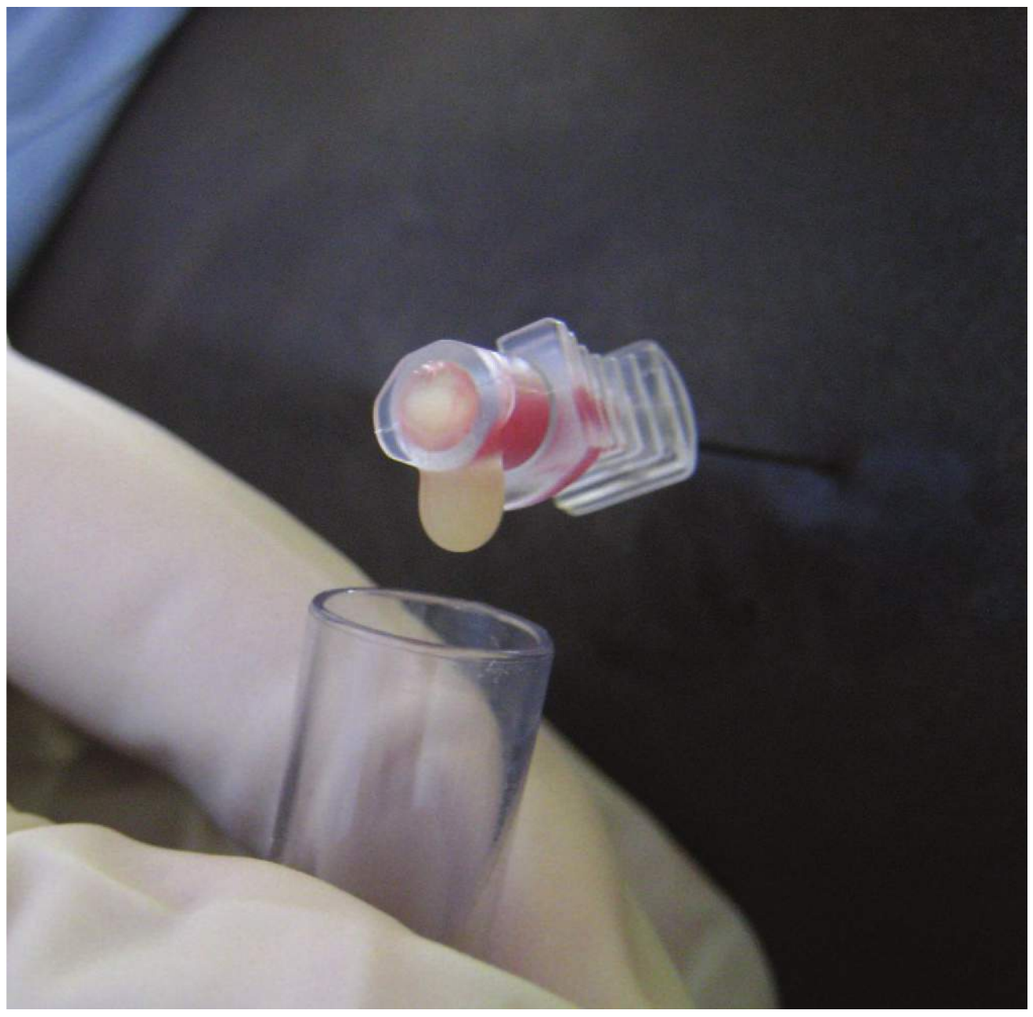


FIGURE 21.29 ■ **Meningitis Spinal Fluid.** CSF obtained from a 33-year-old Ugandan woman treated during an epidemic of serogroup A meningococcal meningitis. (Photo contributor: Seth W. Wright, MD.)

Management and Disposition

The mainstays of treatment are antibiotics and supportive care. Penicillins, cephalosporins, and ampicillin are still the treatments of choice in many settings, and are usually efficacious.

Pearls

1. Single-dose oily chloramphenicol injections have been used with success and allowed for easier management of large numbers in limited-resource settings. A second dose is given in 24 hours if there has been no improvement.
2. Massive outbreaks of meningitis have occurred during the Hajj, a pilgrimage to Mecca in Saudi Arabia. They are the only country requiring proof of vaccination before entry.
3. An often-used critical threshold to define an epidemic in Africa is 15 cases per 100,000 population per week. This would be equal to over 1400 cases per week in the Chicago metropolitan area.
4. Both serogroup A and C are covered by the meningococcal vaccine recommended for travelers by WHO and CDC.



FIGURE 21.30 ■ **Meningitis in a Child with a Very Rigid Neck.** Cerebral malaria should also be in the initial differential diagnosis. (Photo contributor: Richard P. Usatine, MD. Used with permission. From Usatine RP, Smith MA, Mayeaux EJ, Chumley HS. The Color Atlas of Family Medicine. 2nd ed. McGraw-Hill; 2013: Fig. 7-14.)

Clinical Summary

Endemic goiter is one of a spectrum of iodine deficiency disorders and is characterized by thyroid gland enlargement. It can occur in any location where environmental iodine is limited, but is rarely seen in developed countries as dietary iodine supplementation is routine. Goiters are often seen in inland and mountainous areas where iodine is leached from the soil, and access to iodine-rich foods (i.e., fish) is limited. Iodine deficiency disorders are common in widespread areas of Africa, Asia, and South America.

Patients with inadequate iodine in their diet have decreased T3 and T4 production, and resultant increased pituitary thyroid-stimulating hormone (TSH) secretion. The thyroid becomes hyperplastic and enlarged; however, patients are typically euthyroid.

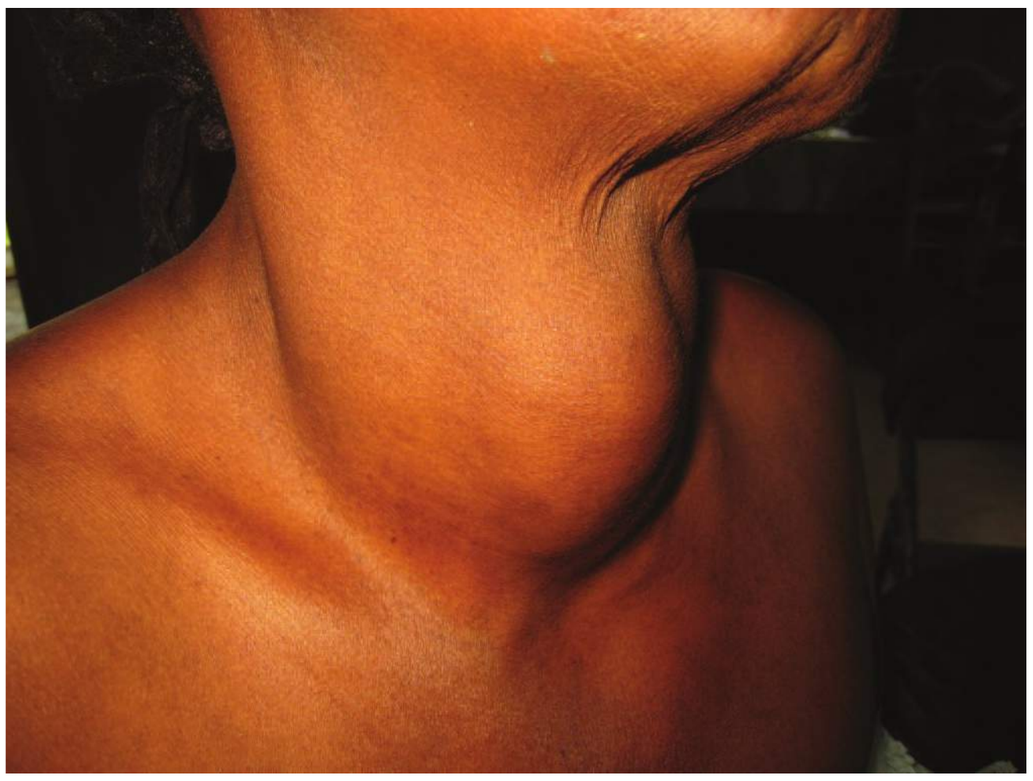


FIGURE 21.31 ■ **Goiter.** Easily visualized and palpable goiter. (Photo contributor: Seth W. Wright, MD.)

Management and Disposition

Prevention with dietary supplemental iodine is the mainstay of goiter management at the community level. Individual patients with goiter can be treated with potassium iodide solution or Lugol iodine, with iodized salt as a permanent solution. Surgical treatment can be indicated for massive goiter, particularly in the setting of tracheal or esophageal compression.

Pearls

1. Goiters in developed countries are most likely to be caused by defects in thyroid hormone production, resulting in an increase in TSH.
2. Iodine deficiency is the most common cause of preventable mental retardation (endemic cretinism) in the world.
3. Some dietary staple items, such as cabbage, cassava, lima beans, and sweet potatoes, have a goitrogenic factor which may be superimposed upon primary iodine deficiency.
4. Malignancy is not a common complication despite the massive size of many goiters, although it may interfere with detection.
5. In children, the goiter is usually diffuse; in adults, however, it is more commonly nodular since individual thyroid nodules proliferate at different rates.

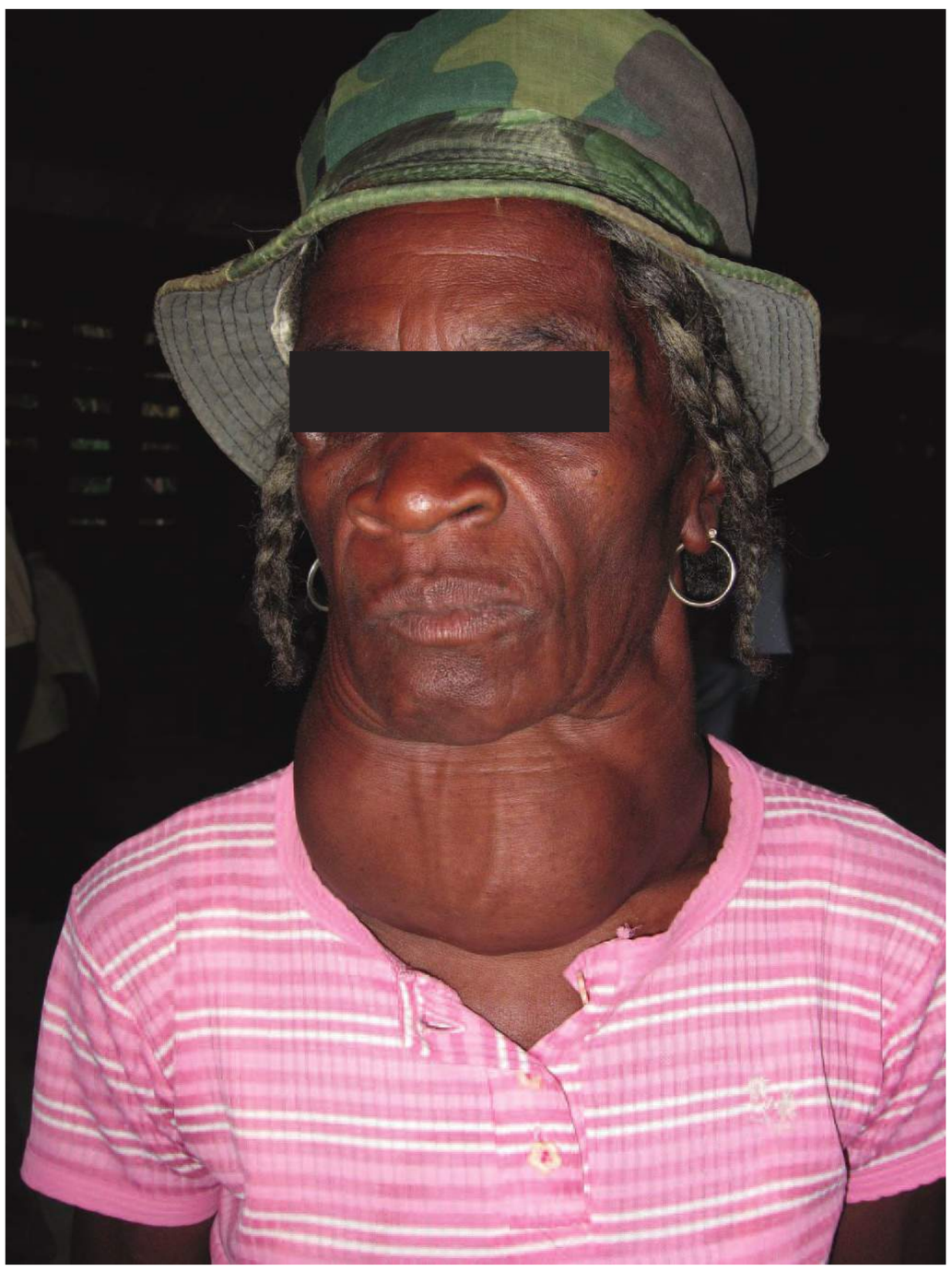


FIGURE 21.32 ■ **Goiter.** Patient from mountainous inland region of Haiti with massive goiter. This island nation has a high rate of goiter due to the complex interaction of poverty, political instability, deforestation, and erosion of soil. (Photo contributor: Ian D. Jones, MD.)

HYDATID CYST

Clinical Summary

Hydatid cysts are formed by the larval stage of Echinococcus granulosus tapeworms. The life cycle is maintained between canines and various farm animals (usually sheep or cattle). Adult worms inhabit the canine small intestine and eggs are passed in the stool. Farm animals become infected when they feed upon stool-contaminated material. The life cycle is completed when dogs or other canines ingest larval cysts formed in infected farm animals. Humans are infected from inadvertent ingestion of canine fecal material and are considered accidental hosts.

Hydatid cysts most commonly affect the liver and the lungs. Most patients who are infected have no symptoms until the cysts rupture, or are large enough to produce mass effect. Diagnosis is centered upon ultrasonography and serologic testing. Changes in farming practices have led to a marked decline in most industrialized countries, but they remain common in many developing areas. Most cases in the United States are immigrants from endemic regions.

Management and Disposition

Hydatid cysts are often an incidental finding when x-rays, CT scans, or ultrasounds are done for other purposes. These patients can be referred for follow-up. Patients with symptomatic cysts may need admission. Depending on cyst size and location, treatment may include percutaneous aspiration, surgical resection, or pharmacotherapy. Care must be taken with surgery to avoid spillage of cysts, which may lead to anaphylaxis.

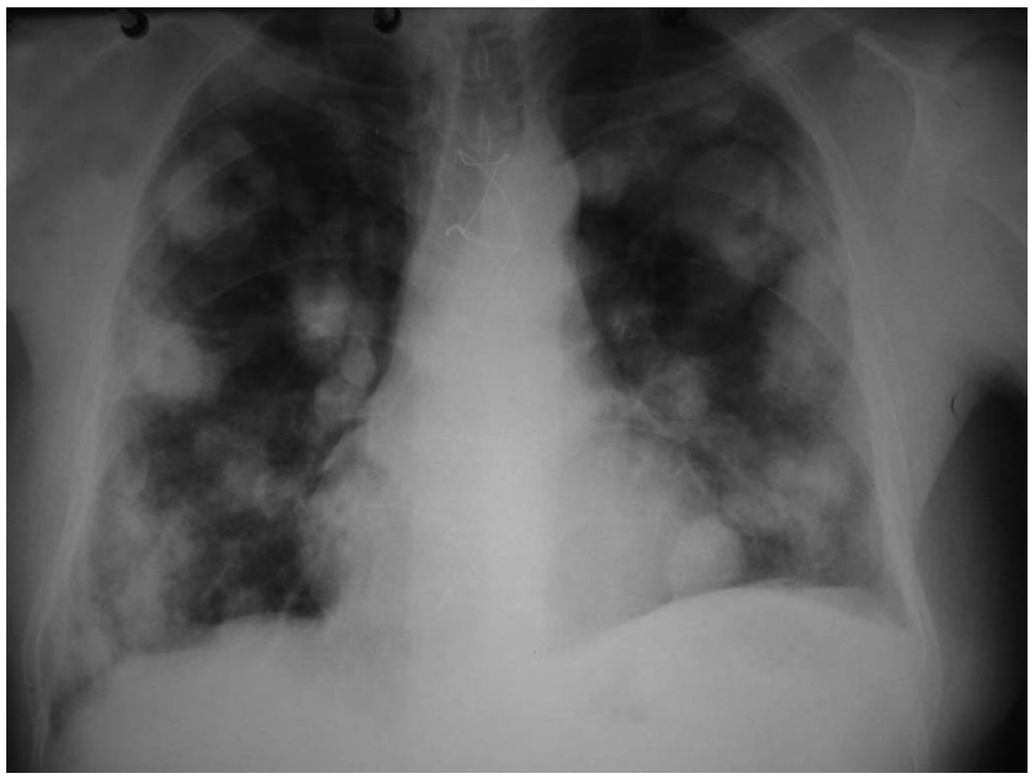


FIGURE 21.33 ■ **Hydatid Cyst Radiograph.** Chest x-ray showing multiple hydatid cysts. (Photo contributors: Rob Greidanus, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

Pearls

1. Liver cysts may produce obstructive jaundice, abdominal pain, or cholangitis.
2. Cysts are slow growing with an estimated average growth of 1 to 1.5 cm per year.
3. A chronic cough, pleuritic chest pain, hemoptysis, and dyspnea may be seen with lung involvement.
4. Though cerebral involvement is uncommon, it is seen more often in children.



FIGURE 21.34 ■ **Hydatid Cyst CT.** Scan demonstrating large hydatid cyst in the liver. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

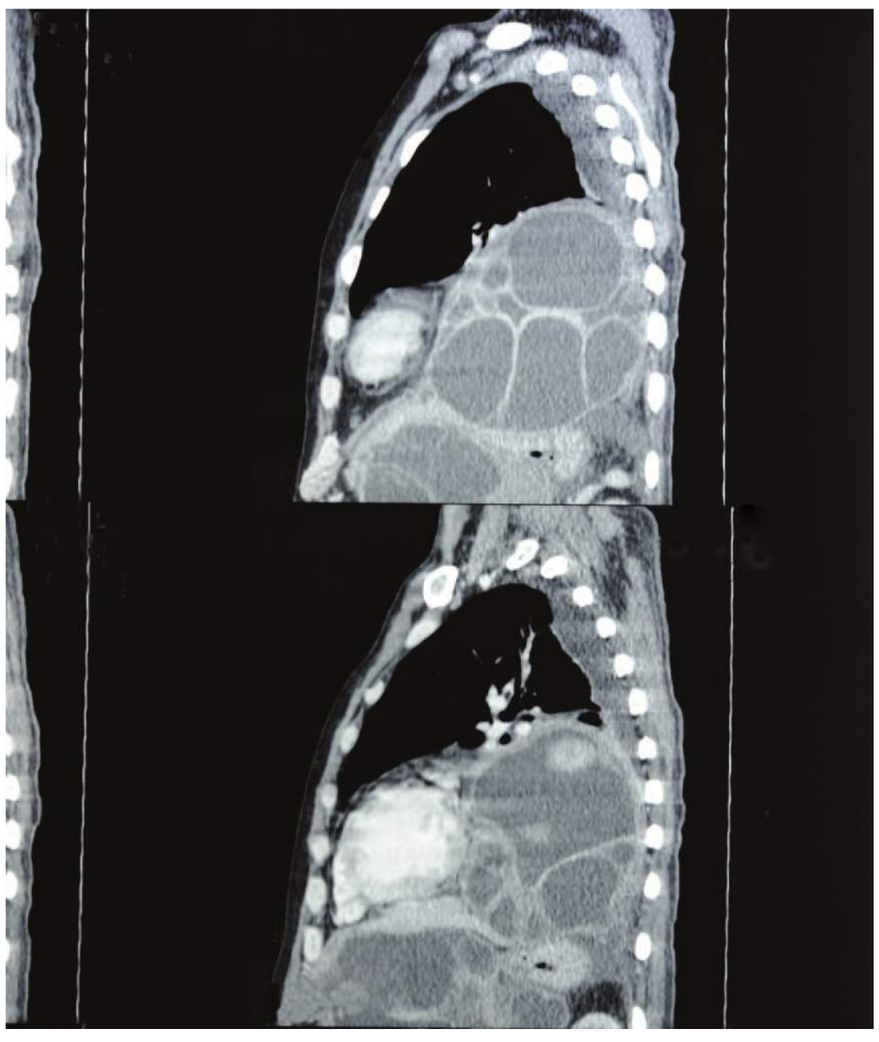


FIGURE 21.35 ■ **Hydatid Cyst CT.** Multiple echinococcal cysts are seen in the chest and liver of this middle-aged Latin American man with chronic cough, chest pain, and hemoptysis. (Photo contributor: Larry Stack, MD.)

Clinical Summary

Leeches are parasitic blood sucking aquatic or land-based invertebrates. Aquatic leeches are found worldwide, have relatively weak jaws, and usually attach to thinner tissues such as the pharynx or vagina. Land leeches are primarily found in tropical and subtropical areas of Asia, the South Pacific, and South America, typically have powerful jaws, can attach to any body surface, are fast and painless feeders, and often detach from the host without being noticed.

Leeches have been historically used for bloodletting, their saliva utilized to derive modern anticoagulants, and are considered currently for elimination of venous congestion after microvascular surgery. They are not known to directly transmit infectious diseases, but bacterial infection can follow bites. Leeches are prodigious feeders, so large infestations can cause significant anemia. Bites from detached land leeches can also bleed for prolonged periods due to injected anticoagulants. Allergic and anaphylactic reactions may occur. Aquatic leeches have potential to cause complications, particularly when attached within the esophagus or airway.

Management and Disposition

Treatment of detached leech bites consists of local wound care, hemostasis if needed, tetanus prophylaxis, and antibiotics if secondarily infected. Attached leeches are usually detected while in the field. They can be detached by carefully using a fingernail to break the suction under the sucker. Many other modalities (perhaps associated with increased risk of infection or other injury) have been suggested, including use of saline solution, vinegar, or a lighted match.

Pearls

1. The presence of a small y-shaped lesion on the lower extremity is suggestive of a detached land leech bite.
2. Aeromonas infections have been reported following use of medicinal leech therapy. Ciprofloxacin may aid in prevention.
3. Hemostasis with QuikClot gauze has been reported to be effective in patients with prolonged bleeding.
4. Leeches have been used for bloodletting in ancient medicine through the 19th century; it was believed the withdrawal of blood would help correct imbalances in four humors of the body or, later, treat a wide range of ailments.
5. Direct thrombin inhibitors, bivalirudin and lepirudin, are derived from the salivary gland of leeches.



FIGURE 21.36 ■ **Leech.** Nonengorged leech attached to thigh of patient in remote area of Tasmania. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.37 ■ **Leech.** Engorged leech attached to toe of a backpacker in Papua New Guinea. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.38 ■ **Leech.** Leech bite wound on the leg seen after detachment of a land leech. Note the sharp wound edges and surrounding ecchymosis. (Photo contributor: Seth W. Wright, MD.)

Clinical Summary

Leishmaniasis is a protozoan, zoonotic parasite spread by sand fly bites. It is endemic in over 80 countries and has an incubation period of 2 to 6 months, though it may range from days to years, with relapse possible. Infection occurs when flagellated forms of more than 20 infective species of Leishmania are injected into the skin, taken up by macrophages, and multiply. Often, the bite goes unnoticed.

The primary cutaneous lesion begins as an enlarging papule, develops a scaly or ulcerative lesion with indurated edges and a central crater, and usually heals spontaneously in 6 to 12 months, but may progress to more diffuse syndromes. Mucocutaneous involvement or "espundia" is mostly seen in Latin America following initial cutaneous infection with Leishmania braziliensis. Nasal congestion and epistaxis may progress to septum perforation and nasal bridge collapse, causing a "tapir nose" deformity. Visceral involvement, known as "kala-azar," involves the spleen and liver. It is associated with fever, anemia, cachexia, splenomegaly, and may progress to hemorrhagic symptoms, secondary infections, and GI involvement. Visceral leishmaniasis is generally fatal without treatment.

Coinfection with advanced HIV causes synergistic immunologic disturbances due to the lack of a critical CD+ T-cell response.

The characteristic parasites can be identified from a smear or biopsy of the lesion identifying parasite amastigotes (Leishman-Donovan bodies). Culture and PCR are also diagnostic options.

Management and Disposition

No specific emergency treatment exists for leishmaniasis. Treatment of cutaneous or mucosal disease is with pentavalent antimony, miltefosine, pentamidine, or amphotericin B after referral to an experienced clinician. Protective clothing, bed nets, and insect repellent are the most effective ways of avoiding transmission as the flies are usually active from dusk to dawn. Visceral leishmaniasis should be managed by a tropical medicine or infectious disease specialist as the treatment is difficult and often toxic.



FIGURE 21.39 ■ **Cutaneous Leishmaniasis.** Lesions in a girl from the highlands of Peru. Most leishmania lesions are on exposed body areas. (Photo contributors: Rob Greidanus, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)



FIGURE 21.40 ■ **Cutaneous Leishmaniasis.** Lesion involving the face of an HIV-infected patient. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

Pearls

1. More than 90% of visceral leishmaniasis cases are in India, Bangladesh, Nepal, Sudan, and Brazil, while more than 90% of cutaneous forms are in Afghanistan, Algeria, Brazil, Iran, Iraq, Peru, Saudi Arabia, and Syria.
2. Leishmaniasis is common in the Middle East and many cases have been identified in troops deployed to that region.
3. Leishmaniasis should be considered in immigrants from Latin America with chronic skin or mucosal lesions.
4. With visceral involvement, the primary cutaneous lesion will usually have resolved before clinical symptoms of kala-azar have developed.
5. After treatment of the visceral form, depigmented or nodular cutaneous lesions that are often confused with leprosy may occur.



FIGURE 21.41 ■ **Mucocutaneous Leishmaniasis.** Mucous membrane involvement (espundia) and "tapir-nose" deformity from Leishmania braziliensis. (Photo contributors: Rob Greidanus, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)



FIGURE 21.42 ■ **Mucosal Leishmaniasis.** Oral mucosal leishmaniasis caused by L braziliensis. Mucosal involvement can occur years after spontaneous resolution of a nontreated cutaneous lesion. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

Clinical Summary

Leprosy (Hansen disease) is caused by Mycobacterium leprae and presents with anesthetic skin lesions and peripheral nerve complications. This chronic disease is common in many parts of the developing world, including areas of South America, South Asia, and Africa. Illness severity is inversely proportional to the patient's ability to produce a cell-mediated response and creates a range from localized (tuberculoid leprosy) to disseminated disease (lepromatous leprosy), with many having borderline cases falling between the two extremes.

Localized disease is limited to one to three skin lesions, typically sharply demarcated, flat, hypopigmented, anesthetic plaques with elevated margins. This is often accompanied by easily discernible peripheral nerve thickening. The most severe forms are characterized by poorly demarcated erythematous macules, papules, or nodules. The peripheral nerves have less palpable findings, but more diffuse nerve involvement is seen in a stocking glove pattern. This results in loss of bone length and insensate extremities, often leading to repetitive trauma, infection, and loss of digits. With diffuse infiltration of the face, the characteristic "leonine facies" is exhibited.

Diagnosis is primarily clinical along with staining of a slit skin smear or biopsy for acid-fast bacilli. PCR can aid in the diagnosis.

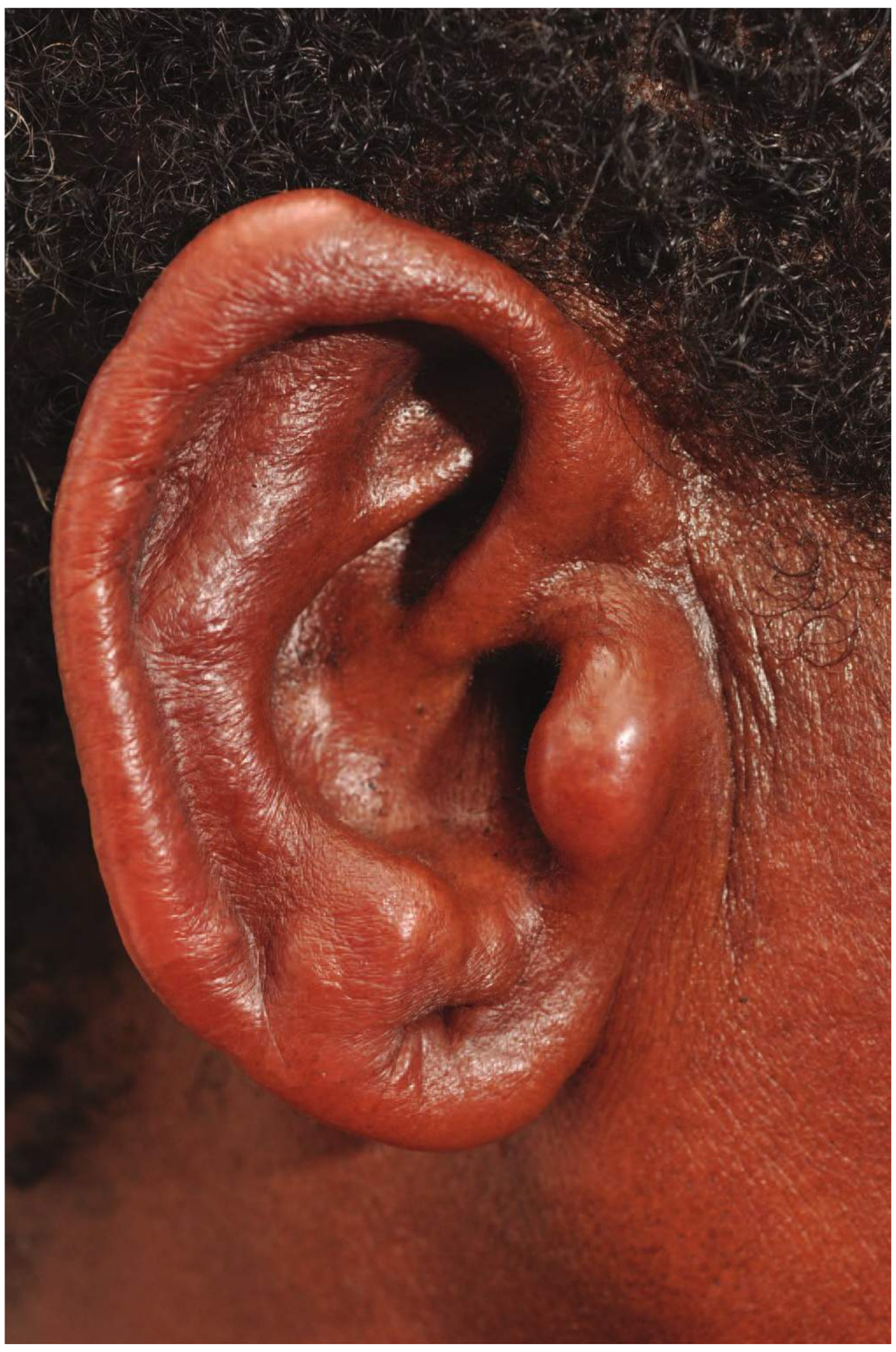


FIGURE 21.43 ■ **Leprosy.** Typical thickened skin on the external ear of a patient with lepromatous leprosy. (Photo contributor: Larry Stack, MD.)

Management and Disposition

There is no specific emergency therapy for leprosy. Patients should be referred to the national treatment program or a clinician experienced with leprosy management. Multidrug therapy is curative using varying combinations of dapsone, rifampin, and clofazimine. The class of disease dictates type and length of treatment. An ophthalmic exam is essential due to a high propensity for corneal abrasions and ulcers.

Pearls

1. Leprosy acquired in the southern United States is highly associated with armadillo handling.

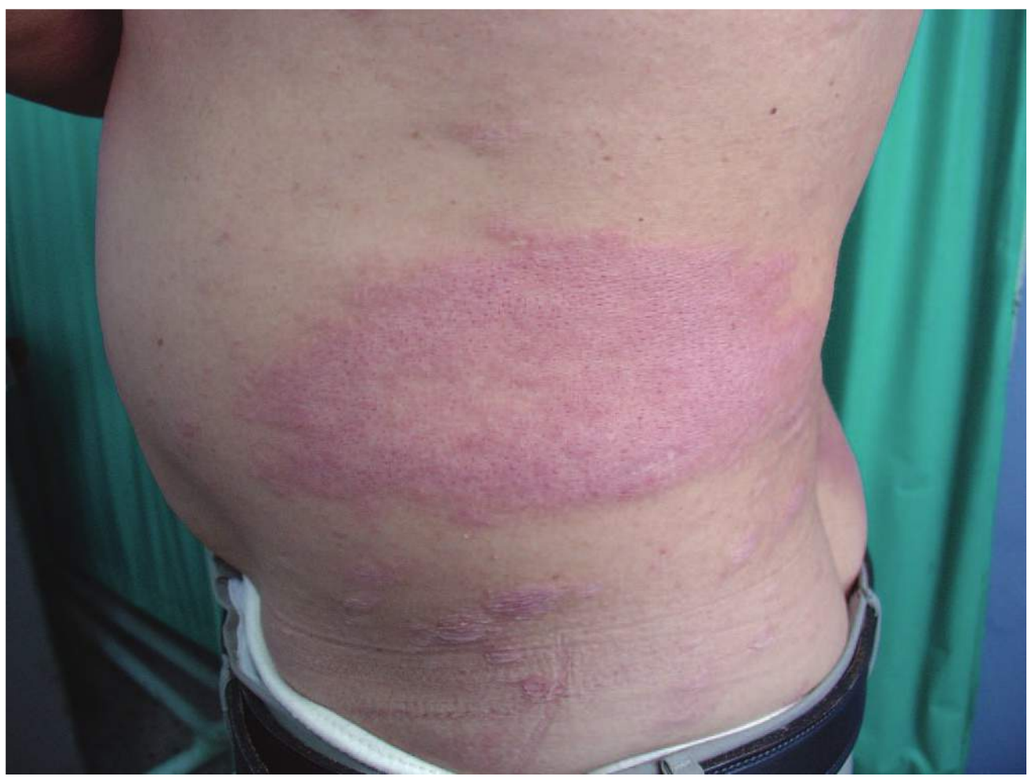


FIGURE 21.44 ■ **Leprosy Skin Lesions.** Anesthetic skin lesions in a Peruvian patient with borderline leprosy. Borderline disease is not as localized as tuberculoid leprosy and not as widespread as lepromatous disease. (Photo contributors: Rob Greidanus, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

2. The presence of an anesthetic skin lesion should suggest the diagnosis of leprosy.
3. Multidrug therapy has been made available by WHO free of charge to all patients worldwide and provides a highly effective cure for all types.
4. The simpler WHO classification categorizes leprosy into localized (paucibacillary) or disseminated (multibacillary) disease.



FIGURE 21.45 ■ **Leprosy.** Thickened skin due to bacillary infiltration. Later stage may require digit amputation. (Photo contributor: David Effron, MD.)



FIGURE 21.46 ■ **Leprosy.** Chronic foot changes with ulceration and shortening of the toes. Extremity damage in leprosy results from loss of sensation, repeated trauma, neurotrophic atrophy, and direct bacillary deposition. (Photo contributor: Seth W. Wright, MD.)

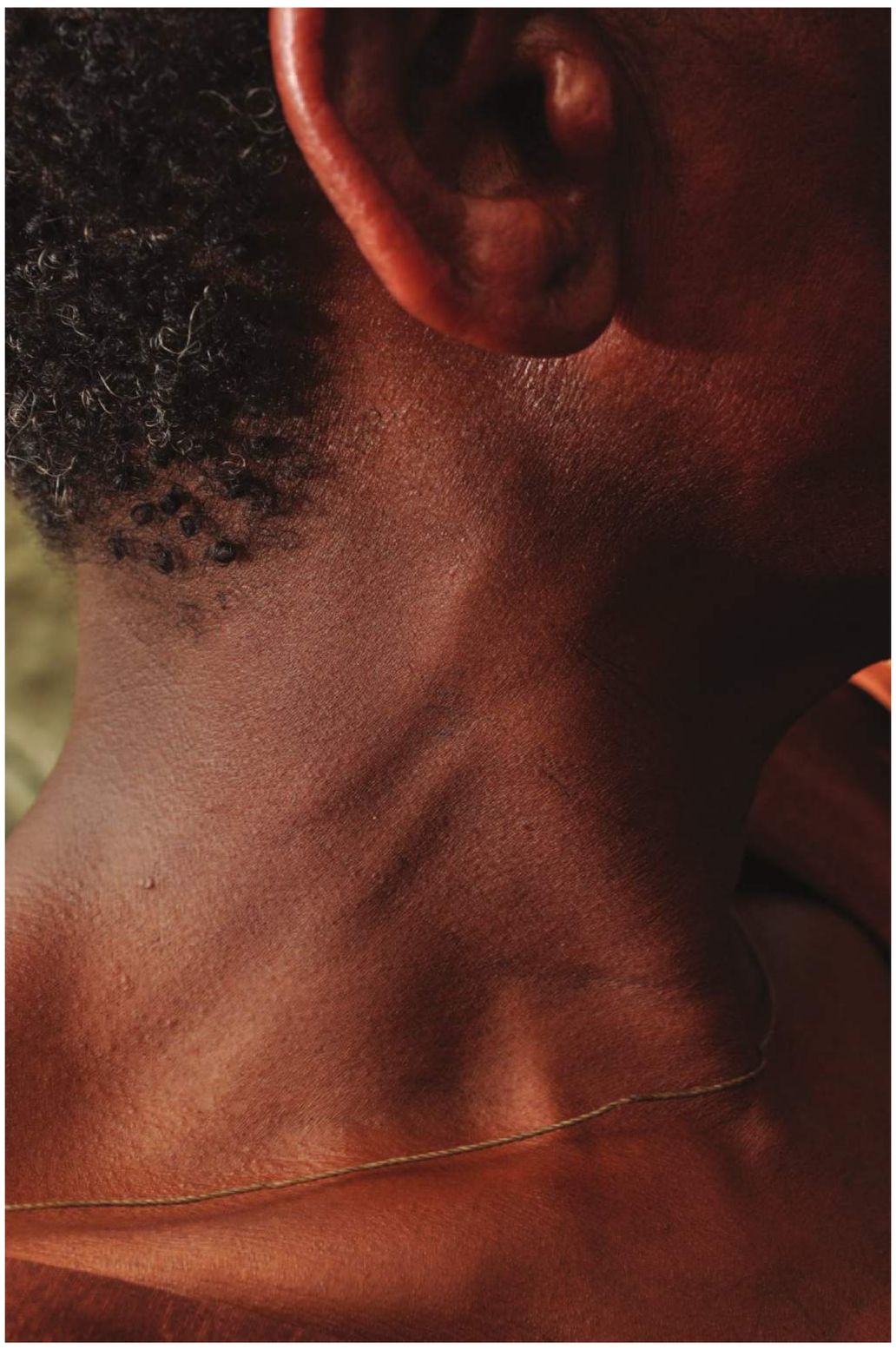


FIGURE 21.47 ■ **Leprosy.** Enlargement of the greater auricular and transverse cervical nerves of a patient with leprosy. Enlarged peripheral nerves are a cardinal manifestation. (Photo contributor: Larry Stack, MD.)

Clinical Summary

Leptospirosis is a zoonotic bacterial disease acquired by exposure to water contaminated by the urine of infected animals. Rodents are the usual reservoir, although pigs, dogs, and cattle can harbor the organism. It is most commonly attained by indirect skin or mucosal contact through occupational work, such as sugarcane or rice farming, or through recreational activities such as rafting, swimming, and hiking. Slaughterhouse workers can acquire leptospirosis through contact with infected body fluids. Leptospirosis is most common in tropical regions, especially during the rainy season.

The incubation period is usually 7 to 14 days; more than 90% of cases are asymptomatic, or mild and self-limited. Typical symptoms include the abrupt onset of high fever, chills, headache, severe myalgias, conjunctival suffusion (dilatation of vessels without inflammation or exudate), cough, jaundice, abdominal pain, rash, or diarrhea. Aseptic meningitis is common. Dermatologic findings are nonspecific and include macular, papular, and petechial rashes. Approximately 10% of patients develop the potentially life-threatening icteric form known as Weil syndrome. The initial symptoms are indistinguishable from the milder form until several days after the onset when complications such as renal failure, liver failure, rhabdomyolysis, pulmonary hemorrhage, or cardiopulmonary failure develop.

Laboratory findings in leptospirosis are nonspecific. An elevated creatine kinase is common. Markedly elevated bilirubin values are seen in Weil syndrome. Culture of the organism is possible, but diagnosis is usually serologic. Rapid test kits are available in some countries.

Management and Disposition

Empiric treatment is often indicated. Outpatient management with doxycycline, penicillin, amoxicillin, or a macrolide can be considered for mild illness. Severe disease is treated with IV penicillin or ceftriaxone. Supportive care, including optimal fluid management, is essential to help prevent or ameliorate renal dysfunction. Severely ill patients often need ventilatory support in an intensive care unit and dialysis.

Pearls

1. About half of the cases reported in the United States are from Hawaii, with increased outbreaks during periods of heavy rainfall and flooding.
2. Consider leptospirosis in an acutely febrile adventure traveler with recent freshwater exposure.
3. The presence of conjunctival suffusions, with or without jaundice, is highly suggestive of leptospirosis.
4. A Jarisch-Herxheimer reaction may occur after treatment with antibiotics.
5. Weekly doxycycline is effective prophylaxis for short-term travelers to highly endemic areas.

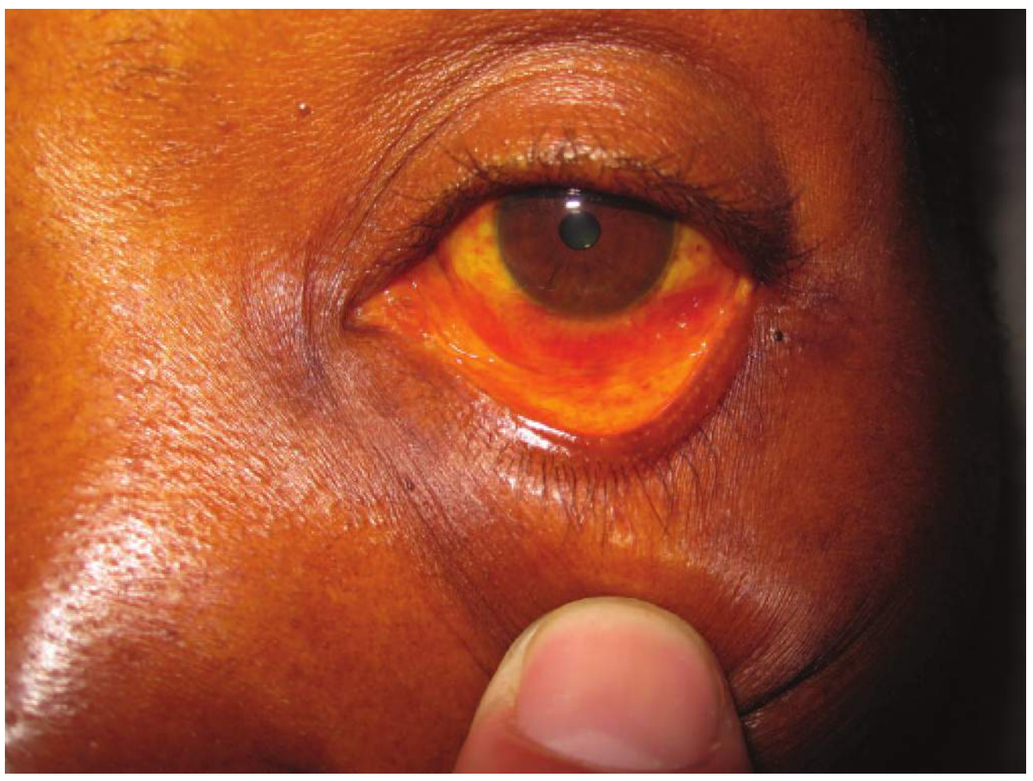


FIGURE 21.48 ■ **Leptospirosis.** Jaundice and conjunctival suffusions in a Haitian patient with laboratory-confirmed leptospirosis. The patient also had renal failure and myocarditis, both commonly seen in Weil syndrome. (Photo contributor: Seth W. Wright, MD.)

Clinical Summary

Malaria infects up to 300 million people annually with over 1 million deaths; it is the most deadly vector-borne disease in the world. This parasitic disease is transmitted by the night-biting female Anopheles mosquito and is caused by four protozoa of the genus Plasmodium (Plasmodium falciparum, Plasmodium malariae, Plasmodium ovale, and Plasmodium vivax), with P falciparum causing the most morbidity and mortality. It is most common in tropical areas, particularly sub-Saharan Africa and Southeast Asia, with specific species predominating in each area.

The parasites undergo a hepatic cycle before entering circulating red blood cells and replicating. Lysis of the cell then occurs, releasing toxic by-products and further parasites into the blood, thereby causing cyclical clinical manifestations. Symptoms include fever, rigors, headache, myalgias, and malaise. The classic cyclical fevers do not always occur. P falciparum infection can cause massive hemolysis due to overwhelming parasitemia. Parasitized erythrocytes lose flexibility leading to microcirculatory obstruction, hypoxia of vital organs, and splenomegaly. Symptoms from P ovale and P vivax may be delayed for many months due to hepatic dormancy. Pregnancy is a risk factor for severe P falciparum infection and can result in maternal anemia, prematurity, and increased infant mortality.

Diagnosis is established by identification of the parasites on thick and thin smears. Rapid antigen kits are available in some regions. Bed nets, insecticides, and protective clothing are effective adjuncts for prevention. Chemoprophylaxis with an appropriate agent is highly recommended for travelers to endemic regions.

Management and Disposition

Treatment depends upon geographic location, suspected species, and illness severity. Close adherence to CDC, WHO, or national guidelines is highly recommended for both prophylaxis and treatment. Oral treatment of uncomplicated malaria is feasible with one of several regimens. Quinidine is the only approved treatment for severe malaria in the United States, although artesunate is available on an emergency basis from the CDC when quinidine is not tolerated or available. Artemisinin combination therapy for severe disease is now



FIGURE 21.49 ■ **Malaria Jaundice.** Jaundice due to acute hemolysis in a Haitian child with documented P falciparum infection. (Photo contributor: Seth W. Wright, MD.)

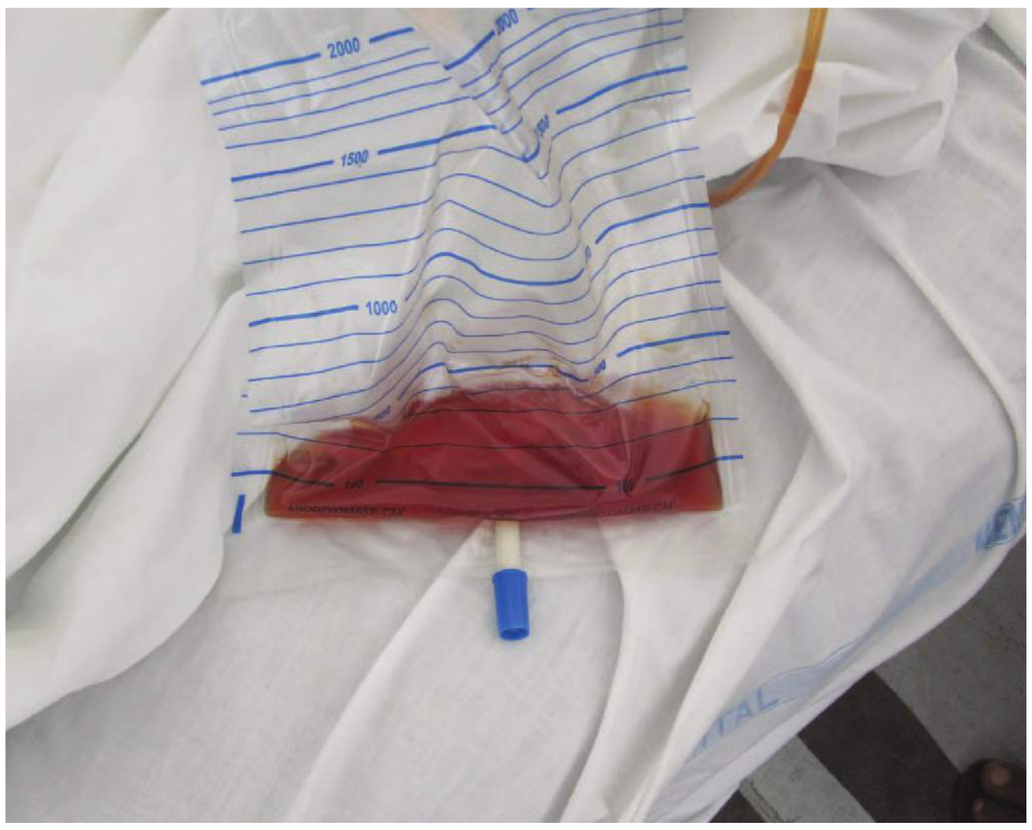


FIGURE 21.50 ■ **Malaria.** Hemoglobinuria following hemolysis in the patient from Fig. 21.49. The patient was severely anemic with a hemoglobin of 3.0 mg/dL. (Photo contributor: Seth W. Wright, MD.)

available in many endemic countries, although IV quinine remains a common treatment. Patients in developing countries are often treated as outpatients. With rare exception, returning travelers with suspected malaria should be admitted for management and treatment. Admission is always warranted for patients with suspected or confirmed P falciparum, symptoms of cerebral malaria, children, pregnant women, or immunocompromised individuals.

Pearls

1. Malaria should be strongly considered in any patient exhibiting fever following recent travel to the tropics. It is the single most common cause of fever in this population.
2. An emerging species, Plasmodium knowlesi, causes severe disease, but is currently limited in distribution to Southeast Asia.
3. The average incubation period for P falciparum is about 13 days; average incubation periods are longer for the other species. Incubation periods for all species can be variable.
4. Parasitemia fluctuates over time with the highest incidence during episodes of fever. Failure to identify the parasites on initial smears is not an indication to withhold therapy when the diagnosis is likely.
5. Residents of highly endemic areas will often have partial immunity to malaria due to repeated exposure. Immigrants visiting their native country are at high risk for severe infection due to loss of immunity.



FIGURE 21.51 ■ **Malaria.** Massive hyperreactive malarial splenomegaly in a surgical patient. This condition can occur with chronic infection, but malaria smears are often negative. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)



FIGURE 21.52 ■ **Malaria.** A toddler with cerebral malaria. Abnormal motor posturing including decorticate, decerebrate, and opisthotonic posturing are all common in this condition. (Photo contributor: Seth W. Wright, MD.)

MUMPS

Clinical Summary

Mumps is a highly contagious viral disease best known as a cause of parotitis. Vaccination has markedly reduced the rate of disease in the developed world, but many resource-limited countries do not include mumps in their vaccine programs. It remains common worldwide; occasional large outbreaks occur even in countries with well-developed vaccine programs. Mumps is most common in children and spread by respiratory droplets, direct contact, or fomites. Subclinical infection is common in younger children. Disease in adolescents and adults is associated with a higher rate of serious complications.

A nonspecific illness with malaise, myalgias, fever, and headache follows an incubation period of about 2 to 3 weeks. Unilateral or bilateral parotid swelling is the hallmark, although orchitis is seen in about one-third of postpubertal males and pancreatitis in about 4% of patients. Diagnosis is usually clinical, particularly in developing countries. A high amylase from a salivary or pancreatic source might be present; viral isolation, PCR, and serology are available for definitive diagnosis.

Management and Disposition

Mumps is a self-limited, usually mild, disease. Treatment is supportive and consists of antipyretics, fluids, and analgesics. Patients with orchitis might benefit from scrotal support. Patients should be advised to stay home from school or work for 5 days after symptom onset.

Pearls

1. Natural infection confers life-long protection, although rare recurrent cases have been reported.
2. Aseptic meningitis with CSF pleocytosis occurs in more than one-half of patients with mumps, although most are minimally symptomatic. Mumps encephalitis also occurs, but is extremely rare.
3. Mumps should be considered in the differential diagnosis of aseptic meningitis, even in the absence of parotitis.
4. Mumps is one of the leading causes of acquired deafness in developing countries.
5. Ovarian and breast inflammation are occasionally seen in females, although it is not as common as orchitis among males. Sterility is rarely seen with either condition.



FIGURE 21.53 ■ **Mumps.** Parotid swelling in a Haitian girl with mumps. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.54 ■ **Mumps.** Note the classic submandibular and preauricular enlargement of the parotid gland. (Photo contributor: Centers for Disease Control and Prevention.)

Clinical Summary

Mycetoma is a localized, chronic, granulomatous infection of subcutaneous tissue with possible extension to underlying bone. Two classifications include eumycetoma, caused by filamentous fungi, and actinomycetoma, caused by bacteria of actinomycetes species. The organism is inoculated into subcutaneous tissue following minor trauma, most commonly the lower extremity and hand, though it may arise anywhere on the body. Both types are similar clinically; eumycetoma, however, causes more morbidity. Initially, a painless subcutaneous swelling is seen with induration, numerous suppurative nodules, and chronically draining sinus tracts. Remote abscesses may rarely be seen due to hematogenous extension. Actinomycetoma occurs more frequently (60%) and has a much better outcome. Mycetomas are rarely fatal, but may cause significant dysfunction and disfigurement.

Expulsion of "grains" containing aggregates of the organisms is common. The presence of black grains is diagnostic of a fungal origin, while pale grains could be either fungal or from an actinomycetes species. Further identification of the causative organism may be done by means of various stains, culture, or by serology.

Management and Disposition

There is no specific emergency management except recognition and referral. Surgical resection of large lesions may reduce organism load, but relapse rates can be as high as 50%. Amputation is common. Eumycetoma is treated with 1 to 2 years of antifungal agents, while actinomycetoma responds to various combinations of trimethoprim-sulfamethoxazole, dapsone, streptomycin, and amikacin.

Pearls

1. Mycetoma is usually seen in young male adults living in rural areas of Africa, Mexico, South America, or India who work as farmers or laborers. Walking or working barefoot is a risk factor for lower extremity disease.
2. Patients may complain of a deep itching sensation rather than pain. If pain is present, it may indicate secondary infection or bone involvement.
3. Sweating of the affected area is commonly seen.
4. A similar condition, botryomycosis, is caused by a chronic Staphylococcus infection with sinus formation.
5. Mycetoma was first described in the Madura district of India and is often referred to as "Madura foot."



FIGURE 21.55 ■ **Mycetoma.** A eumycetoma with typical chronic sinus drainages in the foot and ankle of a 33-year-old agricultural worker in Peru. Treatment was with itraconazole. (Photo contributors: Rob Griedanus, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

FIGURE 21.56 ■ **Mycetoma.** A large actinomycetoma of the upper leg with extensive active and healed sinus tracts. (Photo contributors: Stuart Skinner, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

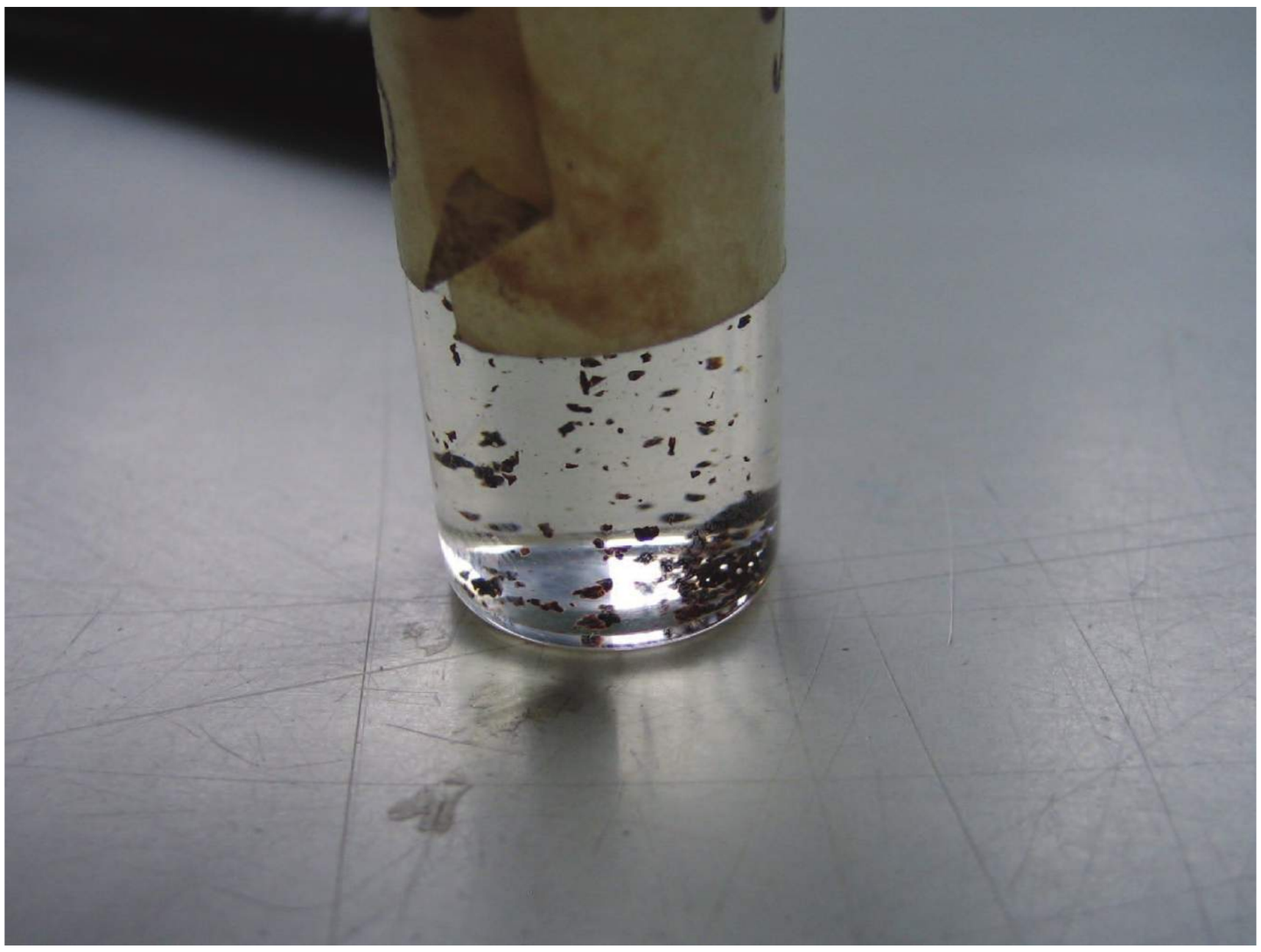


FIGURE 21.57 ■ **Mycetoma Granules.** Sinuses discharge characteristic dark granules (sclerotia) from a eumycetoma. The granules represent microcolonies of the organism. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)



FIGURE 21.58 ■ **Mycetoma.** Mycetoma of the foot from Nocardia sp.: typical location with multiple draining sites. (Reproduced with permission from Goldsmith LA, Katz SI, Gilchrest BA, Paller AS, Leffell DJ, Wolff K. Fitzpatrick's Dermatology in General Medicine. 8th ed. McGraw-Hill; 2012: Fig. 185-6, p. 2249.)

Clinical Summary

Myiasis is the invasion of living human or animal tissue by maggots, the larvae of flies. Infection is most commonly subcutaneous, but may be seen in wounds and body cavities. The human botfly (Dermatobia hominis) is a major cause of furuncular myiasis in the New World; mosquitoes courier eggs to the host. A papule develops as the larvae feed, followed by a pruritic furuncle, often with drainage from a central punctum. After maturation, the larva emerges and falls to the ground, where it pupates in the soil and evolves into an adult fly.

The tumbu fly, found in Africa, also causes furuncular myiasis and is spread from eggs deposited in soil, sand, or clothes. Screwworm species are found in the Old and New Worlds. These worms are notorious for direct deposition of eggs after flying into the nasal cavity, leading to nasal cavity myiasis. Feeding maggots can cause extensive tissue damage. Wound myiasis, caused by numerous fly species, is seen in open sores and gangrenous tissues. Wearing protective clothing, utilizing insect repellant, and covering wounds are preventative measures.

Management and Disposition

Lidocaine injection at the lesion base, or application of suffocating occlusive substances such as Vaseline, may cause the larva to surface and facilitate removal in furuncular myiasis. Incisional extraction is challenging due to the larva's tapered shape and many rows of spines and hooks that it uses to grip tissue. Wound myiasis is treated with thorough debridement. Secondary infection is the only indication for antibiotics; it is uncommon due to bacteriostatic activity in the gut of the larvae, but may occur by leaving portions of them after removal.

Pearls

1. Patients with lesions may have the sensation of something moving under the skin.
2. Myiasis is self-limiting and usually not harmful, but it does cause psychological distress to the host, especially when the larvae surface. Though rare, deaths from meningitis have occurred after tissue penetration from infection in the eye, nose, or ear canal.
3. Screwworms are major pests of livestock. A sterile male release program has eliminated the screwworm from the United States.
4. Some feel that fly maggots are a useful method of wound debridement. Packets of fly maggots have been commercially developed for this purpose.



FIGURE 21.59 ■ **Myiasis.** Preserved specimens of D hominis. (Photo contributors: Rob Greidanus, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)



FIGURE 21.60 ■ **Wound Myiasis.** Wound myiasis in an elderly patient following dressing removal. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.61 ■ **Myiasis.** Wound on the arm of a woman who had visited the Amazon jungle. A D hominis larva was extracted from the wound. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)



FIGURE 21.62 ■ **Tumbu Fly Lesions.** Multiple lesions from tumbu fly infestation on the skin of a 14-month-old American boy living in Botswana. The child had been wearing clothes dried on infected ground. (Photo contributor: Anonymous.)

Clinical Summary

Mycobacteria other than tuberculosis and leprosy cause disorders primarily seen in tropical countries. As opposed to Mycobacterium tuberculosis and M leprae, they are usually environmental saprophytes, not obligate human/animal pathogens. Human exposure may be via inhalation, contaminated water, or preexisting wound contamination. Overall, nontuberculosis mycobacteria can cause four different clinical syndromes: pulmonary disease, regional lymphadenitis, disseminated disease in immunocompromised patients, and skin/soft tissue infections.

Buruli ulcer is the most important disorder caused by infection with Mycobacterium ulcerans. They are seen throughout the tropics, but are particularly prevalent in Africa. The ulcer is slow-growing with extensive necrosis and deep undermining of wound edges. Serious complications can occur, including osteomyelitis, contractures, and scarring.

A variety of other cutaneous forms of mycobacterial diseases exist. Fish tank granuloma (from Mycobacterium marinum) is seen following handling of tropical fish tanks and occasionally in fishermen. Posttraumatic abscesses are caused by the rapidly growing mycobacteria, Mycobacterium fortuitum and Mycobacterium chelonae.

Disseminated disease from Mycobacterium avium complex is a late-stage opportunistic infection in HIV/AIDS, but is occasionally seen in immunocompetent individuals.

Management and Disposition

Providers should consider these illnesses in the differential diagnosis when patients present with typical skin lesions and in severely immunocompromised AIDS patients. Buruli ulcer is typically treated for 8 weeks with rifampin and either streptomycin or clarithromycin. Surgical excision with grafting may be needed. Other infections are treated with macrolides or other antibiotics.

Pearls

1. Buruli ulcer is the third most common mycobacterial disease worldwide, after tuberculosis and leprosy.
2. Consider M marinum infection on patients with a hand lesion following tropical fish tank manipulation.
3. Fast-growing mycobacteria such as M chelonae cause "cold abscesses," often seen after nonsterile injections that are given for cosmetic purposes. A recent outbreak of this organism was also seen following tattooing.
4. Some postulate that Crohn disease and sarcoidosis may be caused by a yet unidentified mycobacteria.
5. Mycobacterium haemophilum is considered the second most common cause of cervical lymphadenitis in children.



FIGURE 21.64 ■ **Cold Abscess.** A cold abscess following a cosmetic injection for weight loss. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)



FIGURE 21.63 ■ **Buruli Ulcer.** Typical shallow base and deep undermining of the edges in a patient from Benin. (Photo contributor: World Health Organization.)



FIGURE 21.65 ■ **Cold Abscesses.** A patient with multiple recurrent cold abscesses from M chelonae following nonsterile weight loss injections in Peru. (Photo contributors: Rob Greidanus, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

PROTEIN-ENERGY MALNUTRITION—KWASHIORKOR AND MARASMUS

Clinical Summary

Protein-energy malnutrition (PEM) applies to a group of disorders including kwashiorkor and marasmus. These are characterized by an imbalance between the body's supply and demand of energy and nutrients. Kwashiorkor means "the sickness of weaning" as it is often seen following weaning after birth. It usually occurs between the ages of 1 and 4 with a deficiency of dietary protein in the presence of normal to high carbohydrate intake. The etiology is complex, but there is decreased synthesis of proteins resulting in hypoalbuminemia. Kwashiorkor is an acute illness manifested by edema secondary to fluid and sodium retention. Patients have peripheral edema, anasarca, moon facies, apathy, and a protuberant abdomen secondary to hepatomegaly. The skin can become hyperkeratotic and may split open in pressure-prone areas.

Marasmus is often seen under the age of 1 and is associated with inadequate intake of both protein and calories. The body's own energy stores are utilized, resulting in emaciation. These individuals do not have edema, but rather a loss of subcutaneous fat, muscle wasting, and wrinkled loose skin. Marasmic-kwashiorkor refers to the combination of both forms simultaneously. Laboratory studies are useful, but often are not available. Patients with PEM may be left with permanent neurological and physical deficits due to lack of calories, vitamins, and essential amino acids.

Management and Disposition

These conditions are rarely seen or treated in developed countries, so acute treatment is often in settings with limited resources. These children often have other acute illnesses and are also at greater risk for infection, specifically pneumonia, gastroenteritis, and sepsis. IV fluids are limited to those with shock as they are prone to congestive heart failure; WHO oral rehydration salt solution is usually used for the first 6 to 10 hours. Low-protein milk formula is then started with a goal



FIGURE 21.66 ■ **Kwashiorkor.** A Zambian child with typical light hair, moon facies, peripheral edema, and dry skin of kwashiorkor. (Photo contributor: Meg Jack, MD.)



FIGURE 21.67 ■ **Marasmus.** An African infant with severe marasmus due to poor feeding following maternal death. The infant is severely underweight with loose skin and little subcutaneous fat. (Photo contributor: Meg Jack, MD.)



FIGURE 21.68 ■ **Kwashiorkor.** A Ugandan boy with edema typical of acute kwashiorkor. The "enamel paint peeling" rash is occasionally seen. The patient died several days after the photo was taken. (Photo contributor: Seth W. Wright, MD.)

of 100 kcal/kg/day. A normal diet is gradually started over a few weeks. Inpatient admission for evaluation, intervention, and arrangement of long-term care is advised when possible.

Pearls

1. PEM is one of the leading causes of death among children <5 years of age worldwide.
2. Depigmentation of dark hair causes it to turn reddish, and curly hair may become straight and brittle. Intermittent periods of proper nutrition may lead to alternating bands of light and dark hair known as the "flag sign."
3. Kwashiorkor is an acute illness, often of only several days duration. It is commonly precipitated by acute infections.
4. The mid upper arm circumference (MUAC) is often used for rapid assessment of nutritional status in developing countries.
5. Newer nutritional products, such as "Plumpy'nut," have been developed and allow for outpatient management.

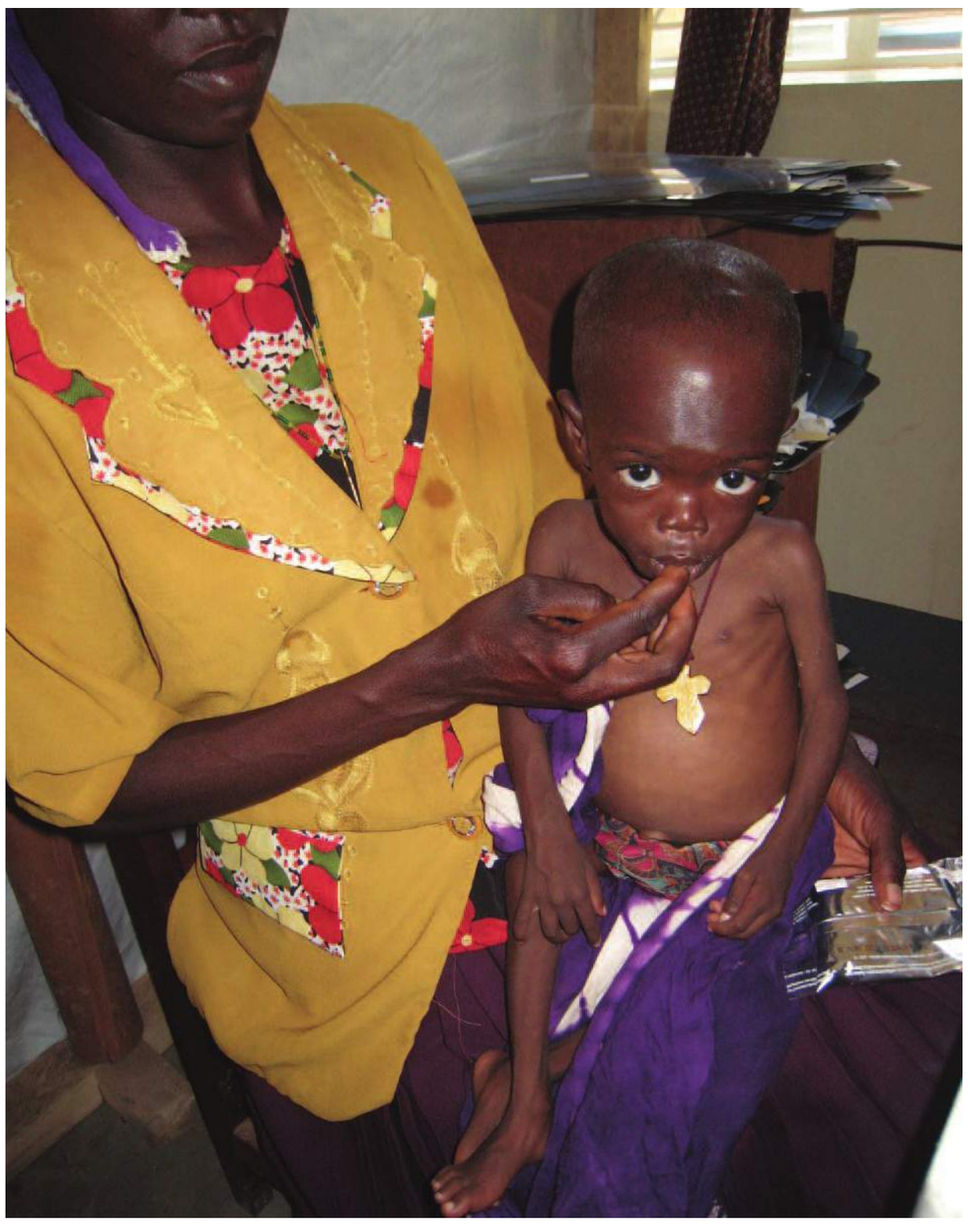


FIGURE 21.69 ■ **Marasmus.** An HIV-positive Ugandan girl recovering from marasmus. She is eating Plumpy'nut, a commonly used ready-to-use therapeutic food. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.70 ■ **Marasmus.** An African boy with marasmus. (Photo contributor: Seth W. Wright, MD.)

Clinical Summary

Rabies is a viral zoonotic disease typically acquired through the bite of an infected animal. Transmission via aerosolized virus has been documented in bat-infested caves and in laboratory workers. Human-to-human transmission has only been documented in transplant recipients. Infection can be prevented by preexposure or postexposure vaccination.

Rabies has the dubious distinction of having the highest case fatality rate of any infectious disease. It is a severe progressive encephalitis and presents in one of two forms. In 80% of cases, patients will develop the encephalitic (furious) form, with typical symptoms of fever, agitation, laryngeal spasms, severe pain on swallowing, hydrophobia, confusion, and a host of other neurologic signs and symptoms. Patients with the paralytic (dumb) form (20%) present with an ascending paralysis similar to Guillain-Barré syndrome. Patients with either form often have preceding paresthesias and pain at the exposure site, as well as a nonspecific prodromal phase lasting up to 1 week. Autonomic instability is seen in all patients prior to death. The diagnosis of rabies in developing countries is primarily clinical. Fluorescent antibody testing, biopsy, and PCR are done in areas where available.

Management and Disposition

Management of clinical rabies is traditionally palliative as the outcome has been uniformly fatal. Vaccination after development of clinical rabies is futile. Treatment in developing countries is usually limited to sedation with benzodiazepines and other comfort measures. Survival of several patients using the "Milwaukee protocol" has been reported. This uses an induced coma (midazolam and ketamine) with continuous electroencephalography (EEG) monitoring, antiviral agents (amantadine), and aggressive diagnosis and treatment of cerebral vasospasm. This regimen is considered experimental in the United States and is not practical in most developing countries.

Pearls

1. Insect-eating bats cause the majority of US rabies cases, but most patients are unable to recall a bite. Dog bites are the most common cause worldwide.
2. The shortest incubation periods are associated with extensive bites and bite wounds to the face and scalp.
3. Rabies vaccine and immunoglobulin are crucial in avoiding rabies following an exposure. They are, however, contraindicated after the onset of clinical rabies as they might lead to an alteration of the natural immune response.
4. Human-to-human transmission is extremely rare or nonexistent. Nevertheless, postexposure prophylaxis of close contacts is recommended as the virus is present in saliva.
5. A report from Peru has suggested the possibility of subclinical rabies disease in Amazon residents.



FIGURE 21.71 ■ **Rabies.** A 20-year-old Ugandan patient with furious rabies on the fourth day of clinical illness after a dog bite 2 months earlier. The neck is visibly swollen due to subcutaneous emphysema. The patient developed spontaneous pneumomediastinum due to forceful vomiting during a period of severe laryngeal spasms. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.72 ■ **Rabies.** Same patient on the sixth day, now unable to swallow or control secretions due to laryngeal pain and spasms. (Photo contributor: Seth W. Wright, MD.)

Clinical Summary

Over 200 million people worldwide are infected with schistosomiasis, with approximately 200,000 annual deaths. Transmission has been documented in over 75 tropical countries; it is considered the second most devastating parasitic disease, behind malaria. Humans become infected by contact with blood flukes in contaminated freshwater, usually through bathing, swimming, fishing, or agricultural work. The infectious larval form of the worm, known as cercariae, penetrates the skin, develops into an adult, and deposits eggs causing a host immune response and tissue damage.

Acute symptoms are most often seen in nonimmune individuals such as travelers. Cercarial dermatitis, or swimmer's itch, is a pruritic papular rash discovered immediately following exposure. Katayama fever may occur 2 to 8 weeks after exposure from systemic hypersensitivity and resembles serum sickness: fever, cough, chills, myalgias/arthralgias, diarrhea, lymphadenopathy, and hepatosplenomegaly. Resolution usually occurs after a few weeks, but patients can progress to coma or death.

Chronic symptoms are related to parasite load and involved organs. Intestinal symptoms (caused by Schistosoma mansoni) include abdominal pain, poor appetite, bloody diarrhea, with possible hepatosplenomegaly, periportal fibrosis, ascites, and portal hypertension. Urogenital disease (caused by Schistosoma haematobium) typically causes hematuria and dysuria, as well as genital lesions and vaginal bleeding in women. It can progress to fibrosis and calcification of the bladder and ureters, leading to hydroureter/nephrosis, infection, glomerular disease, and bladder cancer. Schistosomiasis is also associated with neurologic and pulmonary complications, including transverse myelitis, seizures, and pulmonary hypertension. Diagnosis is made through egg detection via stool or urine microscopy. Anemia and eosinophilia can be found in up to two-thirds of patients. Serology is useful for diagnosis in travelers.

Management and Disposition

Cercarial dermatitis usually resolves within 1 week; topical or systemic glucocorticoids may be considered. Therapy for Katayama fever is supportive, although glucocorticoids might be a useful adjunct. Praziquantel is effective against all forms of chronic disease but is not active on immature worms. Thus, patients with acute Katayama fever require delayed treatment in order to eliminate matured worms. Central nervous system (CNS) disease is treated with a combination of praziquantel and steroids.

Pearls

1. Hematuria is a common presenting symptom in areas endemic for S mansoni. These patients are often treated empirically with praziquantel.
2. Schistosomiasis can impact other infections, leading to more severe disease in patients with HBV, HCV, HIV, and malaria.
3. Prevention includes avoiding high-risk waterways, wearing protective boots, and vigorous toweling/drying following contact/exposure.
4. Swimmer's itch can occur as an initial manifestation of human schistosomal infection but is most often caused by avian schistosomes and is not otherwise infectious.



FIGURE 21.73 ■ **Schistosomiasis.** Bladder thickening and calcification in a patient with S haematobium infection. (Courtesy of the J.D. MacLean Centre for Tropical Diseases at McGill University.)



FIGURE 21.74 ■ **Schistosome Cercarial Dermatitis.** Rash due to avian schistosomes in a man following freshwater exposure in a Guyana river. Cercarial dermatitis usually follows freshwater exposure, but occasionally is seen with salt water. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.75 ■ **Schistosome Cercarial Dermatitis.** A highly pruritic papulovesicular eruption on the knee acquired after wading through a slow-flowing creek. (Reproduced with permission from Wolff K, Johnson RA, Saavedra AP (eds). Fitzpatrick's Color Atlas & Synopsis of Clinical Dermatology. 7th ed. New York, NY: McGraw-Hill; 2013: Fig. 28-26.)

Clinical Summary

The infective spores of Clostridium tetani are widely distributed in soil and resistant to heat and disinfectants. Disease occurs when toxin forms after organism growth in wounds; it is characterized by acute onset of skeletal muscle rigidity and convulsive spasm.

The toxin affects inhibitory GABA and glycine receptors, leading to unopposed contraction and spasm of skeletal muscle. Initial symptoms involve the facial musculature producing trismus (lockjaw) and risus sardonicus (sneering grin). As larger muscles are involved, one may see opisthotonos, arm flexion and abduction, fist clenching against the thorax, abdominal rigidity, and lower extremity extension. Other symptoms include laryngeal spasm resulting in asphyxia, seizures, hyperthermia, hypertension, diaphoresis, and tachycardia. Reflex spasms may be triggered by minimal external stimuli. Fractures, dislocations, and rhabdomyolysis may occur due to forceful sustained muscle contractions.

Management and Disposition

Diagnosis is primarily clinical. Treatment includes airway protection, metronidazole or penicillin, active immunization with tetanus vaccine, tetanus immune globulin, benzodiazepines, and supportive therapy. Wounds should be cleansed and debrided to eliminate further toxin production. Tetanus immune globulin facilitates removal of unbound tetanus toxin, but does not affect toxin attached to nerve endings. Recovery of nerve function requires sprouting of new terminals and formation of new synapses which may take months to occur.

Pearls

1. There are four clinical forms of tetanus: local, cephalic, neonatal, and generalized disease (most common).
2. Neonatal tetanus occurs in infants born without passive immunity 3 to 21 days after birth. Inoculation usually occurs through infection of the umbilical stump and is common in developing countries.
3. Magnesium sulfate decreases spasms and cardiovascular instability.
4. In developed countries, tetanus is primarily a disease of the elderly, diabetics, and inadequately vaccinated immigrants.
5. Tetanus disease does not always lead to immunity.



FIGURE 21.76 ■ **Tetanus.** Fatal generalized tetanus in an 8-year-old Haitian child resulting from an infected puncture wound. Severe opisthotonic posturing and rigid jaw clenching are evident. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.77 ■ **Tetanus.** Abdominal wall contractions in a 12-year-old Ugandan girl with tetanus. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.78 ■ **Tetanus.** Severe facial tetany. The distorted grin and raised eyebrows is known as risus sardonicus. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.79 ■ **Neonatal Tetanus.** Neonatal tetanus in a 11-day-old infant. Opisthotonic posturing, muscular rigidity, and inability to swallow are seen. (Photo contributor: Seth W. Wright, MD.)

Clinical Summary

Trachoma is the leading cause of infectious blindness in the world. It is endemic in areas of Africa, Asia, Latin America, the Middle East, and aboriginal communities in Australia. It is a chronic follicular conjunctivitis caused by Chlamydia trachomatis. Trachoma is prevalent in populations with limited access to adequate sanitation and clean water; it is spread through ocular and respiratory secretions as well as flies.

The distinct active phase produces mild itching, irritation, and eye discharge, as well as conjunctival inflammation, particularly of the superior tarsal plate. This may progress to marked photophobia, blurred vision, and eye pain. The scarring, or cicatricial, phase occurs after repeated or severe infection; chronic inflammation causes the upper lid to shorten (entropion), with subsequent eyelash inversion (trichiasis). Trichiasis may cause painful corneal abrasions and subsequent corneal edema, ulceration, scarring, opacities, and, ultimately, blindness. Lacrimal gland involvement leads to dryness and increased irritation. Trachoma usually affects both eyes. Diagnosis is largely clinical, but may be confirmed by culture. Community-based efforts on hygiene education and behavior modification can decrease the incidence.

Management and Disposition

For acute and subacute infections, the most effective treatment is a single dose of azithromycin; tetracycline ophthalmic ointment is an alternative. Eyelid surgery to correct trichiasis and entropion may prevent blindness.

Pearls

1. Trachoma was once endemic to North America and Europe, but has disappeared with improved sanitation and living conditions.
2. It is extremely contagious and may be spread through direct contact with eye, nose, or throat secretions.
3. Young children are particularly susceptible, but the disease progresses slowly and the more painful symptoms may not emerge until adulthood.
4. Adult women are at much greater risk due to their close contact with small children who are the main reservoir of infection.
5. The WHO is attempting to eradicate trachoma by 2020 through their public health program known as SAFE. This multifaceted approach involves surgery for advanced disease, antibiotics, facial cleanliness, and environmental/sanitation improvements.



FIGURE 21.80 ■ **Trachoma.** Extensive inflammatory response with trichiasis. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.81 ■ **Trachoma.** Chronic corneal scarring with excessive tearing in an African patient. (Photo contributor: Meg Jack, MD.)

Clinical Summary

According to WHO, traditional medicine is the sum total of the knowledge, skills, and practices based on the theories, beliefs, and experiences indigenous to different cultures, whether explicable or not, used in the maintenance of health as well as in the prevention, diagnosis, improvement, or treatment of physical and mental illness. Such practices are common worldwide and, while frequently used in developed countries, they are even more prevalent in the tropics. As many as 80% of people in some countries rely upon traditional medical practices as their primary form of health care. Poverty, lack of education, decreased access to standard medical care, and mistrust of Western medical concepts all play a role.

Traditional medicine practices are as varied as the societies of the world. Local practices for acute and chronic illnesses might include prayer, meditation, diets, fasting, massage, exercise, herbal remedies, acupuncture, skin scraping, and scarification. Underlying principles of traditional therapies vary greatly; in some societies, they are related to the balance or homeostasis between negative (bad, dark, devil, etc) and positive (good, light, angels, etc) forces. Ayurvedic medicine is practiced throughout South Asia and roughly translates to "the science of life." This common practice seeks to promote spiritual harmony based upon the theory that health exists



FIGURE 21.83 ■ **Evil Eye Make-Up.** Make-up on a girl in Bangladesh used to ward off the "evil eye," a sickness transmitted by someone who is envious, jealous, or covetous. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.82 ■ **Coin Rubbing.** Typical coin-rubbing marks on the back of a Southeast Asian immigrant with a minor illness. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.84 ■ **Traditional Healing Practice.** Razor marks placed by a traditional healer in a Zambian patient with fever and cough. A chest x-ray showed an infiltrate corresponding to the razor marks. (Photo contributor: Seth W. Wright, MD.)

when there is a balance between body, mind, and spirit. Other concepts, such as witchcraft or the "evil eye," are prominent in dozens of countries. The evil eye, or ocularis sinister, is thought to be the cause of a curse, spell, misfortune, or disease, and various amulets, decorations, or procedures may be used to ward off the unwanted effects.

Pearls

1. More than 70% of Americans using traditional therapies will not inform their physician of their use.
2. The majority of pediatric fevers from malaria are treated with herbal medicines at home in many African countries.
3. Coin rubbing (cao gio) is a common treatment for minor illnesses in Southeast Asia and is frequently seen in US immigrants. This should not be mistaken for child abuse.
4. Many common medications, including important antimalarial agents, are derived from traditional herbal remedies.
5. Ayurvedic and Unani systems have a recognized place in national health programs in India, and have hundreds of thousands of registered practitioners.



FIGURE 21.85 ■ **Fire Cupping.** Fire cupping is used in traditional Chinese medicine for a variety of ailments including musculoskeletal pain and various respiratory, digestive, and gynecologic diseases. The distinctive temporary cupping marks develop as a result of vacuum formation within the cups as the heated air cools. (Photo contributor: Allison Bollinger, MD.)



FIGURE 21.86 ■ **Traditional Healing Practice.** Use of plant material for healing of an otherwise untreated open ankle fracture following the 2010 Haiti earthquake. (Photo contributor: Seth W. Wright, MD.)

TROPICAL SPOROTRICHOSIS

Clinical Summary

Sporotrichosis is a subcutaneous infection caused by the fungus Sporothrix schenckii and is acquired from moss, decaying vegetation, hay, and soil. It is usually seen in those whose vocation brings them into contact with the environment. The two primary manifestations involve the cutaneous and lymphocutaneous systems; however, osteoarticular, pulmonary, and disseminated forms (primarily in immunocompromised patients) may be seen from direct inoculation or through hematologic seeding.

The extremities are usually involved with the initial lesion at the site of injury from a thorn, barb, pine needle, or wire. After approximately 1 to 10 weeks, a localized red, purple, or pink papule develops, often resembling an insect bite. The papule evolves into one or more nodules that form painless chronic ulcers with a nonpurulent, clear discharge. In the lymphocutaneous form, the nodules will progress proximally along lymphatic tracts and blood vessels. Many strains do not grow at temperatures above 35°C, decreasing their ability to spread and commonly resulting in a localized lesion. It is not communicable from person to person though it may be acquired through exposure to infected animals, with cats being the most infectious.

Wearing gloves and long sleeves while working in the outdoors and avoidance of skin contact with sphagnum moss are the mainstays of prevention. Confirmatory diagnosis is made with biopsy or culture.

Management and Disposition

Treatment with oral itraconazole for 3 to 6 months, or alternatively potassium iodide or terbinafine, is effective with complete recovery in cutaneous and lymphocutaneous forms. Variable response to treatment, often initially with amphotericin B, is seen in patients with severe disease or systemic involvement.

Pearls

1. This diagnosis should be considered when a cutaneous lesion is found on a patient involved with landscaping, rose gardening, Christmas tree farming, berry picking, baling of hay, and in veterinarians.
2. Lesions typically are noted on the distal upper extremity and the patient may have already failed multiple treatments with antibacterial agents.
3. While first described in the southern and central United States, it is most common in Mexico, Central/South America, Japan, Australia, and Africa. In Peru, the incidence is approximately 1 in 1000 people.
4. Sporotrichosis is easily confused with leishmaniasis, and the two illnesses coexist in many locations. Lymphatic spread is more characteristic of sporotrichosis, but possible with either. Other diseases or causes on the differential include nocardia, tularemia, and mycobacteria.



FIGURE 21.87 ■ **Sporotrichosis.** Typical sporotrichoid spread up the arm from an inoculation of the hand. Note the ulcers that resemble pyoderma gangrenosum in this Panamanian child. (Photo contributor: Richard P. Usatine, MD. Used with permission. From Usatine RP, Smith MA, Mayeaux EJ, Chumley HS. The Color Atlas of Family Medicine. 2nd ed. McGraw-Hill; 2013: Fig. 174-13.)



FIGURE 21.88 ■ **Sporotrichosis.** Typical lymphatic spread of sporotrichosis along the lateral aspect of the foot in a 30-year-old resident of the Peruvian Amazon. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)

Clinical Summary

Tuberculosis (TB) is a chronic bacterial infection spread from human to human through respiratory droplets containing M tuberculosis, an acid-fast bacillus. Upon inhalation, the organisms are transported to regional lymph nodes where the immune system forms granulomas or "tubercles." Most people undergo complete healing following exposure with only a positive PPD. However, it may lie dormant as latent disease until the immune system is suppressed, leading to organism release. TB occurs worldwide, but is more common in impoverished nations.

The organisms are dependent on high oxygen content and are typically found in the upper lobe or superior segment of the lower lobes of the lungs. Though it is primarily a respiratory illness, 15% will exhibit extrapulmonary manifestations involving the adrenal glands, long bones, vertebrae, GI tract, GU tract, skin, lymph nodes, meninges, pericardium, or peritoneum. Patients with HIV infection have a much higher overall prevalence and rate of extrapulmonary disease. Common symptoms of active TB are fever, night sweats, malaise, weight loss, cough, hemoptysis, and pleuritic chest pain. Diagnosis is made by the PPD and chest x-ray, but is confirmed through AFB smears and sputum culture. The mainstay of diagnosis in resource-limited countries is the AFB smear.

Management and Disposition

Typical first-line chemotherapy consists of isoniazid, rifampin, pyrazinamide, and ethambutol for 2 months, followed by isoniazid and rifampin for 4 months. Patients in developed countries are usually admitted with respiratory isolation. Outpatient therapy is common in developing countries. After 2 weeks of treatment, the patient is usually no longer contagious. Poor long-term adherence is a major contributing factor to the development of multidrug-resistant strains. Directly observed therapy (DOT) where patients are observed taking the medication may be required to ensure compliance and is routine in most countries.

Pearls

1. Any immunocompromised person with pulmonary symptoms should be placed in respiratory isolation until TB is excluded.
2. "Scrofula" refers to cervical lymph node involvement of TB, while vertebral involvement is called "Pott disease."
3. Isoniazid combined with rifapentine for treatment of latent TB, given weekly for 12 weeks, is now an alternative to the 9-month regimen of daily isoniazid.
4. TB is the most important opportunistic infection in HIV patients in sub-Saharan Africa. The HIV epidemic has led to a massive increase in the incidence of TB in many countries.



FIGURE 21.89 ■ **Tuberculosis Adenopathy.** Bilateral cervical adenopathy (scrofula) in a Peruvian child with documented TB. Pediatric cervical adenopathy in highly endemic areas is strongly suggestive, and the most common form, of extrapulmonary TB. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)



FIGURE 21.90 ■ **Tuberculosis Scarring.** Chronic discharging sinuses with extensive scar formation in a Peruvian man with extrapulmonary tuberculosis. (Photo contributors: Seth W. Wright, MD, and Universidad Peruana Cayetano Heredia, Lima, Peru.)



FIGURE 21.91 ■ **Tuberculosis Cold Abscess.** Cold abscess on the back of a Haitian woman with untreated Pott disease. (Photo contributors: Seth W. Wright, MD.)



FIGURE 21.92 ■ **Tuberculosis Pott Disease.** Severe spinal deformity ("gibbus") in an 8-year-old Ugandan boy with Pott disease. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.93 ■ **Abdominal Tuberculosis.** Severe wasting and ascites in a Ugandan boy with abdominal tuberculosis. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.94 ■ **Clubbing.** Clubbing of the fingers in a Ugandan man with longstanding pulmonary tuberculosis. (Photo contributor: Seth W. Wright, MD.)



FIGURE 21.95 ■ **Pulmonary Tuberculosis.** Severe wasting in a Sudanese soldier with smear positive pulmonary tuberculosis. His initial weight was 38 kg with a BMI of 11. His weight increased to 52 kg and BMI to 15 by the end of the 2-month intensive phase of treatment. (Photo contributor: Seth W. Wright, MD.)

TUNGIASIS

Clinical Summary

Tungiasis is an ectoparasitic condition caused by the cutaneous penetration of a Tunga penetrans flea. These tiny fleas are commonly found in sandy beaches and areas with warm, dry soil. Risk factors for infection include household dirt floors, lack of closed footwear, and the presence of farm animals. In some poverty-stricken regions, the prevalence of tungiasis approaches 50%. Tungiasis is also common in returned travelers, usually following beach vacations.

The female flea burrows into the skin of a host, feeds on blood, matures, lays eggs, and eventually dies in the cutaneous tissue. The rear of the flea remains visible as a black spot and serves for respiration and excretion of feces. A pronounced inflammatory response is seen leading to characteristic lesions. The vast majority of lesions are seen on the feet with the area at the rim of the toenails being the preferred site.

Early stage lesions demonstrate erythema surrounding a 0.5- to 2.0-mm black dot. A yellowish-white halo then develops around the dot with increasing erythema, pain, and pruritus. Later stage lesions develop a brownish coloration with crusting and desquamation. Secondary bacterial infection is common. Tungiasis is usually a clinical diagnosis; a small central dot representing the flea is an important clue. A biopsy will show the flea, but is not required for diagnosis.



FIGURE 21.96 ■ **Tungiasis.** Acute tungiasis lesion from an adult flea seen on the foot of a 30-year-old American aid worker in the Democratic Republic of Congo. Lesions are more obvious in patients who do not habitually walk barefoot. (Photo contributor: Seth W. Wright, MD.)

Management and Disposition

No oral drug has been proven to be effective against an embedded flea. Occlusive ointments will kill the flea, but does not aid in removal. The flea can be removed with excision or gently with a needle. Untreated, the flea will eventually die after 4 to 6 weeks with subsequent sloughing of the flea and eventual resolution of the inflammatory lesion. Antibiotics are indicated for secondary infection.

Pearls

1. Tungiasis is known by numerous local names including sand fleas, jiggers, bicho de pie, nigua, pigue, suthi, and chigoe.
2. The causative flea is native to the West Indies and was first reported in crewman who sailed with Christopher Columbus. They are now widely found throughout Latin America, Africa, and Asia.
3. The initial stage of penetration is manifested by a stinging sensation followed by itching that is surprisingly described as being pleasant.
4. Numerous cases of tetanus and gangrene have been reported in patients with secondarily infected T penetrans lesions.



FIGURE 21.97 ■ **Tungiasis.** Chronic lesions in a Kenyan man. Crusted scales are common in those with chronic infections and thicker skin. (Photo contributor: Larry Stack, MD.)

Chapter 22

AIRWAY

Steven J. White
Richard M. Levitan
Kevin High
Lawrence B. Stack



Glottic Opening. Standard direct laryngoscopy reveals the glottic opening in this cadaver. A Mac 4 blade is used like a Miller blade in this instance as the epiglottis lies anterior to the blade tip. (Photo contributor: Lawrence B. Stack, MD)

MAXIM: Endotracheal intubation is not always the best initial intervention for respiratory failure.

Clinical Summary

Some patients in respiratory distress may benefit from other interventions, short of intubation. Patients with flash pulmonary edema may have dramatic improvement with intravenous nitroglycerin, intravenous furosemide, and BiPAP (Bi-Level Positive Airway Pressure) ventilation (see Fig. 22.1). Patients with airway narrowing (edema, neoplasm, stricture, foreign body) can have significant decreased work of breathing by decreasing airway resistance to inspired gas using administration of helium-oxygen (HELIOX) mixture. HELIOX, usually as a 78%: 22% helium: oxygen mixture, is much less dense than either air or 100% oxygen by virtue of helium replacing nitrogen or oxygen, respectively. This lowers resistance to laminar flow by as much as 20% to 25% and the effects are immediate.

Pearl

1. Alternative ventilatory adjuncts include HELIOX, CPAP, BiPAP, and Vapotherm (see Fig. 22.2). These adjuncts may prevent the need for intubation in selected patients.



FIGURE 22.1 ■ **Bi-Level Positive Airway Pressure.** This patient with COPD rapidly improved with the application of BiPAP. (Photo contributor: Steven J. White, MD.)



FIGURE 22.2 ■ **Vapotherm.** Application of Vapotherm (high-flow warmed oxygen) rapidly improved this toddler with pneumonia and reactive airway disease. (Photo contributor: Lawrence B. Stack, MD.)

MAXIM: The most important initial airway intervention may be to ask for help.

Clinical Summary

Clinical scenarios where asking for help include:

1. Laryngeal injury/tracheal disruption, for whom a nonendoscopic intubation attempt can result in tracheal disruption and fatally lost airway (see Fig. 22.3).
2. Recent neck surgery, with pending loss of airway from an expanding hematoma; definitive and life-saving intervention in this case is to open up the recent incision and evacuate the hematoma.
3. Suspected epiglottitis, where an immediate operative tracheostomy or cricothyroidotomy may be required if intubation fails due to epiglottic edema.
4. Severe angioedema with tongue, oropharynx swelling where endotracheal or nasotracheal intubation may be obstructed (see Fig. 22.4).

Pearls

1. Emergency physicians are emergency airway experts, but know your limitations.
2. Do not let your ego get in the way of proper patient airway management and care.

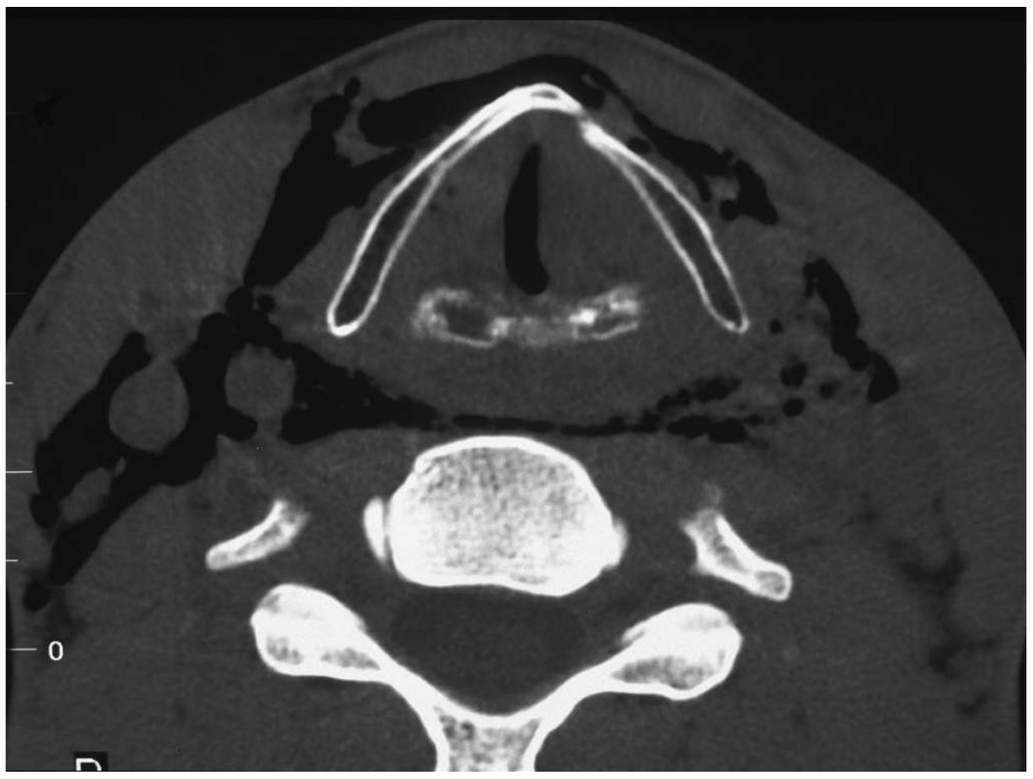


FIGURE 22.3 ■ **Laryngeal Fracture.** This 17-year-old male was kicked in the neck by a bull at a rodeo causing a laryngeal fracture. (Photo contributor: Rudy Kink, MD.)



FIGURE 22.4 ■ **Angioedema.** This patient with ACE-inhibitor angioedema and respiratory failure required awake fiber optic nasotracheal intubation by anesthesia. (Photo contributor: David Effron, MD.)

MAXIM: Most patients with airway/respiratory problems should be positioned for their comfort, not ours.

Clinical Summary

If mentating normally and physically able, a patient with airway difficulty will assume a position which optimizes their airway patency and gas exchange, usually sitting up and leaning forward. Such patients include those with incomplete airway obstruction, flash pulmonary edema, and massive airway bleeding from oropharyngeal trauma. Unfortunately, during preparation for intubation, such patients often are placed supine prematurely, increasing the patient's respiratory distress and anxiety, increasing the likelihood of spontaneous emesis and aspiration, and decreasing his ability to handle oropharyngeal bleeding or secretions. In these clinical situations, we should rethink the desire to immediately place a patient supine for endotracheal intubation. Intubation can be accomplished with the patient sitting up, by either of the following.

1. Altering the intubation technique (see Fig. 22.5).
2. Altering the intubator position relative to the patient (see Fig. 22.6).

Pearls

1. Keep patients in optimal position for spontaneous ventilation until they are sedated just prior to intubation; then, place them supine.
2. Consider intubating a patient sitting upright if you feel the supine position will compromise their ability to be ventilated.
3. Titrating ketamine in small doses (10 mg every 1-2 minutes) may facilitate awake intubation in a patient sitting upright.

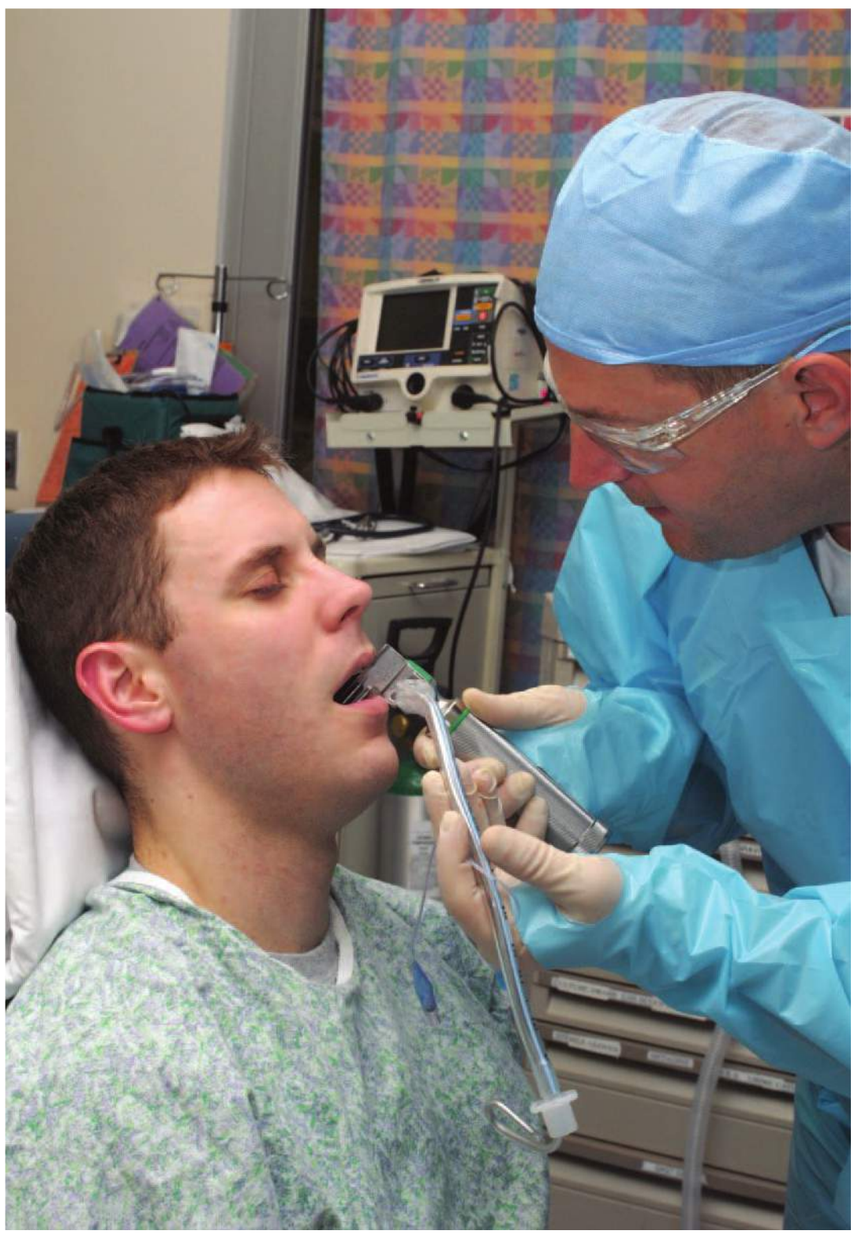


FIGURE 22.5 ■ **Intubation Facing the Sitting Patient or "Tomahawk Technique."** Holding the laryngoscope in the right hand and displacing the mandible forward and passage of the tube with the left hand is an alternative position to supine intubation. (Photo contributor: Lawrence B. Stack, MD.)

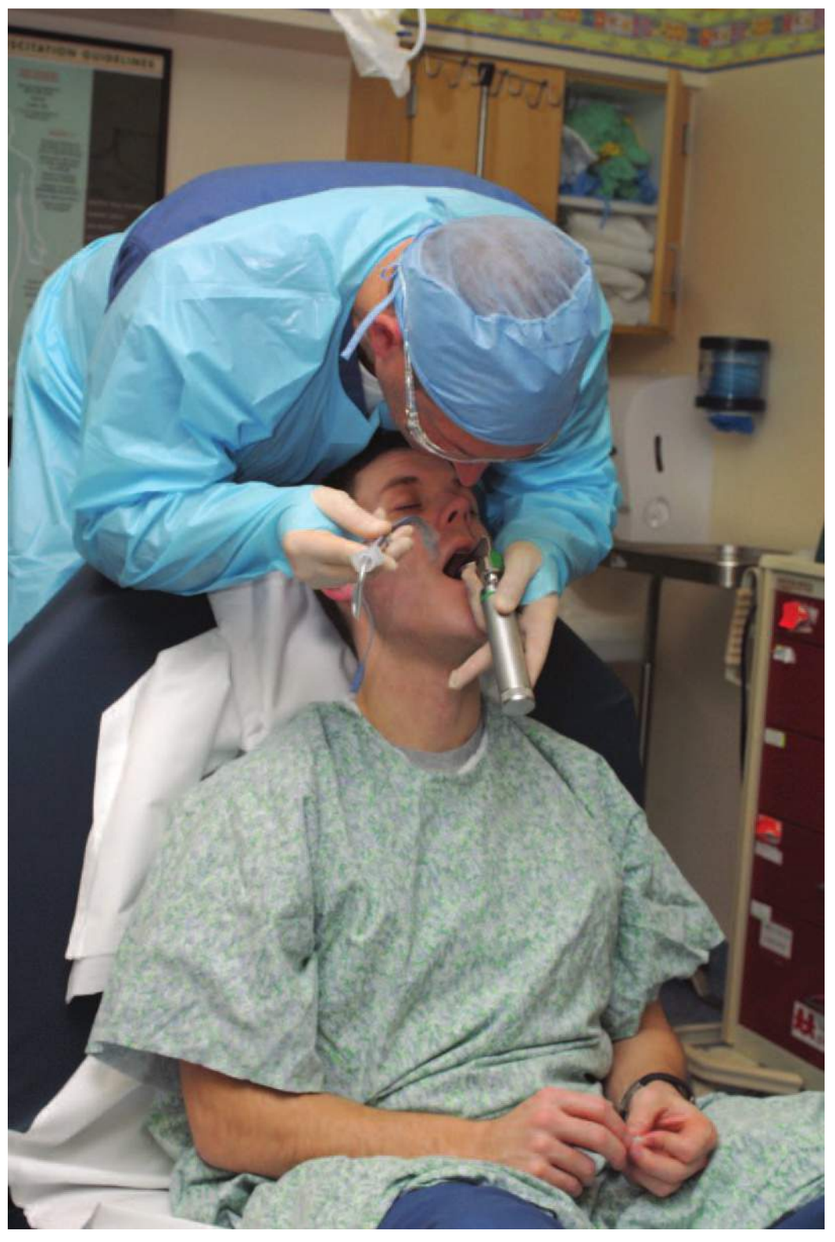


FIGURE 22.6 ■ **Intubation above the Sitting Patient.** Standing above a patient and intubating in the conventional manner is an alternative airway management position in a patient who may be difficult to ventilate in the supine position. An assistant may be necessary to hold the patient upright if sedated and/or paralyzed. (Photo contributor: Lawrence B. Stack, MD.)

MAXIM: Think before you paralyze.

Clinical Summary

Before committing to rapid-sequence induction (RSI) for direct laryngoscopy, consider the following:

1. Are any planned medications contraindicated?
2. Can rescue ventilation be achieved?
3. Is direct laryngoscopy possible? (See Fig. 22.7.)
4. What are my secondary and tertiary backup plans in the event of primary plan failure?
5. Are my equipment and personnel ready for RSI?

Pearls

1. A good backup plan to direct laryngoscopy should have at least one alternative intubation technique, one alternative ventilation technique, and one surgical airway technique.
2. Prepared equipment, correct patient position, proper drug dosing, having a backup plan, effective communication, and good technique will promote first-pass intubation (see Figs. 22.8 and 22.9).



FIGURE 22.8 ■ **Difficult Intubation.** This morbidly obese man with beard and no neck was successfully intubated after the third attempt. Ventilation was extremely difficult after paralysis. (Photo contributor: Lawrence E. Heiskell, MD.)



FIGURE 22.7 ■ **Wired Mandible.** This 33-year-old male with recent mandibular wire fixation of his mandibular fracture. A nasal or neck approach would be the only options for an emergency airway in this patient if no wire cutters were available. (Photo contributor: David Effron, MD.)



FIGURE 22.9 ■ **Difficult Intubation.** Anticipation of difficulty ventilating this patient if paralyzed prompted planning to optimize intubation success, which required three attempts. (Photo contributor: Lawrence E. Heiskell, MD.)

MAXIM: Patients will not die if they are not intubated; they will die if their lungs are not ventilated and their blood is not oxygenated.

Clinical Summary

The goal for airway management in any patient must be to maintain adequate ventilation and oxygenation. This does not necessarily mean intubation. Correct bag-valve-mask (BVM) ventilation/oxygenation technique is an underrated skill that will buy you time in the patient with a difficult airway. Proper steps for optimal two-person BVM ventilation include:

1. Positioning—ear-to-sternal notch alignment (when clinical scenario permits). Neck slightly flexed, head slightly extended (see Fig. 22.10).
2. Jaw thrust—displace mandible anteriorly with pressure from long, ring, and small fingers on mandible, not on soft tissues.
3. Mask compression—thumb and index fingers should apply firm pressure to face and nasal bridge (see Figs. 22.11 and 22.12).
4. Oral/nasal airways—may help maintain airway patency during BVM ventilation (see Fig. 22.13).
5. Use 7 mg/kg tidal volume, over 1 to 2 seconds at 12 breaths/min.



FIGURE 22.10 ■ **Optimal Ear-to-Sternal Notch Alignment.** Optimal position for ventilation and laryngoscopy occurs when the external auditory canal and the sternal notch are aligned in the horizontal plane (**B**) and the face is parallel to the ceiling (**C**). This position optimizes airway patency and ventilation mechanics. (Photo contributor: Lawrence B. Stack, MD.)

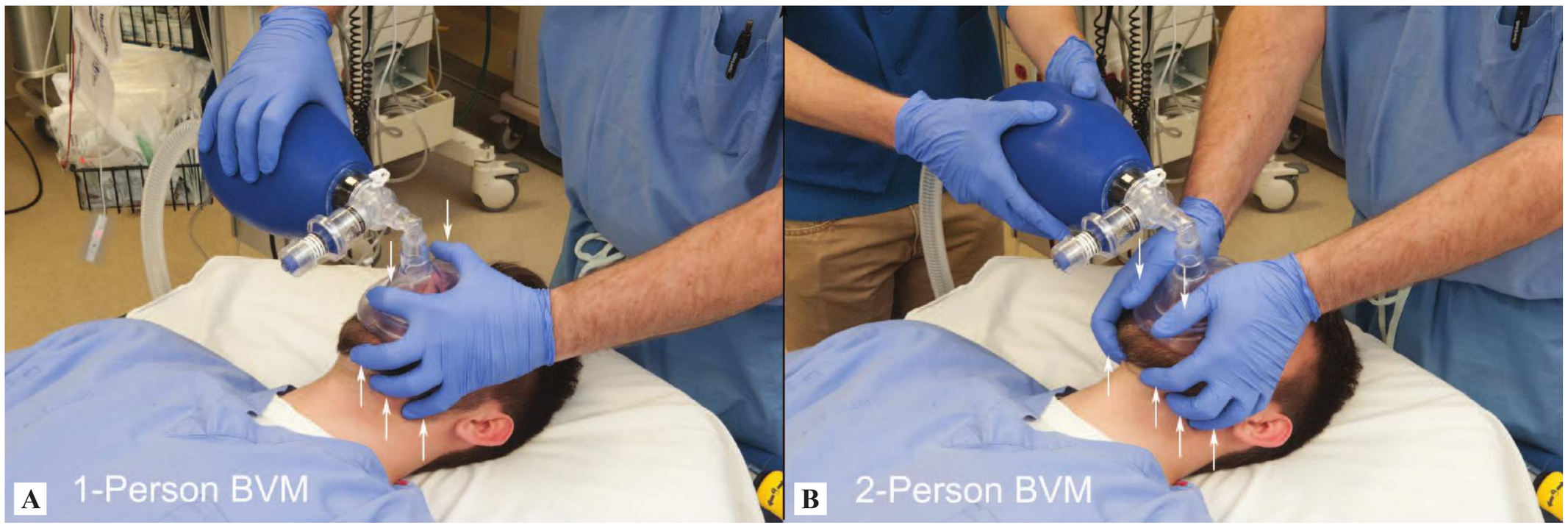


FIGURE 22.11 ■ **Bag-Valve-Mask Ventilation.** Correct positioning and forces during bag-valve-mask ventilation are demonstrated for the one- and two-person (thumbs) techniques. Upward force on the mandible, not soft tissue, is key to effective jaw-thrust technique. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. Be an expert at BMV ventilation.
2. Ear-to-sternal notch positioning is most beneficial in obese patients and those with obstructive sleep apnea.
3. Keeping dentures in place facilitates BVM, while removing them facilitates orotracheal intubation.
4. Midface and mandibular disfiguration from whatever cause will interfere with optimal BVM ventilation.
5. Consider LMA, King LT, or Air-Q if unable to intubate or obtain adequate seal for BVM ventilation.

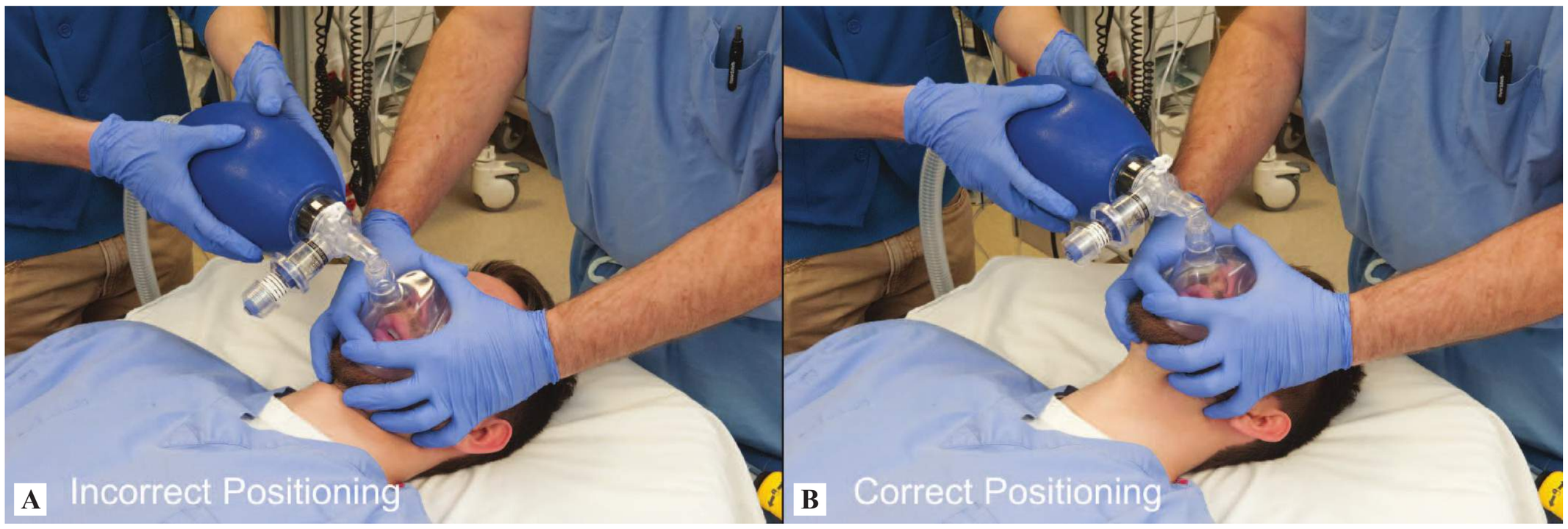


FIGURE 22.12 ■ **Bag-Valve-Mask Ventilation.** Incorrect (**A**) and correct (**B**) positioning and forces during bag-valve-mask ventilation are demonstrated for the two-person techniques. Lack of jaw thrust during BVM will allow the tongue to block air passage. (Photo contributor: Lawrence B. Stack, MD.)

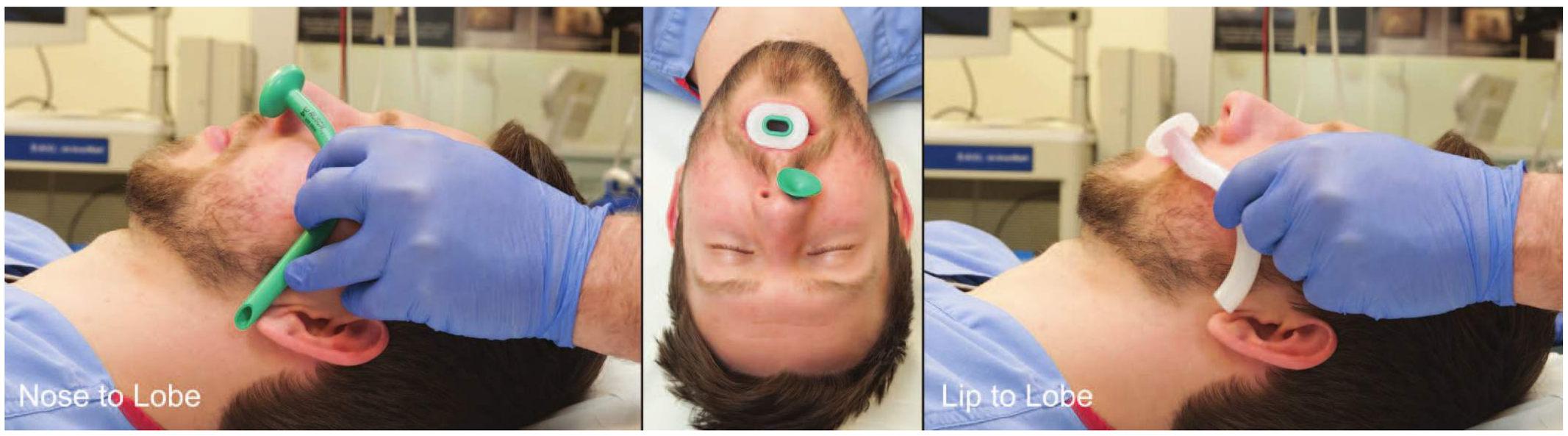


FIGURE 22.13 ■ **Nasal and Oral Airways.** Appropriately sized and placed nasal and oral airways maximize upper airway patency during bag-valve-mask ventilation. (Photo contributor: Lawrence B. Stack, MD.)

Patient Positioning

The optimal position to maximize laryngoscopic visualization of the larynx is:

1. The head extended.
2. The neck flexed.
3. The base of the ear is aligned with the sternal notch (see Fig. 22.10).
4. The facial plane is horizontal, parallel to the ceiling (see Fig. 22.10).
5. The facial plane should be positioned between the laryngoscopist's xiphoid process and umbilicus (see Fig. 22.14). This position most closely replicates in a supine posture which the patient would assume sitting up. For very large individuals or those with significant morbid obesity, this may require creation of a textile ramp of blankets, sheets, or towels to raise the head and shoulders to proper elevation and alignment.

Preoxygenation and Passive Apneic Oxygenation

Preoxygenation, when possible, should be used to create an oxygen surplus in the blood and tissue which permits a period of apnea to occur without arterial oxygen desaturation. This process hopefully begins in the prehospital setting with the placement of a nonrebreather mask with high-flow oxygen on the patient. This should continue in the ED in the event intubation is required. In addition, a nasal cannula on high flow (15 L/min) should also be placed when the patient arrives in the ED if there is a high likelihood of intubation. High-flow oxygen delivered by nasal cannula provides passive apneic oxygenation during the apneic phase of endotracheal intubation.

Technique

Stylet Shape

A stylet should be used with all oral intubations in which a laryngoscope is employed. The laryngoscopist will typically



FIGURE 22.14 ■ **Correct Intubating Height.** Optimal height for intubation is typically when the plane of the patient's face is between the intubator's umbilicus and xiphoid process. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.15 ■ **Preoxygenation and Passive Oxygenation.** In preparation for intubation, simultaneously placing an NRBM (preoxygenation before induction) and nasal cannula (passive oxygenation after paralysis) at 15 L/min will help prolong tissue oxygenation during the apneic phase of intubation. (Photo contributor: Lawrence B. Stack, MD.)

fashion an inexpensive stylet, essentially a malleable wire, into a shape of their preference, to permit control of the distal endotracheal tube (ETT) tip. One should take care not to place too much bend or curvature to the stylet, because such a configuration can actually impair the glottic view and control of the ETT tip. Optimal stylet configuration has been described as "straight to the cuff" and then a gentle anterior bend of 30 degrees.

Even with a correct bend on the stylet, it may be difficult to advance the styletted ETT into the trachea. The tip of the left-sided beveled tube can impact and catch on the anterior tracheal cartilages, preventing advancement. This can often be remedied with a generous clockwise or rightward rotation of tube and stylet, which acts to rotate the bevel anteriorly and depress the ETT tip.

Optimizing Biomechanics

Holding the laryngoscope handle and blade where the proximal end of the blade is in the palm of the hand creates a natural extension of the forearm and provides fine control of the blade tip. The left thumb should run parallel to the axis of the handle. Effort mechanics are more efficient with this grip. Placing the elbow close to the body requires less effort and more

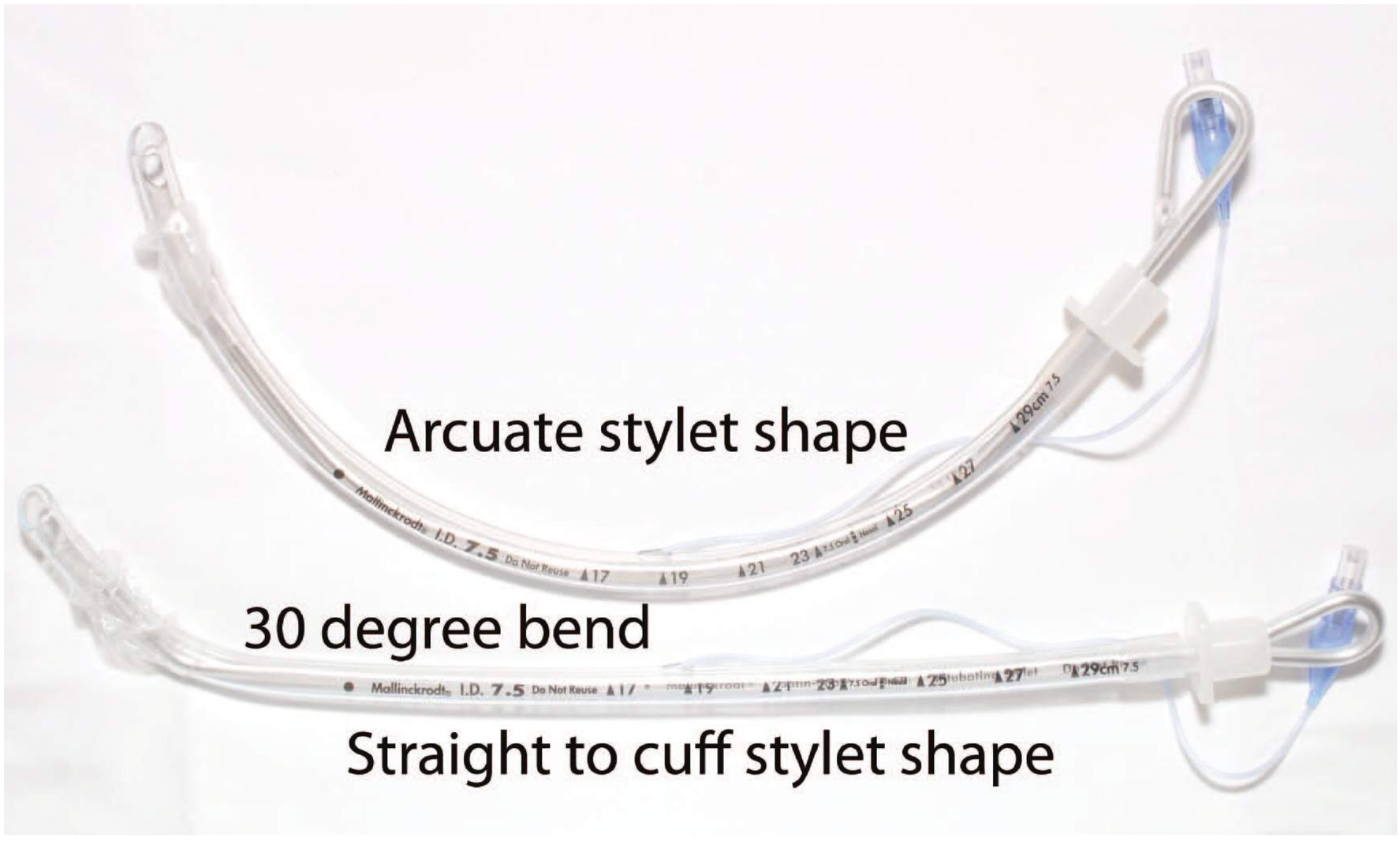


FIGURE 22.16 ■ **Stylet Shape.** Straight to cuff with a 30-degree bend is the optimal stylet shape as this offers the most ETT tip control and view of the glottic opening. (Photo contributor: Lawrence B. Stack, MD.)

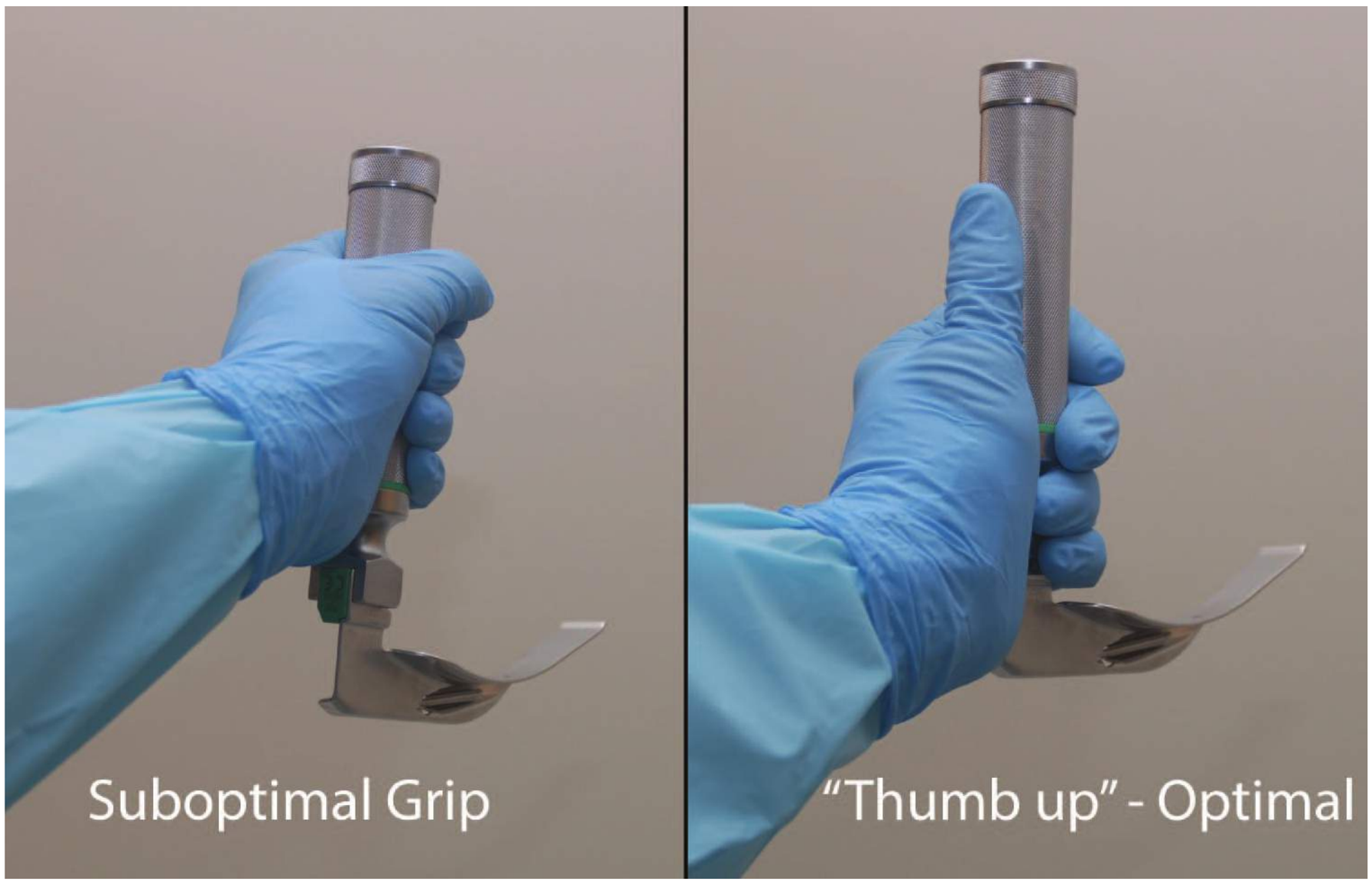


FIGURE 22.17 ■ **Laryngoscope Grip.** The laryngoscope should be gripped as low as possible and the thumb extended. This makes a natural extension of the forearm. (Photo contributor: Lawrence B. Stack, MD.)

mechanical control than if the elbow abducted from the body. A memory aid for these concepts are "Thumb up-Wing in."

Mouth Opening Techniques

The scissor technique places the laryngoscopist's right thumb on the occlusive surface of the patient's lateral mandibular teeth or gum and long or index finger on the maxillary teeth or gum. A scissoring motion opens the mouth allowing blade insertion. The fingers should be removed once the blade is adequately inserted. An alternative technique is to place the laryngoscopist's right thumb on the nasal bridge and long finger between the mental protuberance and alveolar margin. The thumb and long finger are moved away from each other, causing the mouth to open.

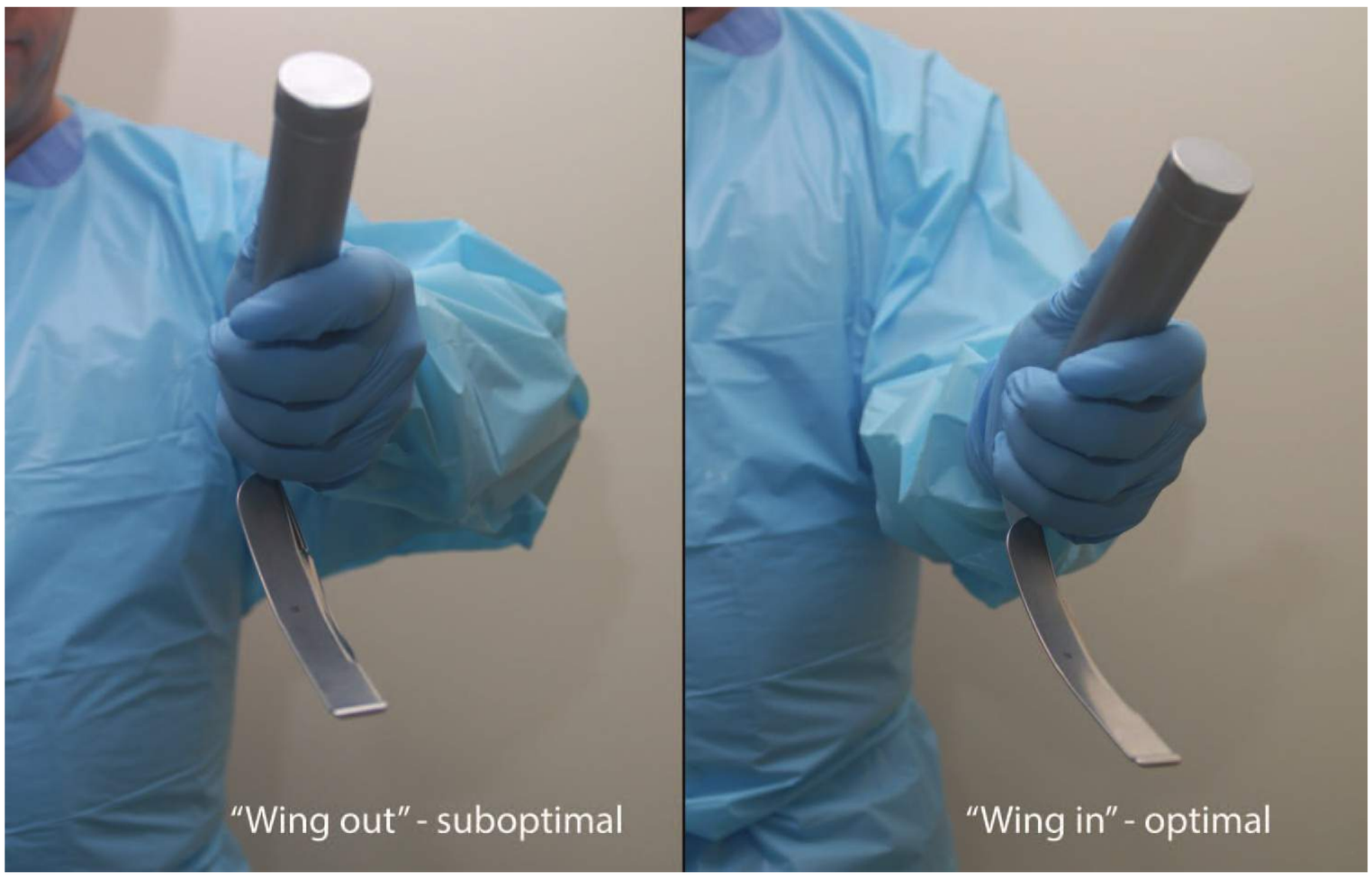


FIGURE 22.18 ■ **Laryngoscope Biomechanics.** Keeping the elbow close to the body requires less effort and more control during laryngoscopy. (Photo contributor: Lawrence B. Stack, MD.)

Blade Insertion

The blade is initially inserted just to the right of midline with the handle pointed toward the patient's feet. As the tip of the blade advances downward along the tongue base and into vallecula, the tongue is swept slightly to the left to increase the amount of workspace. When fully inserted into the vallecula, the angle of the handle is now approximately 40 degrees from horizontal with lifting forces directed upward and forward.

Optimal Epiglottoscopy

The key to using the laryngoscope to optimally visualize the glottic opening is to first visualize and control the epiglottis, a relatively fixed anterior structure. Pulmonary secretions, blood, and/or vomitus that pools in the posterior pharynx may obscure the posterior laryngeal structures, dependent with gravity. The epiglottis itself may be camouflaged in this pool of muck (see Fig. 22.20). One should carefully insert the laryngoscope blade with a goal of adequately visualizing and controlling the epiglottis. Failure to do so risks inserting the laryngoscope blade too deeply, and often results in displacing the larynx anteriorly to expose the esophagus, which, as a consequence of anterior-ward tension on the laryngoscope blade, may then look like a glottic opening begging for a tube. By carefully controlling the epiglottis, the intubating clinician will be able to locate important airway landmarks. Be careful to displace the tongue to the left side as you insert the blade. If the bulk of the tongue wraps around the blade, it can both impair your view and impede insertion of the ETT.

Progressive visualization of laryngeal structures is predictable:

1. Uvula and posterior pharynx (see Fig. 22.21A)
2. Epiglottis (see Fig. 22.21B)
3. Posterior arytenoids cartilages and interarytenoid notch (see Fig. 22.21C)
4. Glottic opening (see Fig. 22.21D)
5. Vocal cords (see Fig. 22.21E)
6. Esophagus (see Fig. 22.21F)

Bimanual Laryngoscopy

Even with well-performed laryngoscopy, adequate visualization of the laryngeal structures may be difficult. Bimanual laryngoscopy (external laryngeal manipulation by the operator), where the laryngoscopist performs laryngoscopy while simultaneously manipulating the external larynx, facilitates optimal visualization. This eliminates any delay or miscommunication between an assistant and laryngoscopist. Once optimal position is found, an assistant can maintain that position. Alternatively, an assistant's hand, placed on the laryngeal structures, guided by the laryngoscopist's hand can maintain optimal position after the laryngoscopist removes his hand from the assistant's hand.

Lip Commissure Retraction

The lip commissure is the junction of the upper and lower lips. Lateral retraction of the patient's right lip commissure by an assistant facilitates visualization of oral structures and insertion of the ETT into the oral cavity. This creates an optimal workspace for tube delivery. After glottis visualization is achieved, an assistant places their right index finger in the patient's right commissure and thumb against the maxilla. The index finger retracts the commissure laterally while applying an opposing force with the thumb to avoid excessive movement of the head. A memory aid of this concept is "pull out commissure, push on maxilla," or "POC-POM." Retraction of the lip commissure aids in keeping the ETT from



FIGURE 22.19 ■ **Mouth Opening Techniques.** The traditional "scissor" technique (**A**) and the "clean hand" technique (**B**) for opening the mouth during endotracheal intubation. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.20 ■ **Epiglottic Camouflage.** Vomitus, blood, and pulmonary secretions may pool in the posterior pharynx and obscure the gravity-dependent epiglottis. Elevating anterior laryngeal structures will expose the epiglottis. (Photo contributor: Lawrence B. Stack, MD.)

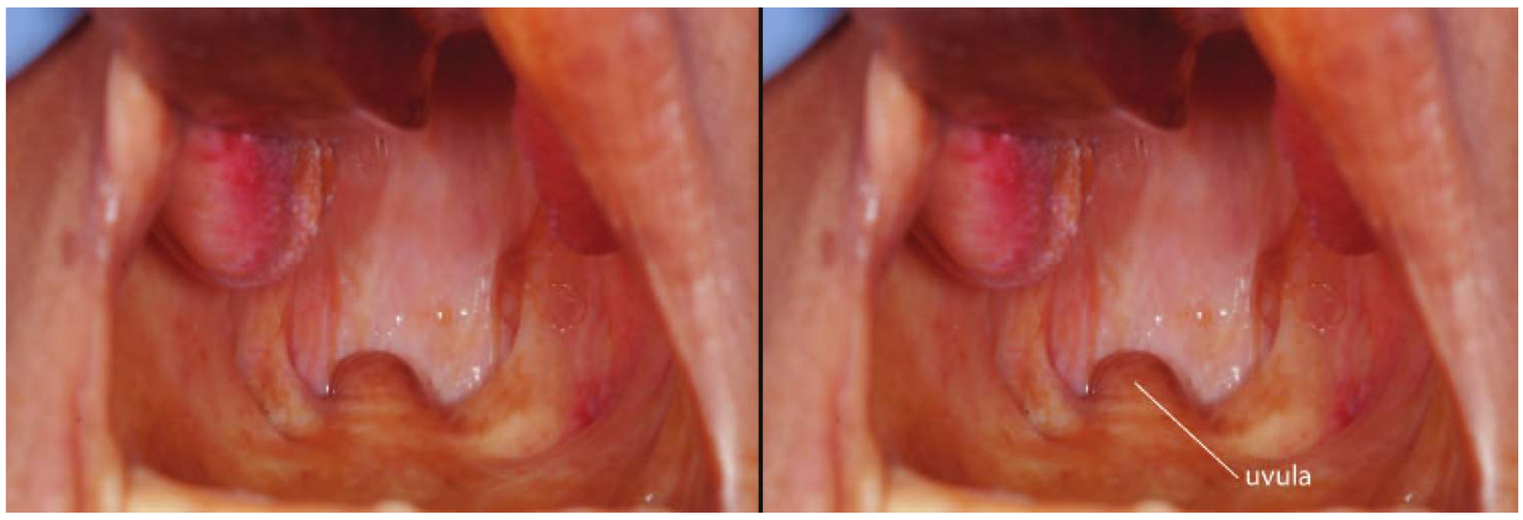


FIGURE 22.21A ■ **Progressive Visualization—Uvula.** The uvula and posterior pharynx are the first structures visualized during correct laryngoscopic technique. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.21B ■ **Progressive Visualization—Epiglottis.** The key to first-pass intubation is finding and identifying the epiglottis laryngoscopy. Following epiglottis will take one to the glottic opening. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.21C ■ **Progressive Visualization—Interarytenoid Notch.** The interarytenoid notch is a vertical cleft between the posterior cartilages. Above the notch lies the glottic opening. Below the notch is the esophagus. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.21D ■ **Progressive Visualization—Glottic Opening.** The posterior glottic opening is seen before the vocal cords are visualized. (Photo contributor: Lawrence B. Stack, MD.)

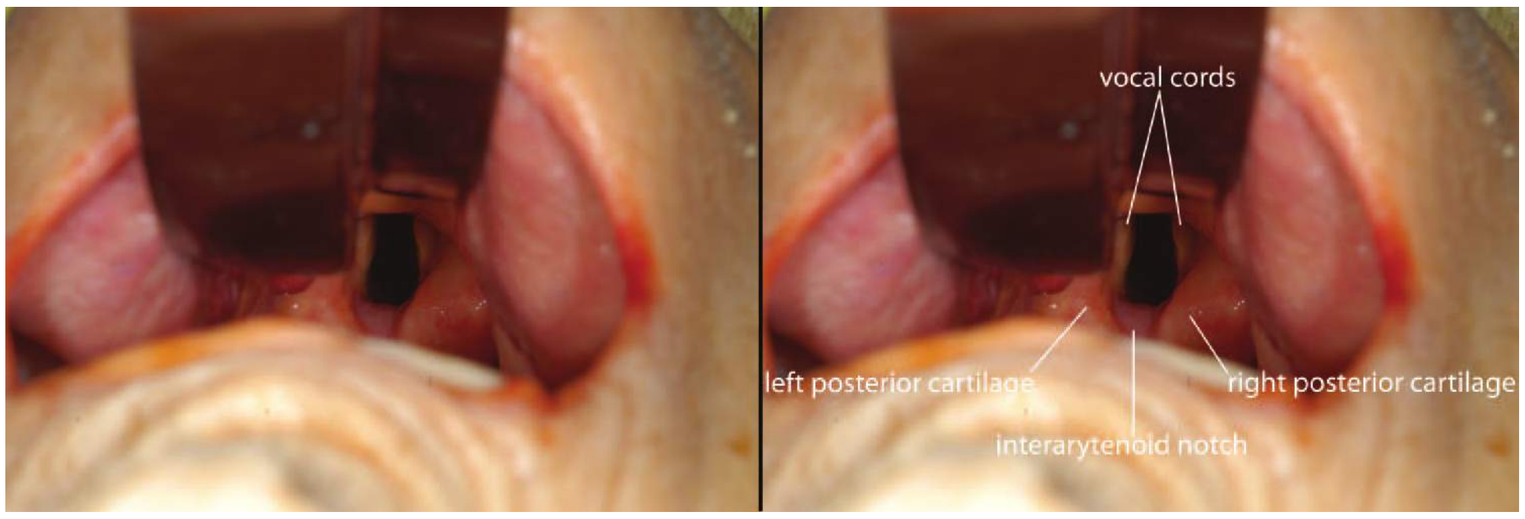


FIGURE 22.21E ■ **Progressive Visualization During Laryngoscopy—Vocal Cords.** The vocal cords have a distinct white appearance. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.21F ■ **Progressive Visualization During Laryngoscopy—Esophagus.** The esophagus lies directly below the interarytenoid notch. (Photo contributor: Lawrence B. Stack, MD.)

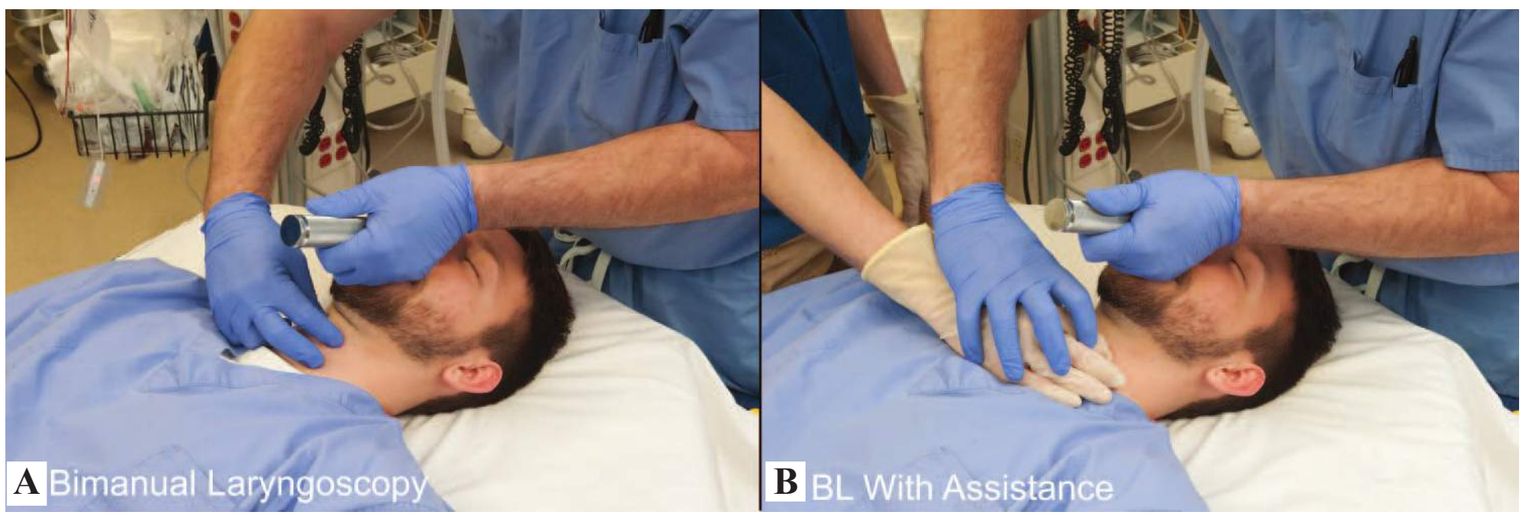


FIGURE 22.22 ■ **Bimanual Laryngoscopy.** Laryngoscopist uses right hand to manipulate laryngeal structures for optimal visualization during laryngoscopy. Once optimal position is identified, an assistant maintains it during intubation. (Photo contributor: Lawrence B. Stack, MD.)

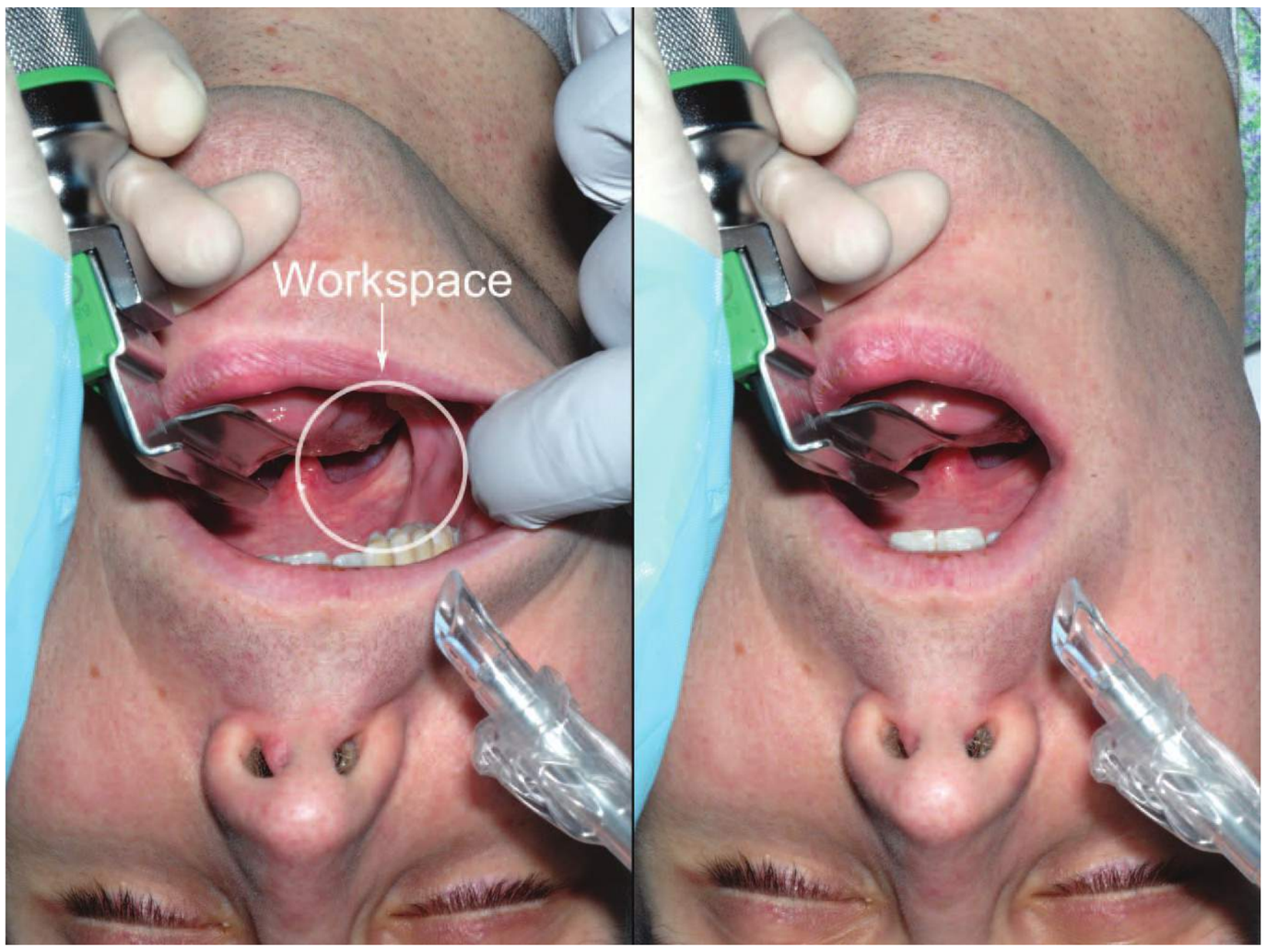


FIGURE 22.23 ■ **Commissure Retraction.** An assistant retracts the right commissure to create a generous "workspace" to visualize and deliver the endotracheal tube. (Photo contributor: Lawrence B. Stack, MD.)

blocking the view of the glottic opening during advancement of the ETT. Using the retracted lip commissure as a fulcrum or "toggle" gives optimal control of the ETT tip, especially when the stylet is in the "straight to cuff" shape. Retraction may also facilitate intubation by allowing clockwise rotation of the tube if the ETT tip becomes hung up on the proximal tracheal cartilages. A memory aid of this concept is "right on rings" or rotate the tube to the right or clockwise if it hangs up on the tracheal rings.

Endotracheal Tube Handling

The thumb, index, and long fingers provide the fine dexterity for the hand, and should be used to control the tube during endotracheal intubation. The ETT should be held like a "throwing dart" between the thumb, index, and long fingers midway along the tube for precise control during the procedure. A trained assistant should hand the tube to the laryngoscopist in this manner.

Endotracheal Tube Delivery

Obtain the best glottic view and have an assistant provide right lip commissure retraction. Introduce the ETT at the 3-o'clock position of the mouth. Advance the tube posteriorly in a manner that will not obstruct the direct view of the "target." Advance the tube through the glottis. The last thing you should see is



FIGURE 22.24 ■ **Pull on Commissure-Push on Maxilla.** When pulling on the commissure with the index finger, simultaneous pushing on the maxilla with the thumb will minimize head movement during intubation. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.25 ■ **Correct ETT Grip.** Using the thumb, index, and long fingers to grip the mid-endotracheal tube like a "throwing dart" will provide the most precise control during tube deliver. (Photo contributor: Lawrence B. Stack, MD.)

the tube going through cords. If resistance is met, it is most likely due to the bevel being caught on a tracheal ring. Turn the tube to the right or clockwise and attempt to advance the tube. Advance the tube to 22 cm at the lip, inflate the cuff, and confirm placement. Secure the tube in place (see Table 22.1).

Pearls

1. The key to first-pass intubation success is identification and control of the epiglottis during direct laryngoscopy.
2. The glottic opening lies between the epiglottis and interarytenoid notch. Identification and passing the tube between these structures will improve first-pass success.
3. While most intubations are performed without difficulty, if a difficult airway is anticipated and there is time to prepare, optimize the patient's ear-to sternal notch position.
4. Approximately 250 mL/min of oxygen will move from the alveoli into the blood during apnea, which may extend the period of adequate arterial oxygenation during intubation.



FIGURE 22.26 ■ **Incorrect ETT Grip.** These ETT grips will provide a powerful force on the tube but will lack precise handling of the tube, critical to tube delivery. (Photo contributor: Lawrence B. Stack, MD.)

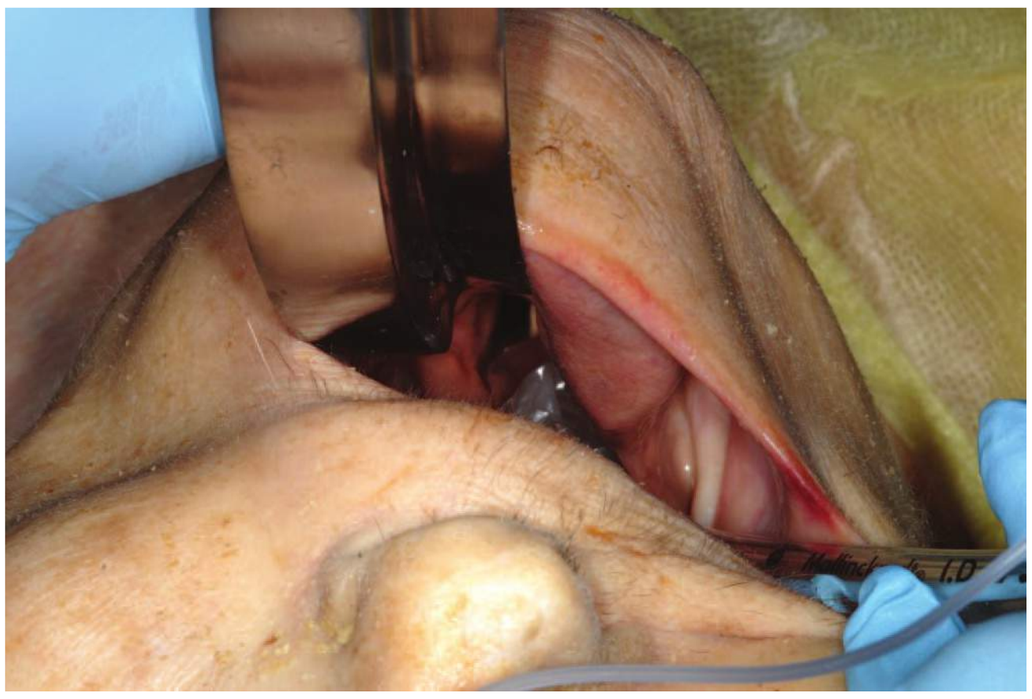


FIGURE 22.27 ■ **Tube Delivery.** Obtain the best glottic view. Introduce the ETT at the 3-o'clock position of the mouth. Advance the tube posteriorly in a manner not to obstruct the view of the "target." Advance the tube through the glottis. (Photo contributor: Lawrence B. Stack, MD.)

TABLE 22.1 ■ ENDOTRACHEAL TUBE (ETT) DELIVERY STEPS

- Obtain best possible glottic view
- Introduce ETT at 3-o'clock position of the mouth
- Advance ETT posteriorly to avoid obstruction of "target" view
- Advance ETT through glottis (last thing to see is ETT through cords)
- If resistance is met (bevel against rings), rotate tube clockwise, then advance
- Inflate cuff
- Confirm tube placement (ETCO2, breath sounds, O_2 saturation monitor)
- Secure tube

Technique

The Eschmann stylet, also known as a "gum elastic bougie" or simply "bougie," is a 60-cm long, flexible introducer, which is designed to assist intubation of the anterior larynx, especially those with an "epiglottis-only" view. The tip of the bougie has an anterior fixed flexion with an angle of 40 degrees to facilitate entering an anterior glottic opening. Using a laryngoscope, one obtains the best view possible of the glottic opening. In the case of limited glottic view, the bougie is inserted such that the tip is introduced just under the epiglottis and probes for the glottic opening. The intubator should feel a tactile "pop" as the bougie enters the trachea and he or she may also have a tactile sensation of "speed bumps" as the bougie is advanced and tracks across the tracheal cartilaginous rings. However, the more sensitive indicator of tracheal bougie position is the resistance encountered as the tip abuts against the carina, at approximately 27 to 30 cm. Should the bougie be inserted into the esophagus, no such endpoint is encountered.

While continuing to hold anterior traction with the laryngoscope, the laryngoscopist should direct an assistant to thread the ETT over the bougie again while maintaining anterior traction; the intubator should then advance the ETT over the bougie to the appropriate insertion depth. Resistance to tube advancement may indicate "arytenoid arrest" and can be remedied by rotating the tube counterclockwise 90 degrees followed by attempts to advance the ETT tip past the arytenoid cartilages.

A one-handed technique can be employed in which the ETT is preloaded onto the bougie, which is then curled in the right hand with approximately 20 cm of bougie protruding through the distal end. The laryngoscopist performs laryngoscopy as before, maintains anterior traction on the mandible, and inserts the bougie through the glottic opening as above. He or she then uncoils the ETT/bougie and advances the pair until resistance of the carina is met, confirming intratracheal position. The ETT is then advanced to the proper depth and the bougie is removed.

Use of the bougie does not have to be limited to the anticipated difficult airway. In fact, to gain proficiency, we recommend that practitioners use it as their routine stylet, as it has come to be used in the UK. The practitioner will then be better prepared for the unanticipated anterior larynx/limited view airway.



FIGURE 22.28 ■ **Bougie Tip Shape.** The bougie tip has a 40-degree bend to help facilitate entering the glottic opening when the glottis is not adequately visualized. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.29 ■ **Epiglottis-Only View.** In this view, despite optimal laryngoscopy, the epiglottis may be all that is seen. The bougie may be a reasonable alternative in this situation (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.30 ■ **Passing the Bougie.** The two-person technique for using the bougie. Standard DL is preformed and the bougie is used like a stylet, but hugging the underside of the epiglottis. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.31 ■ **Bougie under the Epiglottis.** Here the bougie is hugging the underside of the epiglottis. Tracheal rings can be felt by "tactile speed bumps," confirming correct placement. (Photo contributor: Lawrence B. Stack, MD.)

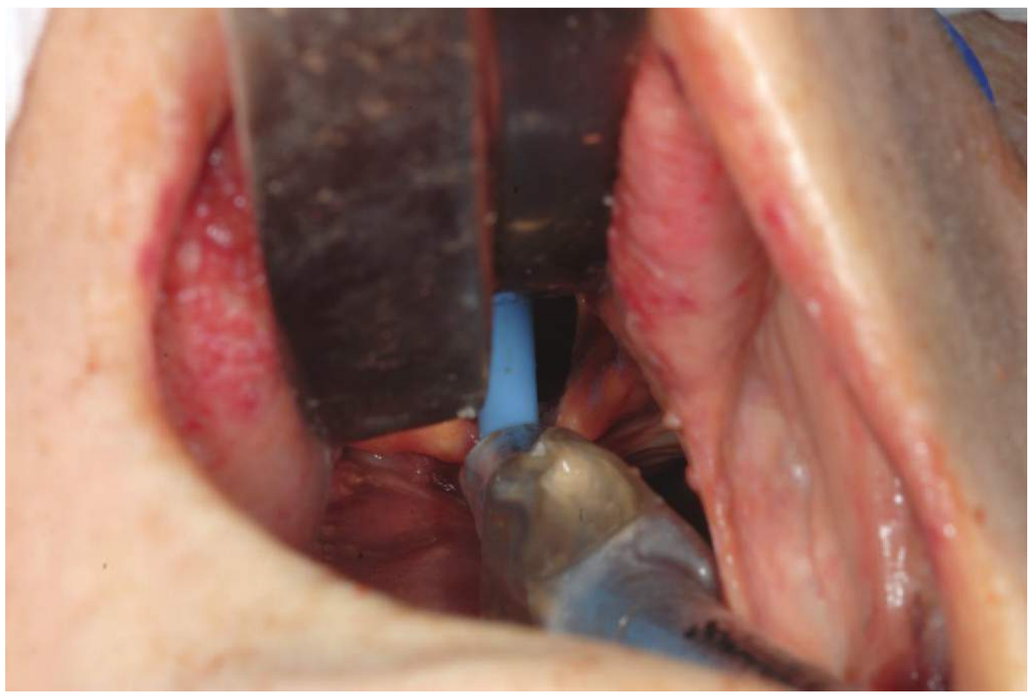


FIGURE 22.33 ■ **Tube Advancement Stall.** Stalling of tube advancement while using the bougie is most likely due to catching the right arytenoids cartilage with the bevel of the ETT. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.32 ■ **Tube over the Bougie.** An assistant places the bougie over the tube while direct laryngoscopy is maintained. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.34 ■ **Arytenoid "Escape."** Turning the tube counterclockwise, as shown here, will release the bevel of the ETT from the arytenoid cartilage and allow the tube to advance. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.35 ■ **One-Handed Bougie Configurations.** Holding the bougie in one of these configurations will allow one person to pass the bougie. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. The bougie is an excellent adjunct in the limited view or "epiglottis view" airway.
2. Bougie-assisted intubations is best deployed as a two-person technique.
3. Stall of ETT advancement when using the bougie is most frequently due to catching the bevel on the right arytenoid cartilage and can be relieved by rotating the tube counterclockwise.



FIGURE 22.36 ■ **One-Handed Bougie Technique.** Passing the bougie using the one-handed technique. See text for details. (Photo contributor: Lawrence B. Stack, MD.)

Equipment

The Glidescope is a video laryngoscope composed of an integrated blade/handle which houses a blade tip–mounted camera and light source, connected by cable to a video LCD display. The Glidescope affords an excellent view of the laryngeal structures with little anteriorly directed force. Use of proprietary stylet, conformed to angle of Glidescope blade, is recommended. A more portable version, suitable for out-of-hospital use, is marketed as the Glidescope Ranger.

Technique

The blade is inserted into the mouth, along the center of the tongue, and advanced to visualize the epiglottis and laryngeal structures. Little-to-no anterior force is required, and in contrast to standard laryngoscope, levering the blade is recommended to enhance the view. Once the tube is passed through the cords, the proprietary stylet is withdrawn 5 cm to allow further passage of the tube. The steep angle of Glidescope blade and stylet causes the tube to abut the anterior tracheal rings and arrest tube delivery in most patients. Withdrawing the stylet 5 cm and rotating the tube clockwise will allow complete tube delivery to an appropriate depth.



FIGURE 22.37 ■ **Glidescope.** The Glidescope video LDC display on a stand with blade (inset) and proprietary stylet (inset). (Photo contributor: Lawrence B. Stack, MD.)

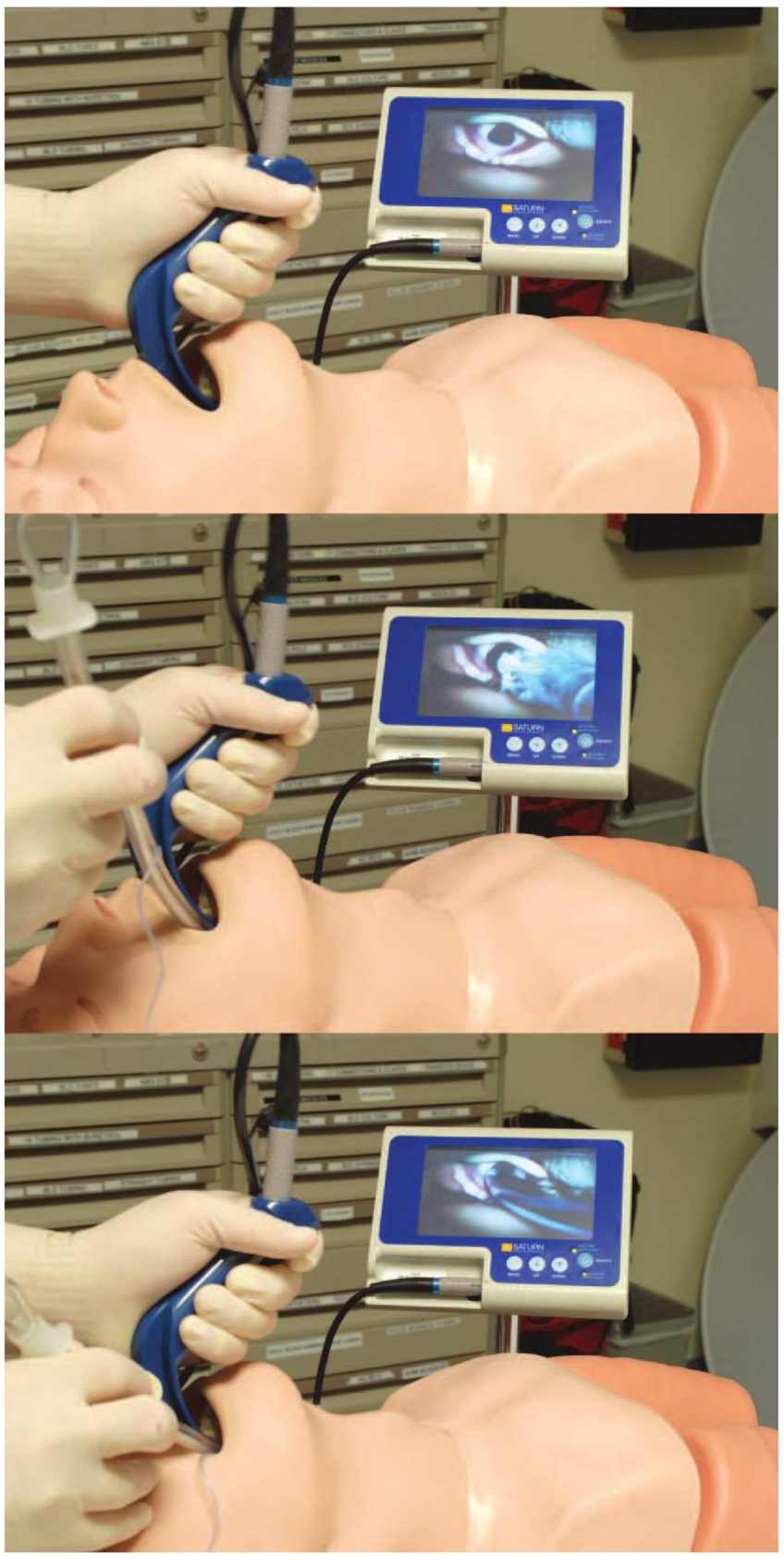


FIGURE 22.38 ■ **Glidescope in Use.** The Glidescope in action demonstrating progressive images of passage of an ETT. (Photo contributor: Lawrence B. Stack, MD.)

Equipment

The Storz C-MAC video laryngoscope system consists of stainless steel, reusable blades, which house a CMOS camera chip and high-output LED light, an interchangeable powered coupling, video screen, and mobile tower. Available blades are similar in size and shape to the Mac 2, Mac 3, Mac 4, Miller 0, Miller 1, and a D-blade designed for patients with difficult airway anatomy. The near-standard shape of the blade allows direct laryngoscopy for glottic visualization as well as video laryngoscopy.

Technique

The blade connected to the power coupling should be handled like a standard laryngoscope blade. It should be inserted just to the right of midline of the tongue with the handle pointing toward the feet. As the tip of the blade advances downward along the tongue base and into vallecula, the tongue is swept slightly to the left to increase the amount of workspace. When fully inserted into the vallecula, the angle of the handle is now approximately 40 degrees from horizontal with lifting forces directed upward and forward. At anytime, the laryngoscopist can switch from a standard direct laryngoscopy to a video laryngoscopy; however, once changed to video laryngoscopy, it is best not to switch back and forth from video laryngoscopy to direct laryngoscopy.



FIGURE 22.39 ■ **Storz C-MAC Tower.** Storz video laryngoscope system including tower and adult blades. (Photo contributor: Lawrence B. Stack, MD)

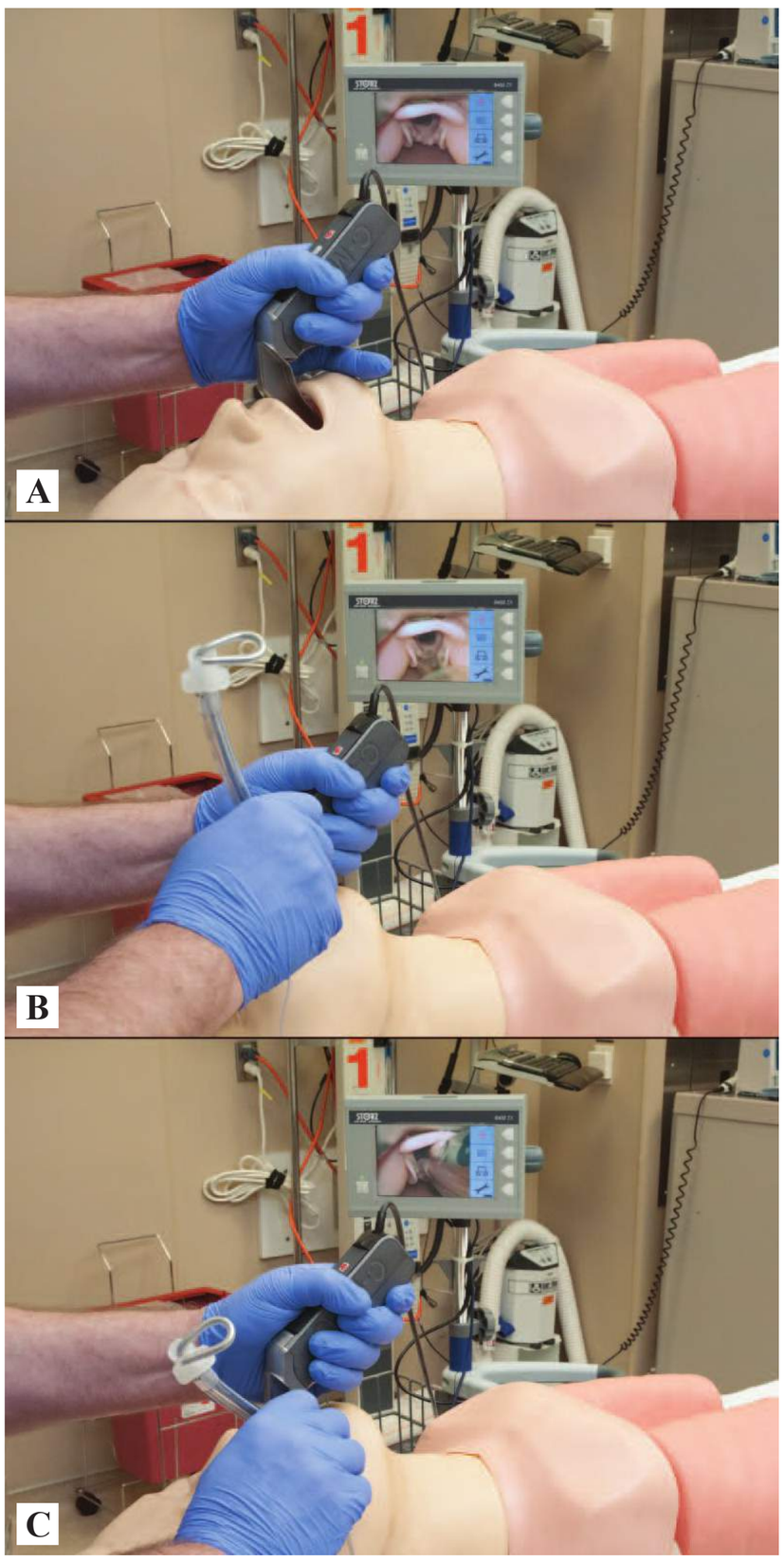


FIGURE 22.40 ■ **Storz C-MAC in Use.** The Storz C-MAC in action demonstrating progressive images of passage of an ETT. (Photo contributor: Lawrence B. Stack, MD.)

Equipment

The McGrath MAC enhanced direct laryngoscope is a combination of direct and video laryngoscope in a compact, battery-powered, device that uses disposable standard curved Macintosh blades and has an adjustable LED screen. It can be used in direct laryngoscopy and video laryngoscopic situations.

Technique

The disposable plastic Macintosh blade is placed onto the McGrath MAC EDL and the device is powered on. The blade is inserted just to the right of midline of the tongue with the handle pointing toward the feet. As the tip of the blade advances downward along the tongue base and into vallecula, the tongue is swept slightly to the left to increase the amount of workspace. When fully inserted into the vallecula, the angle of the handle is now approximately 40 degrees from horizontal with lifting forces directed upward and forward. At anytime, the laryngoscopist can switch from a standard direct laryngoscopy to a video laryngoscopy; however, once changed to video laryngoscopy, it is best not to switch back and forth from video laryngoscopy to direct laryngoscopy.

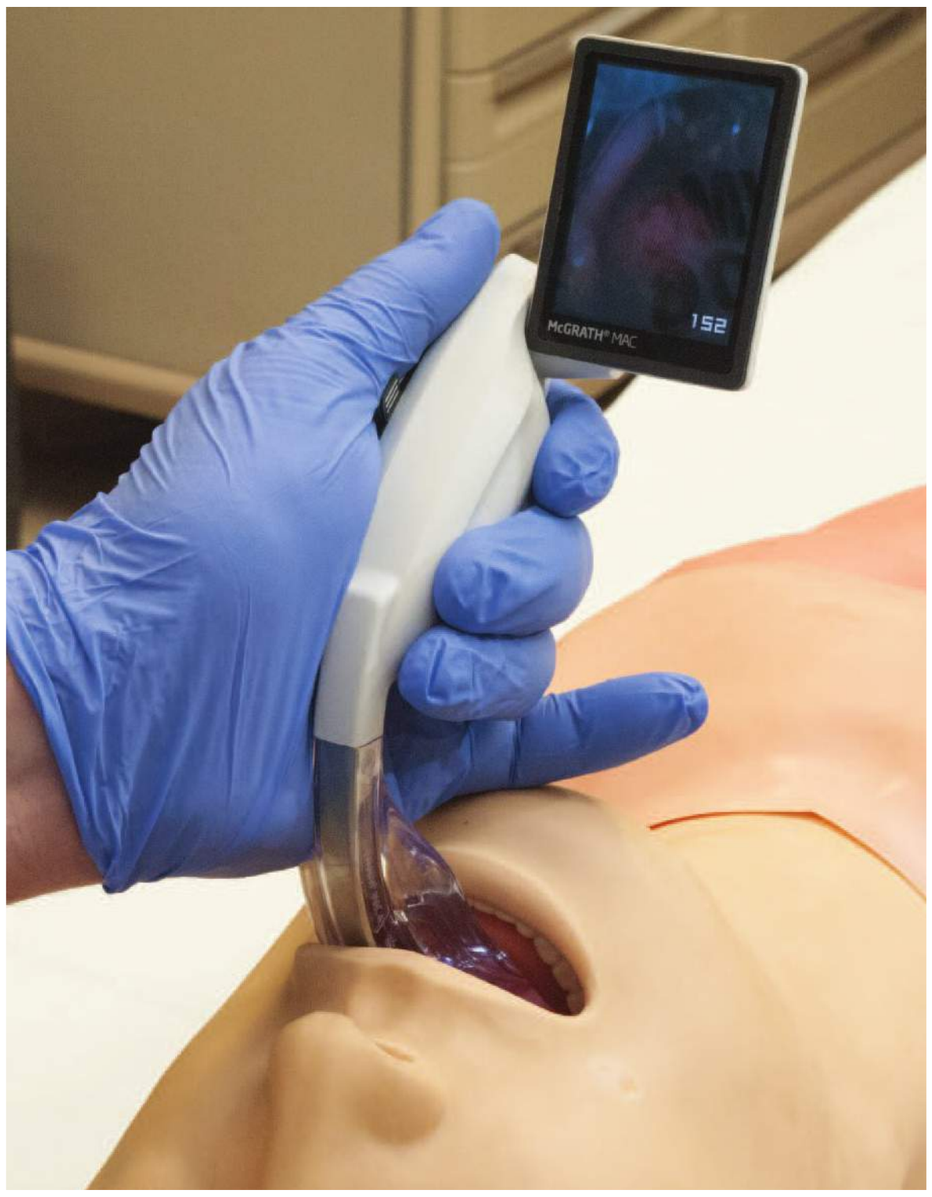


FIGURE 22.42 ■ **McGrath MAC EDL.** Cord visualization using the McGrath MAC which can also be used as a direct laryngoscope. (Photo contributor: Lawrence B. Stack, MD.)

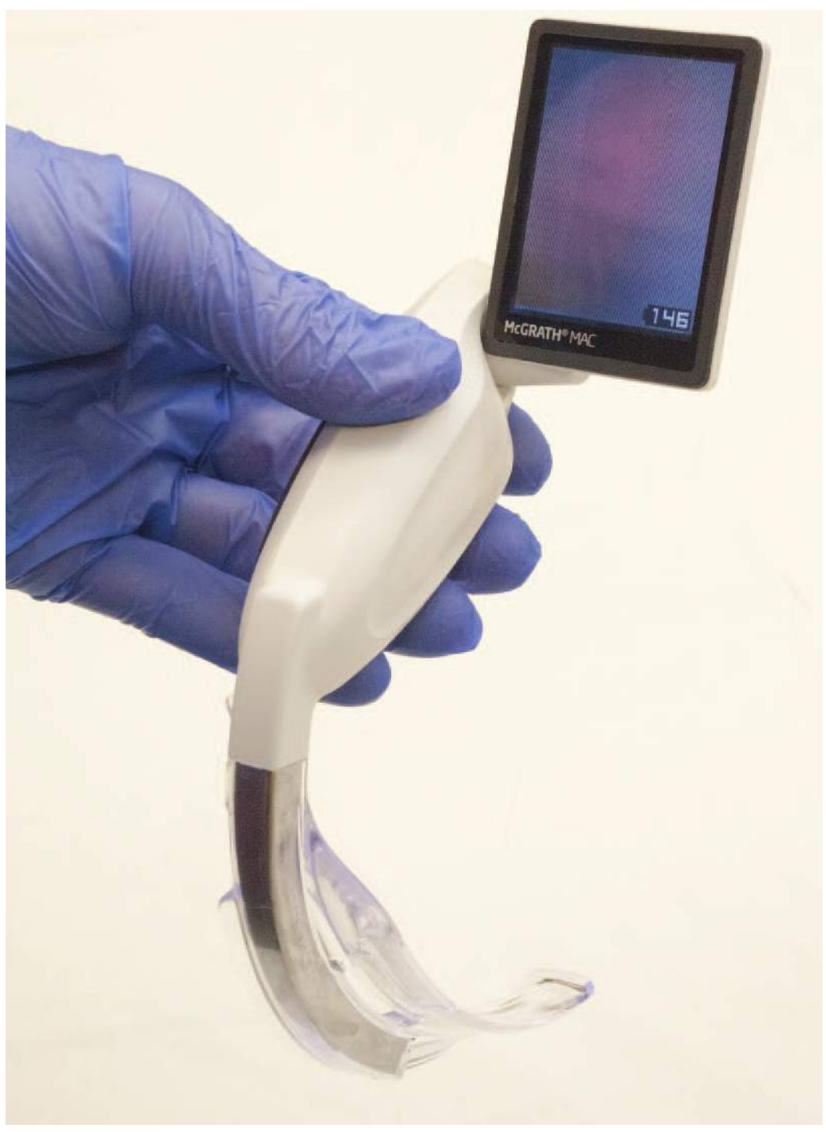


FIGURE 22.41 ■ **McGrath MAC Enhanced Direct Laryngoscope.** Compact video laryngoscope system shown here with a MAC 3 disposable blade. (Photo contributor: Lawrence B. Stack, MD.)

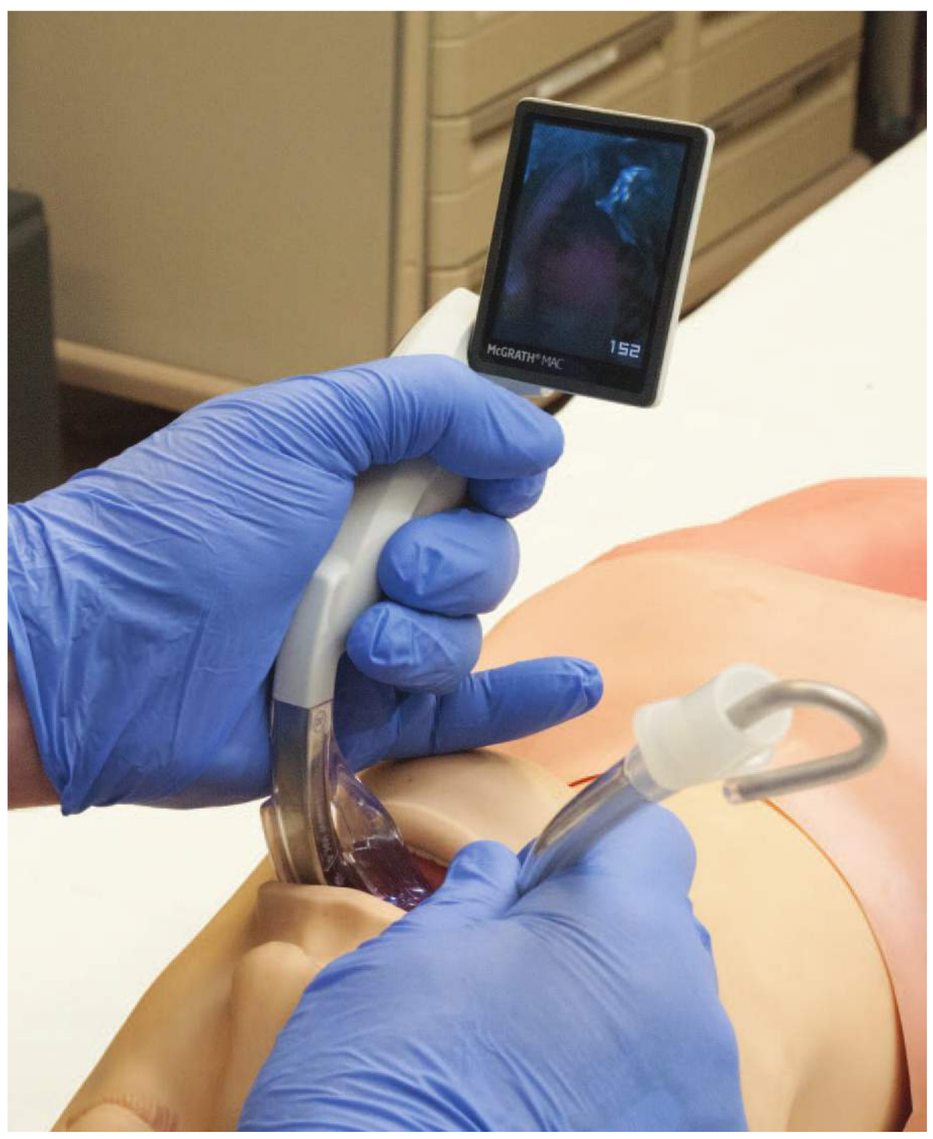


FIGURE 22.43 ■ **McGrath MAC EDL.** Vocal cord visualization and ETT delivery are demonstrated here. (Photo contributor: Lawrence B. Stack, MD.)

The laryngeal mask airway (LMA) was originally designed to facilitate ventilation during anesthesia for short operating room procedures. It has been shown to provide improved ventilation in cardiac arrest and failed airway cases and has a shallow learning curve, promoting its use by relative novices.

Equipment

The LMA consists of a short curved tube connected to a small mask with inflatable cuff. The shape promotes blind insertion with an endpoint detected as resistance as the leading edge of the cuff just enters and obstructs and the esophageal inlet. The inflated cuff then seals around the laryngeal inlet. The distal "mask" incorporates a small grate to prevent prolapse of the epiglottis within the mask. The LMA has several configurations including a disposable model, the LMA "Unique" (Figs. 22.44 and 22.45). LMAs are available in three adult sizes, #3 (30-50 kg), #4 (50-70 kg), and #5 (>70 kg). They are



FIGURE 22.45 ■ **LMA Unique.** The laryngeal surface of the LMA Unique with inflated cuff. Note the "grate" which prevents epiglottic prolapse into the mask. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.44 ■ **LMA Unique.** Adult sizes 3, 4, and 5 and pediatric size 2.5 are seen here. (Photo contributor: Lawrence B. Stack, MD.)

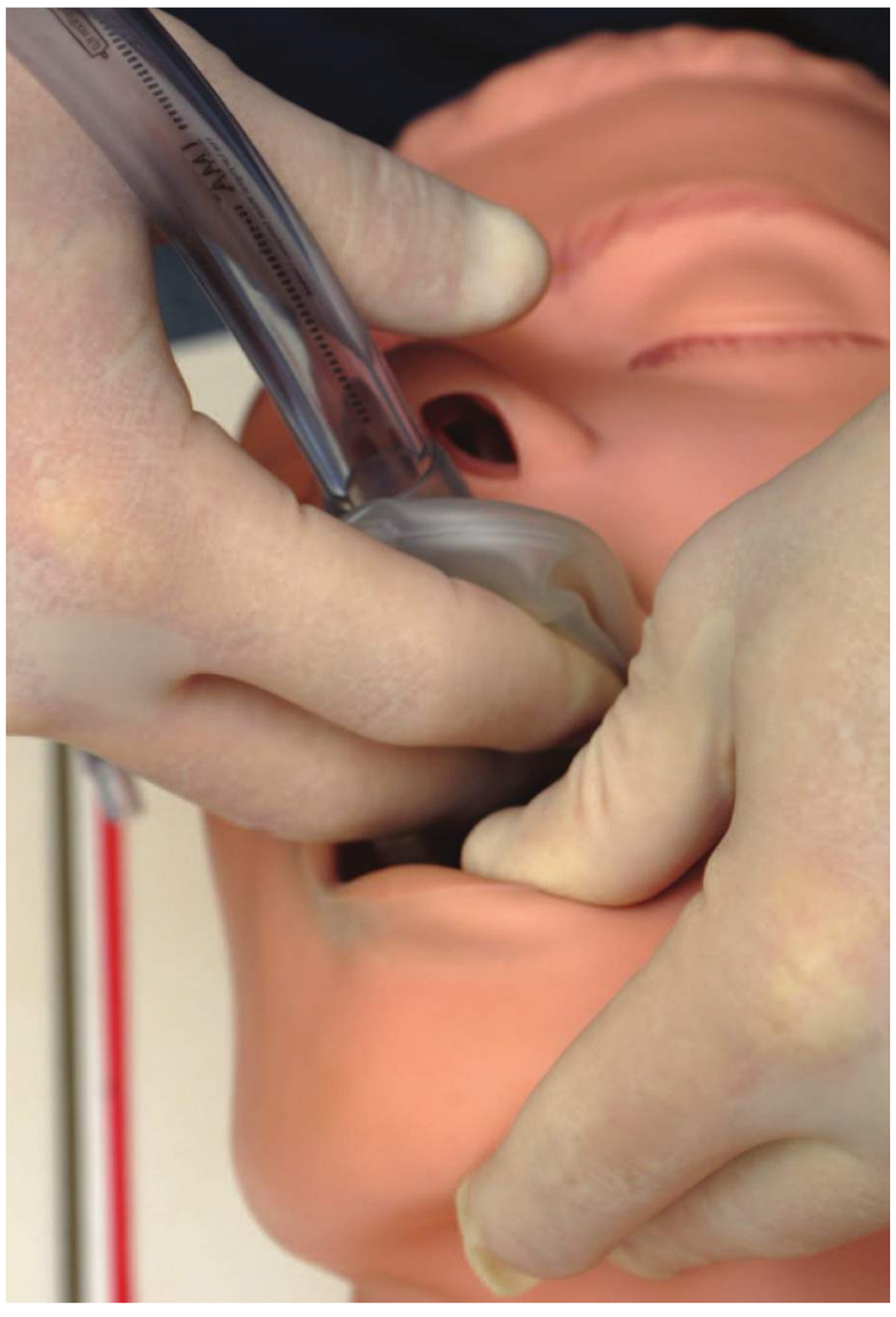


FIGURE 22.46 ■ **LMA Insertion.** LMA with deflated cuff is held against the hard palate during insertion by the index and third fingers. (Photo contributor: Lawrence B. Stack, MD.)

also available in pediatric sizes from size 0 to 2.5 for infants through toddlers.

Technique

The LMA is inserted into the mouth and held against the hard palate while being advanced into the hypopharynx. The LMA is advanced until resistance is met, and the cuff is then inflated with 20 to 40 mL of air to effect a seal. The mask should be lubricated prior to insertion.

Because the LMA aligns with the glottic opening, it is possible to intubate the trachea through the LMA with minimal interruption of ventilation. The distal grate prevents anything larger than a 6.0 ID ETT through a #3 and #4, and a 7.0 ID ETT through a #5. In addition to passing a small ETT directly, it is also possible to pass a flexible fiberoptic scope or a rigid fiberoptic scope (Levitan or Shikani) through the LMA under direct visualization. One must be sure to have an ETT, sized to pass through the LMA, loaded onto the stylet prior to passage. It is recommended to rotate the ETT counterclockwise 90 degrees to orient the bevel posteriorly, rotating clockwise as the tube is passed into the glottis.



FIGURE 22.47 ■ **Correct LMA Placement.** The LMA is correctly seated when resistance is met. Slight adjustments may be needed once seated. (Photo contributor: Lawrence B. Stack, MD.)

Equipment

The tracheal lightwand is a device which consists of a bright light bulb on the end of a wire. The best current embodiment is the TrachLight (Laerdal), which incorporates (1) a very bright distal light encased in a tube-like flaccid stylet, (2) a removable stiffening wire to provide rigidity and allow the stylet to conform to the required bending angle, (3) a handle which comprises a water-proof battery housing, on-off magnetic reed switch, and a track which provides for easy length adjustment of the stylet for different size ETTs. There is cam-like locking device to firmly secure the tube to the stylet. The stylet is marked in centimeter markings to align with those on the ETT, ensuring that the distal bulb is always at the proper position at the tip of the ETT.

Technique

The intubation technique relies on the unique appearance and location of the transilluminated glow of the skin overlying the anterior neck to provide feedback to the intubator about the ETT tip position and the subsequent stylet movements needed for tracheal placement and advancement. For intubation, the stylet is bent at the distal 5 cm, conveniently labeled with "BEND HERE." The bend angle is critical and depends upon the head and neck position of the patient. For a patient who is in a neutral head/neck position, such as a patient in a halo device, the stylet should be configured to about an 80 degree acute angle. If the patient is hyperflexed, a more acute angle is required, approximately 60 degrees. Finally, for patients who are hyperextended, an obtuse angle of 110 or 120 degrees would be appropriate.

The technique can be performed with the intubator facing the patient, or with the intubator at the head of the bed behind the patient. The lightwand should be held in the dominant hand with a pencil grip at the ETT hub/handle junction. With his nondominant hand, the intubator should grasp the tongue with gauze, gently distracting forward. This acts to bring the epiglottis anteriorly. The lightwand/ETT is introduced into the center of the mouth and advanced to the

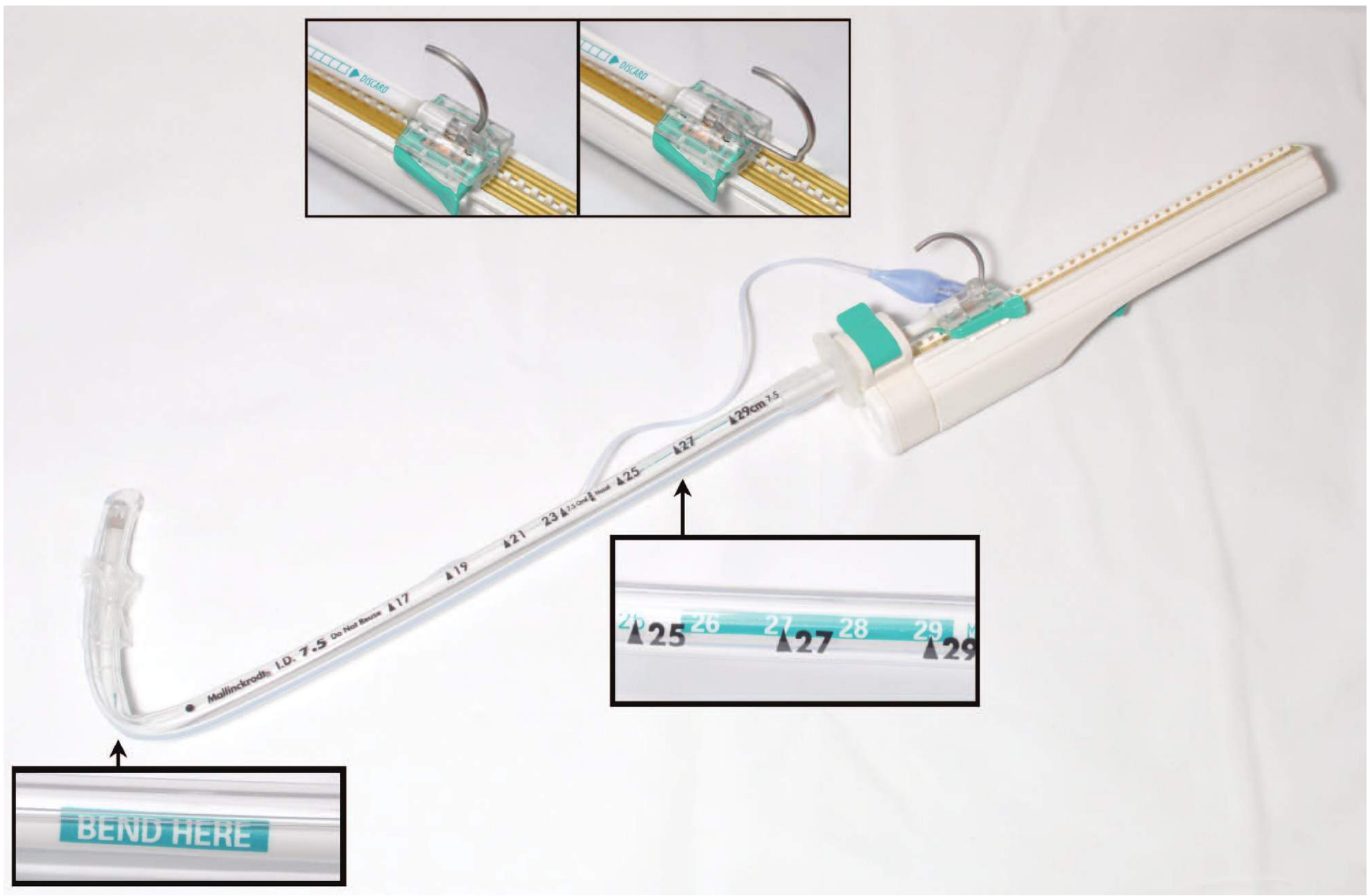


FIGURE 22.48 ■ **TrachLight by Laerdal.** The correctly assembled TrachLight demonstrates the correct alignment of the centimeter markings (inset) on the tube, the correct location of the bend (inset), and the correct engagement of the wire portion of the stylet (inset). (Photo contributor: Lawrence B. Stack, MD.)

hypopharynx while maintaining anterior pressure, holding the elbow high and the arm abducted, to keep the stylet bulb anterior. Advancement stops when the intubator recognizes an orange glow on the anterior neck tissues or when tube advancement meets resistance. The appearance and position of this glow determines the next maneuver.

If transillumination positional feedback indicates that the tube tip is in one of the pyriform fossae, the stylet/ETT is withdrawn slightly and rotated toward the midline and reinserted. This process is continued repeatedly in an iterative manner until the correct midline glow indicating tracheal position is encountered. At that point, the stiffening wire is withdrawn several centimeters, resulting in a caudad displacement of the glow as the intratracheal stylet "relaxes." The tube and stylet is then advanced until the glow just disappears below the sternal notch. The tube is now confirmed to be intratracheal and the tube tip is confirmed to be reliably in the mid-trachea, with no need for confirmatory chest radiograph.

Failure to modify the position of the transilluminated glow usually indicates that the stiffening wire has displaced from the locking notch that keeps the wire from rotating in place.

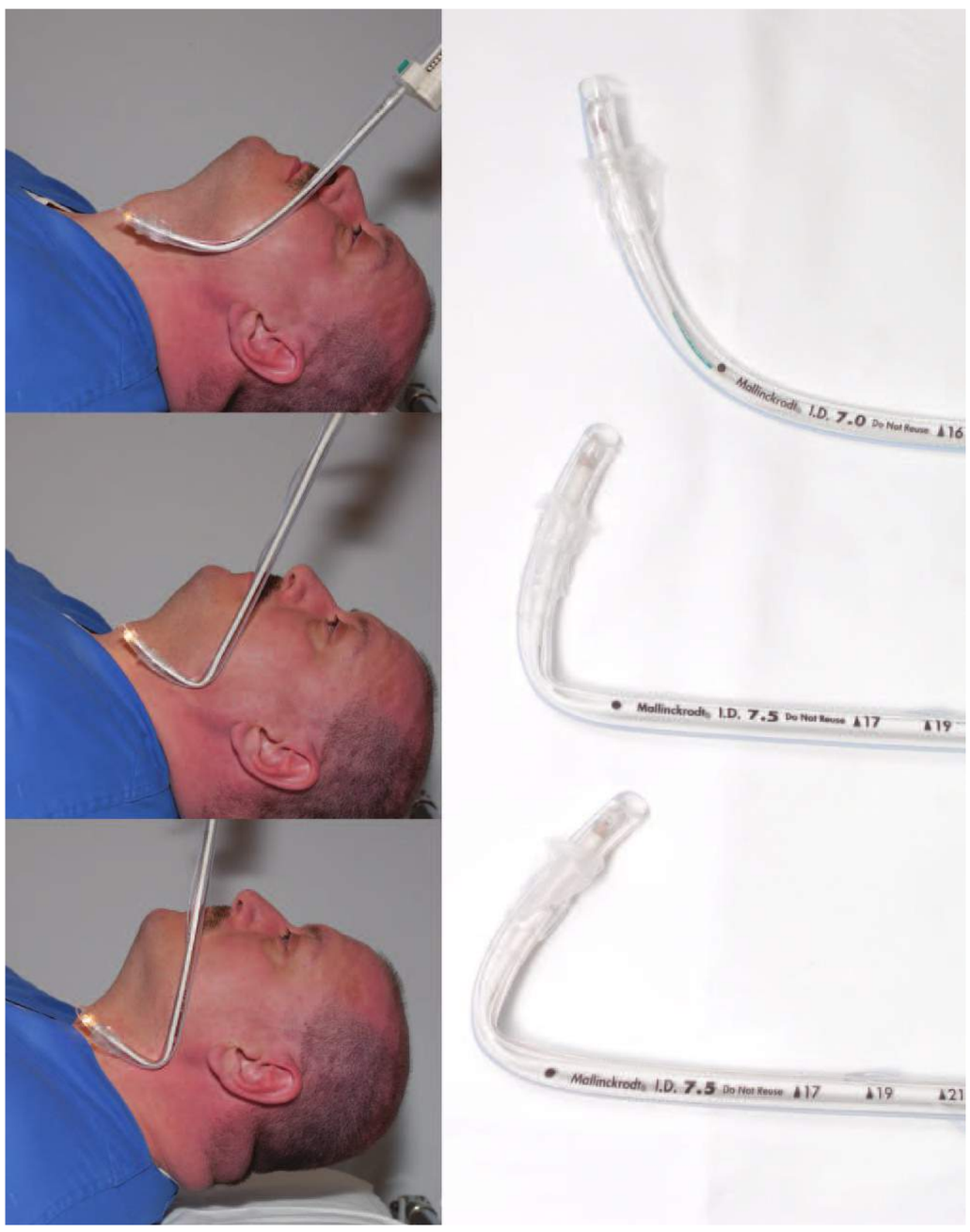


FIGURE 22.49 ■ **TrachLight Bend Angle.** The head/neck position and suggested corresponding bend angle of the TrachLight. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.50 ■ **TrachLight Positioning.** The correct TrachLight position is demonstrated here. The device is placed in the center of the mouth while the left hand is lifting the tongue anteriorly. (Photo contributor: Lawrence B. Stack, MD.)

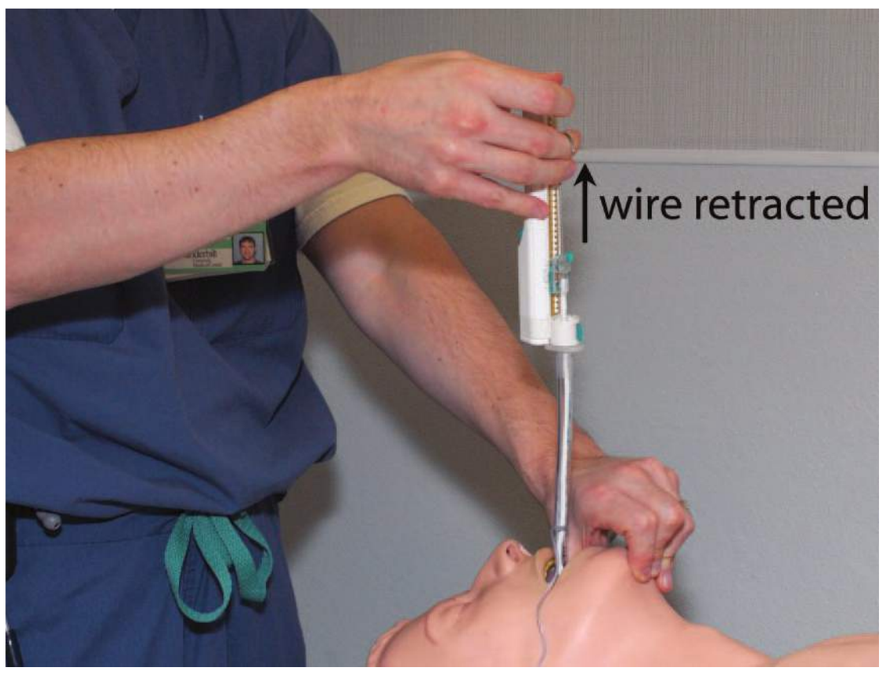


FIGURE 22.51 ■ **TrachLight Technique.** Once the tracheal position is encountered, the stiffening wire is retracted several centimeters before advancing ETT into the trachea. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.52 ■ **TrachLight Technique.** Once the tracheal position is encountered, the stiffening wire is retracted several centimeters before advancing ETT into the trachea. (Photo contributor: Lawrence B. Stack, MD.)

TABLE 22.2 ■ TRACHLIGHT MANEUVER DECISIONS

Glow appearance	Position of glow	Anatomic position of stylet tip	Next maneuver
Bright orange, well localized	High neck/submental 	Vallecula	Remove stylet, decrease acuity of bend angle
Dull orange glow	Right/left of neck midline 	Right/left pyriform fossa	Partially withdraw stylet, rotate slightly to midline, re-advance, keeping tip anterior
No glow or very dim	If present, thin line at lateral neck may be bilateral	Esophagus	Remove stylet, increase acuity of bend angle
Bright midline glow	Thyroid prominence 	Glottic opening	Withdraw stiffening wire by 3 cm, advance stylet/ETT until glow just disappears beyond sternal notch

(Photo contributor: Lawrence B. Stack, MD.)

DIGITAL (TACTILE) INTUBATION

Digital intubation relies on tactile definition of intubation landmarks, primarily the epiglottis but often the posterior cartilages and interarytenoid notch.

Technique

Digital intubation technique is performed with the intubator facing the patient from the side. With the gloved nondominant hand, the intubator should insert the index and third fingers into the patient's mouth. He should next "walk" the fingers down the tongue to progressively displace the tongue anteriorly as the fingers are advanced to the epiglottis. The epiglottis will have a feel similar to the earlobe. As the third finger encounters the epiglottis, the finger traps and holds the epiglottis anteriorly. The ETT, with stylet configured to an open "C," is introduced by the dominant hand into the corner of the mouth and advanced to the palmer side of the intraoral index finger. The intraoral index finger is then used to guide the ETT tip to the third finger, where the ETT is slipped between the epiglottis and third finger. The third finger is used to keep the ETT anterior against the laryngeal surface of the epiglottis while the ETT is advanced by the dominant hand and assisted by the intraoral index finger. It is often possible to palpate the arytenoid cartilages/interarytenoid notch, in which case one need only ensure that the ETT tip remains anterior to those cartilages and is prevented from drifting posteriorly to the esophageal inlet. The posterior cartilages project cephalad, feeling like snake fangs. Correct ETT position can be confirmed by palpating the arytenoid cartilages posterior to the ETT.

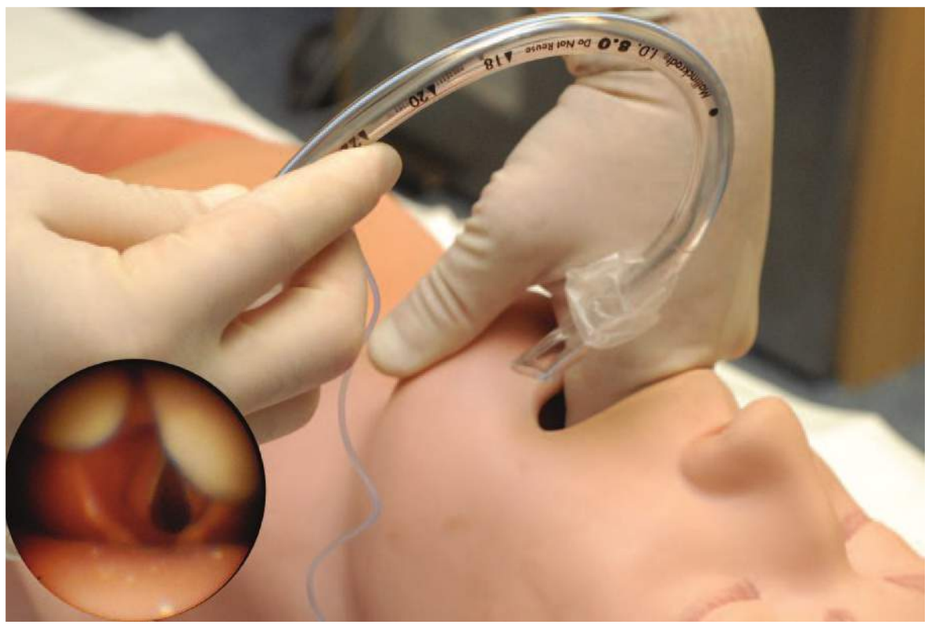


FIGURE 22.56 ■ **Digital Intubation Technique.** Index and third finger inserted into the mouth with "C" shaped stylet held in left hand. Third finger holds epiglottis anteriorly (inset). (Photo contributor: Lawrence B. Stack, MD.)

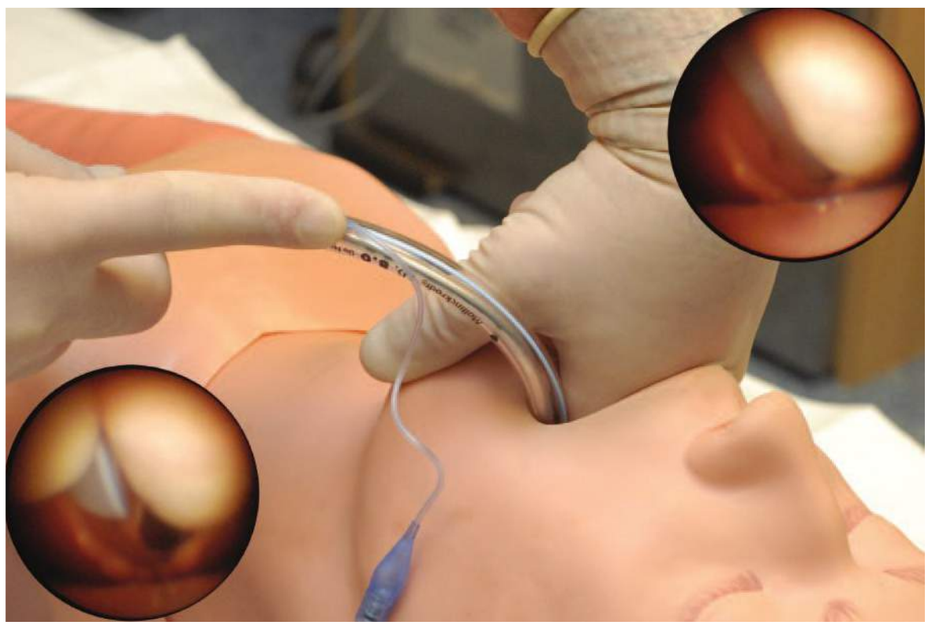


FIGURE 22.57 ■ **Digital Intubation—ET Tube Passage.** Intraoral index finger guides ETT to third finger (inset) which directs the tube into the glottic opening (inset). (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. Digital intubation may be the best airway delivery option in austere conditions or in patient with copious airway secretions or blood.
2. Can only be used in a paralyzed, comatose, or dead patient.

Equipment

The reusable ILA is similar in concept to the classic LMA but has a structural features that provide an easier and more stable insertion with less likelihood of tube kink. In addition, it has a unique "keyhole" like airway outlet which helps to direct an ETT to the midline at the proper glottic entry angle. Because it has a larger lumen, the ILA can accommodate standard ETT up to 8.5 mm ID. The ILA comes in five sizes—1.0 (<5 kg), 1.5 (10-20kg), 2.4 (20-50kg), 3.5 (50-70kg), and 4.5 (70-100kg), accommodating maximum ETT sizes of 4.5, 5.5, 6.5, 7.5, and 8.5, respectively.



FIGURE 22.60 ■ **Intubating Laryngeal Airway Insertion.** The ILA is inserted by displacing the tongue and jaw anteriorly and is advanced down the curvature of the oropharynx and hypopharynx. (Photo contributor: Lawrence B. Stack, MD.)

Technique

The mandible is displaced anteriorly with a jaw lift maneuver, bringing the tongue and epiglottis forward. The ILA is

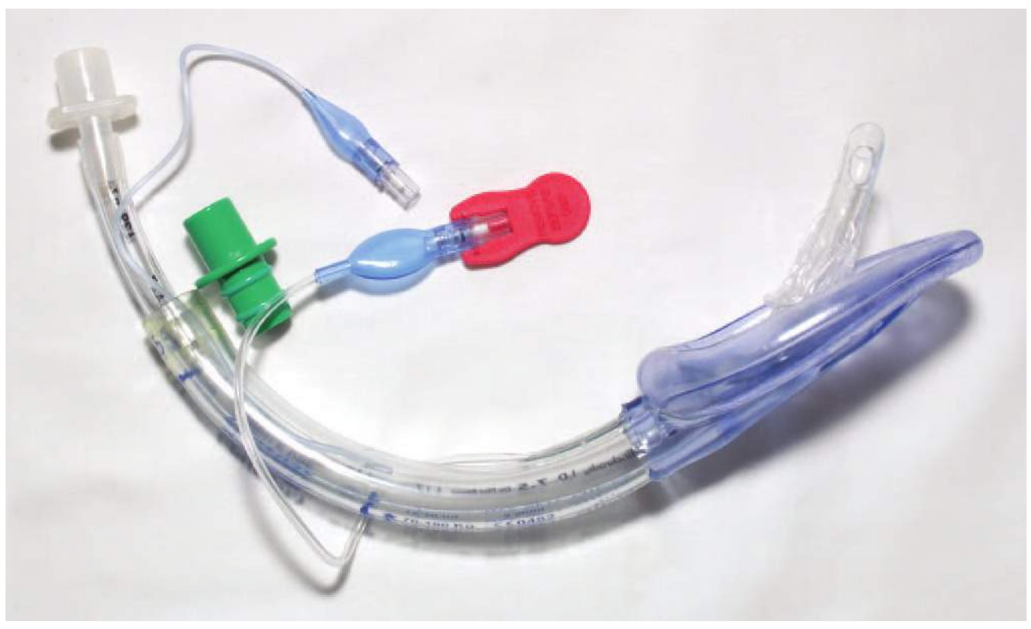


FIGURE 22.58 ■ **Intubating Laryngeal Airway**. Intubating LMA demonstrating an ETT through the device lumen. (Photo contributor: Lawrence B. Stack, MD.)

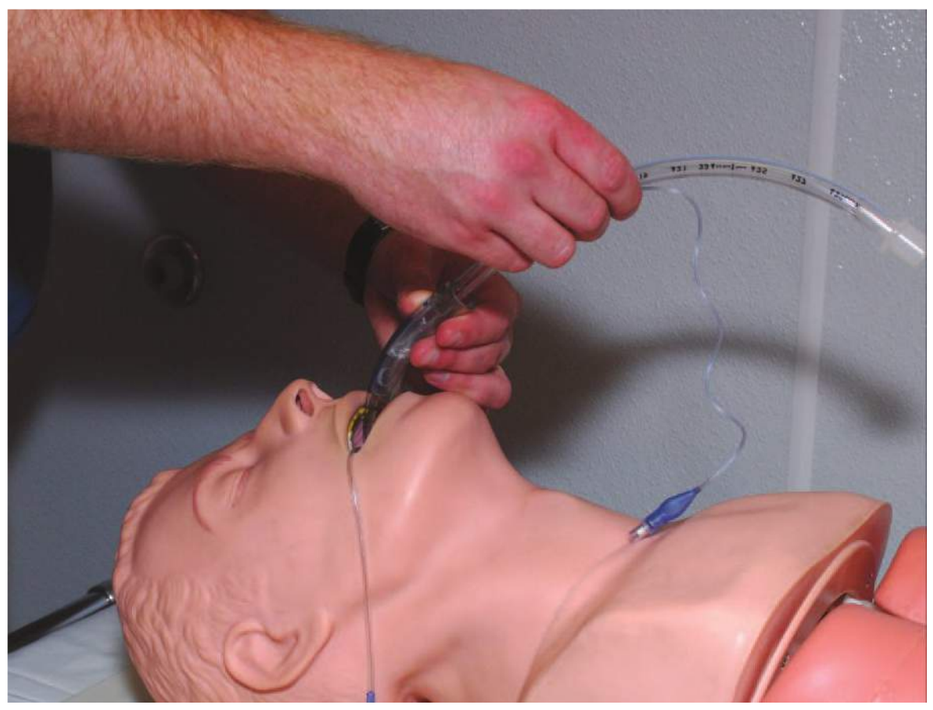


FIGURE 22.61 ■ **ETT Insertion Through the Intubating Laryngeal Airway.** An ETT is advanced through the ILA. (Photo contributor: Lawrence B. Stack, MD.)

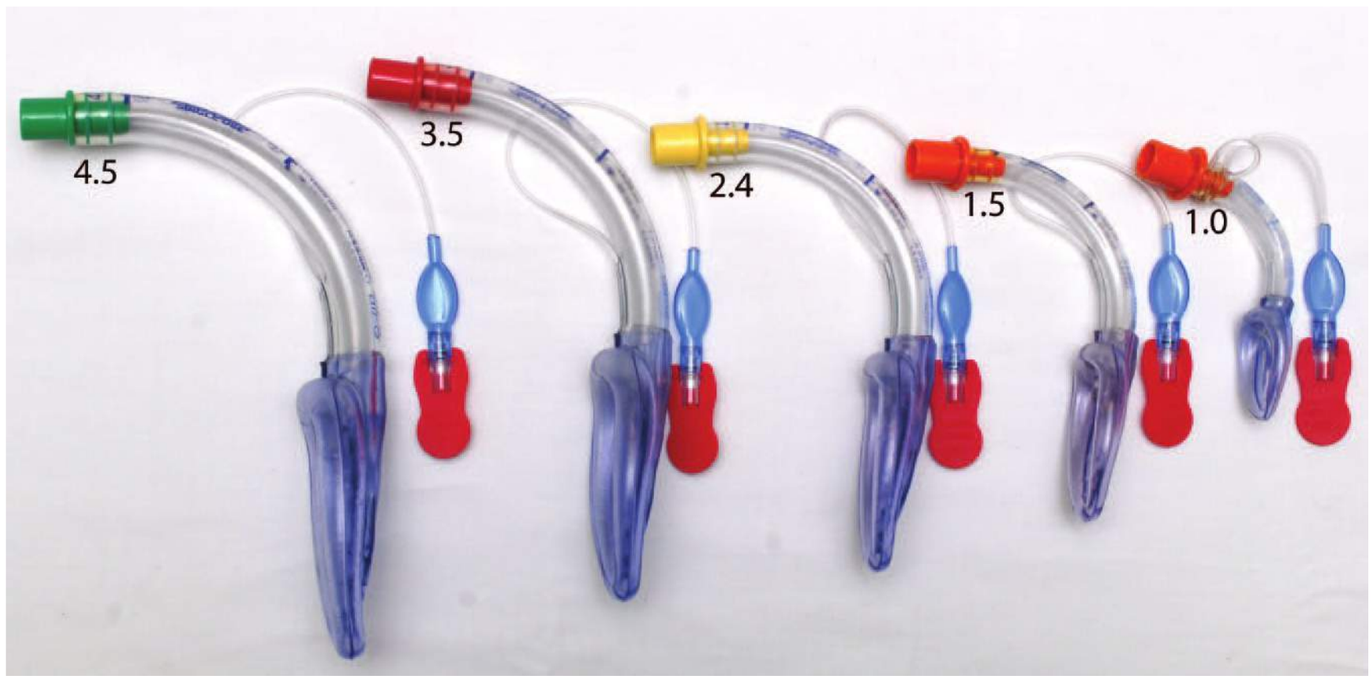


FIGURE 22.59 ■ **Intubating Laryngeal Airway Sizes.** ILA in five different sizes. (Photo contributor: Lawrence B. Stack, MD.)

then inserted into the mouth, advancing with a downward and inward force to follow the curve of the oropharynx/hypopharynx. The insertion endpoint is firm resistance to further insertion. The cuff is then inflated with approximately 20 mL of air or until the pilot balloon is firm. Ventilation can proceed by attaching a ventilating bag to the proximal port.

Intubation can be accomplished through the ILA in a well-sedated/chemically paralyzed patient after first removing the proximal 15-mm adaptor port. A well-lubricated standard ETT can then be inserted through the ILA. This procedure can also be aided by using a lightwand, opitcal sytlet, or fiberoptic intubating bronchoscope, or bougie, through the ETT to facilitate optimal alignment for intubation.

Once the ETT is inserted, the ILA can be easily removed by inserting the disposable tube stabilizer/stylet.

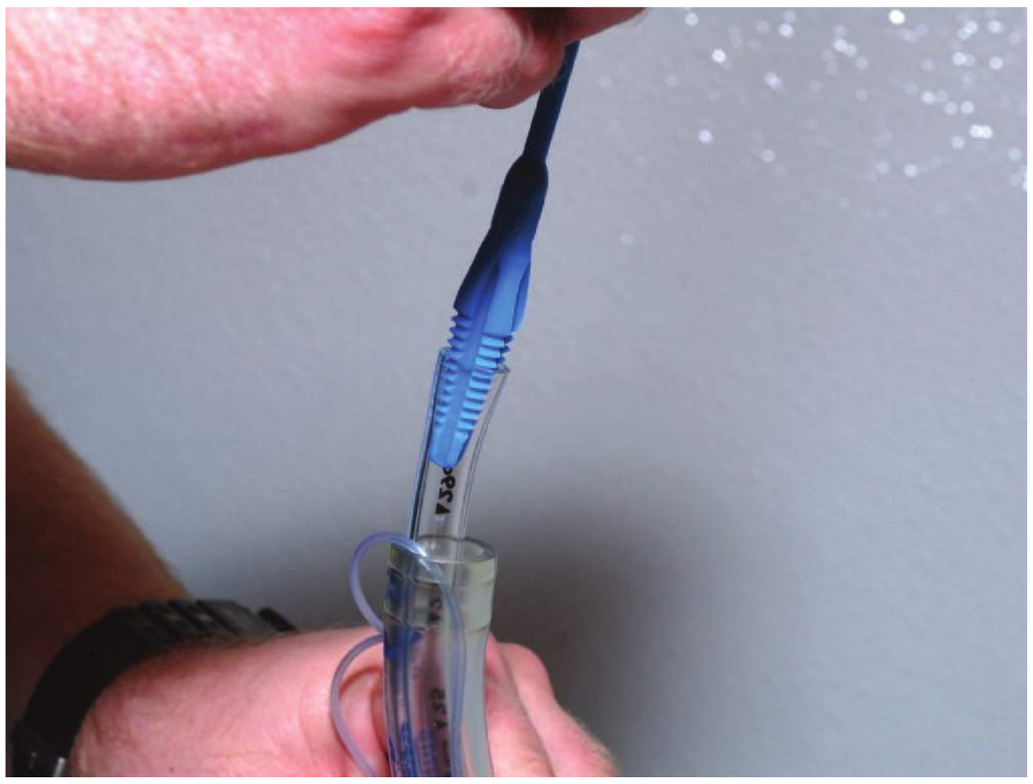


FIGURE 22.62 ■ **Intubating Laryngeal Airway Removal.** Removal of the ILA while keeping the ETT in place is accomplished by using the disposable tube stabilizer (blue). (Photo contributor: Lawrence B. Stack, MD.)

Equipment

The King laryngeal tube (King LT) consists of an airway tube with two cuffs. The distal cuff seals the esophagus and the proximal seals the oropharynx and nasopharynx when inflated. Between the cuffs are multiple ventilation channels. Inflation of the cuffs occurs through one inflation line. A separate gastric drainage channel is present in the disposable model (King LTS-D).

Technique

A lubricated King LTS-D is inserted behind the tongue while preforming a jaw lift, placing the patient in a sniffing position. The tube is advanced until resistance is met or until the connector base is aligned with the teeth or gums. The cuffs are inflated with 60 to 90 mL of air, depending on the size of the King LTS-D. Some "giggling" may be required to properly seat the tube.

Pearls

1. Most unsuccessful King LT placement attempts are due to failure to keep the tube in midline during insertion.
2. Anticipate vomiting when the King LT is removed.



FIGURE 22.63 ■ **King LTS-D Airway.** The King LTS-D is shown with the distal esophageal balloon and the proximal hypopharynx balloon inflated. (Photo contributor: Lawrence B. Stack, MD.)

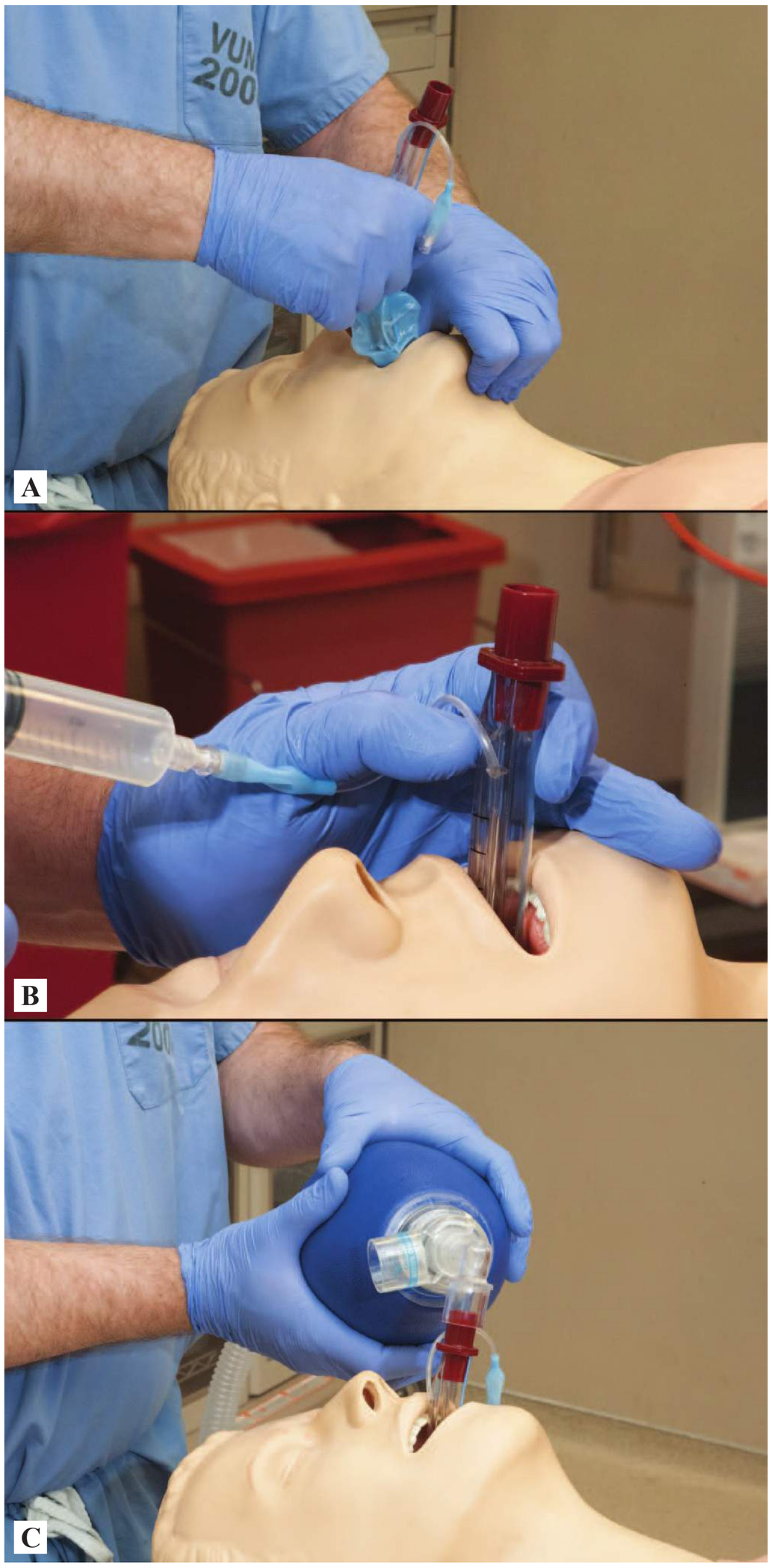


FIGURE 22.64 ■ **King LTS-D Airway Technique.** The King LTS-D is passed down the anterior surface of the tongue while the tongue and mandible are pulled upward. The tube is advanced until resistance is met or the base of the connector is at the level of the lip or gum. The balloon is inflated with 45 to 90 mL of air depending on the size of the tube. (Photo contributor: Lawrence B. Stack, MD.)

Patient Preparation

The nares should be inspected for patency and the side deemed most hospitable to tube passage should be anesthetized and vasoconstricted, Additionally, it is helpful to confirm nasal patency by inserting a well-lubricated nasal trumpet. Lidocaine jelly can be used as the lubricant to provide additional topical anesthesia. We favor the use of disposable atomizers to instill 2 mL of a mixture of tetracaine 1%/phenylephrine 0.5%, which yields excellent mucosal anesthesia and vasoconstriction. Favorable intubation conditions can be achieved in about 5 minutes.

In preparation for nasal intubation, the ETT can be conformed into a circle by inserting the tip into the 15-mm proximal adaptor, and soaked in warm water to soften the tube for nasal passage. Success can also be enhanced by the use of a special ETT, called an Endotrol control tip ETT. This tube incorporates a length of nylon fishing line within a channel in the anterior wall of the ETT. Pulling on the proximal loop "trigger" causes anterior deflection of the tube tip.



FIGURE 22.65 ■ **Verification of Patency.** A well-lubricated nasopharyngeal airway will verify patency of the nasal passage prior to NTT insertion. (Photo contributor: Lawrence B. Stack, MD.)

Technique

Once the selected naris is suitably anesthetized, the intubator faces the patient, who can be sitting up or supine, and inserts the tube into the naris, parallel to the nasal floor/perpendicular to the facial plane (not along the nasal axis). To minimize trauma to the nasal turbinates, the bevel of the tube should face the turbinates. This implies that if the left naris is being intubated, the tube can be inserted along the normal curve of the nose-nasopharynx; however, if the right naris is being intubated, the tube should be inserted with the curve of the tube in a cephalad direction until the tube is advanced to the nasopharynx, at which point the tube is rotated to the correct anatomic configuration. As the tube is slowly advanced to the hypopharynx, the intubator listens closely for transmitted breath sounds, rotating the tube right or left to maintain maximal transmission. It takes a more-severe-than-expected rotation externally to generate a small rotation of the supraglottic tube tip. The tube is then rapidly advanced unless resistance is encountered, as would be expected with pyriform sinus tube tip position. Correct intratracheal placement should elicit cough, a reflex deep inspiration, and a loss of phonation. The intubator may also be sprayed with coughed secretions. If breath sound transmission decreases with advancement, the tube is withdrawn and re-advanced, preferably with more

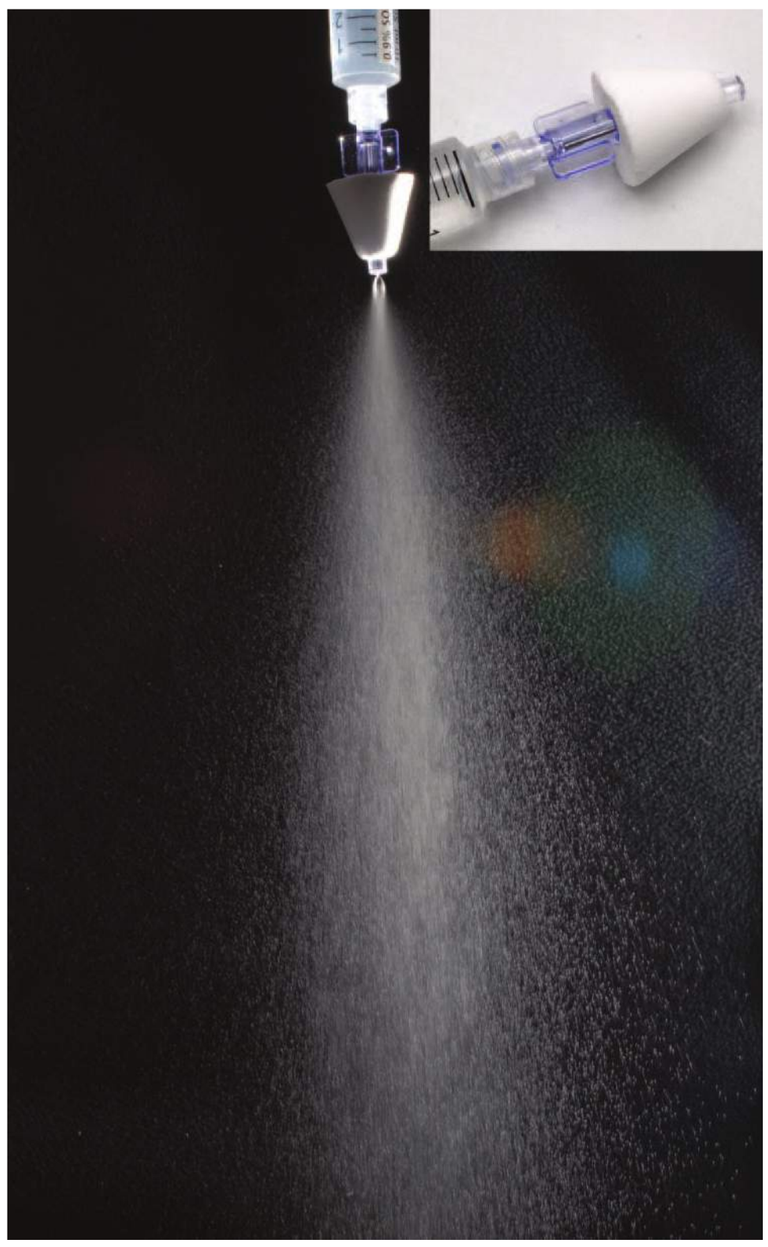


FIGURE 22.66 ■ **Atomizer.** A disposable atomizer used to deploy topical vasoconstrictors and analgesics prior to NTT insertion. (Photo contributor: Lawrence B. Stack, MD.)

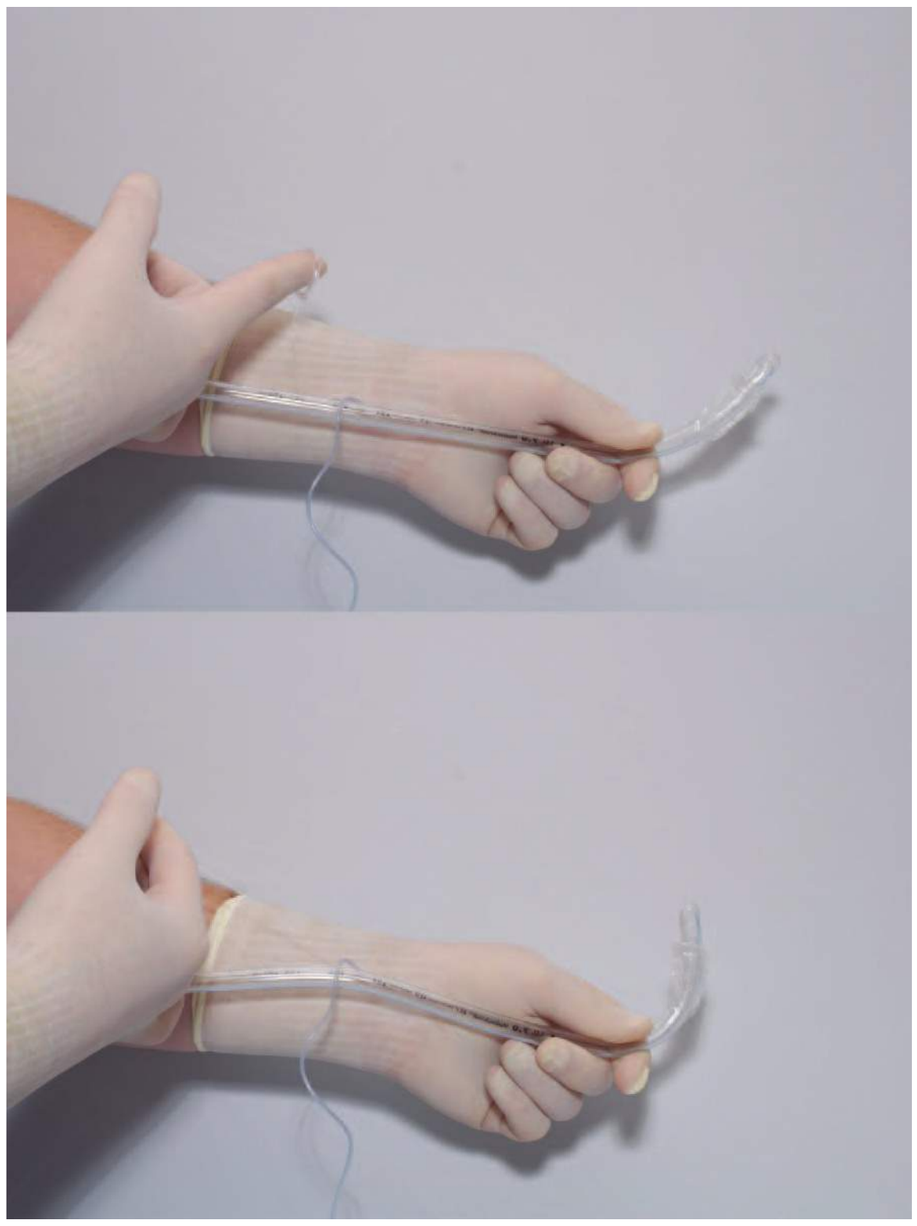


FIGURE 22.67 ■ **Endotrol ETT.** Demonstration of the ETT tip control using the Endotrol trigger. (Photo contributor: Lawrence B. Stack, MD.)

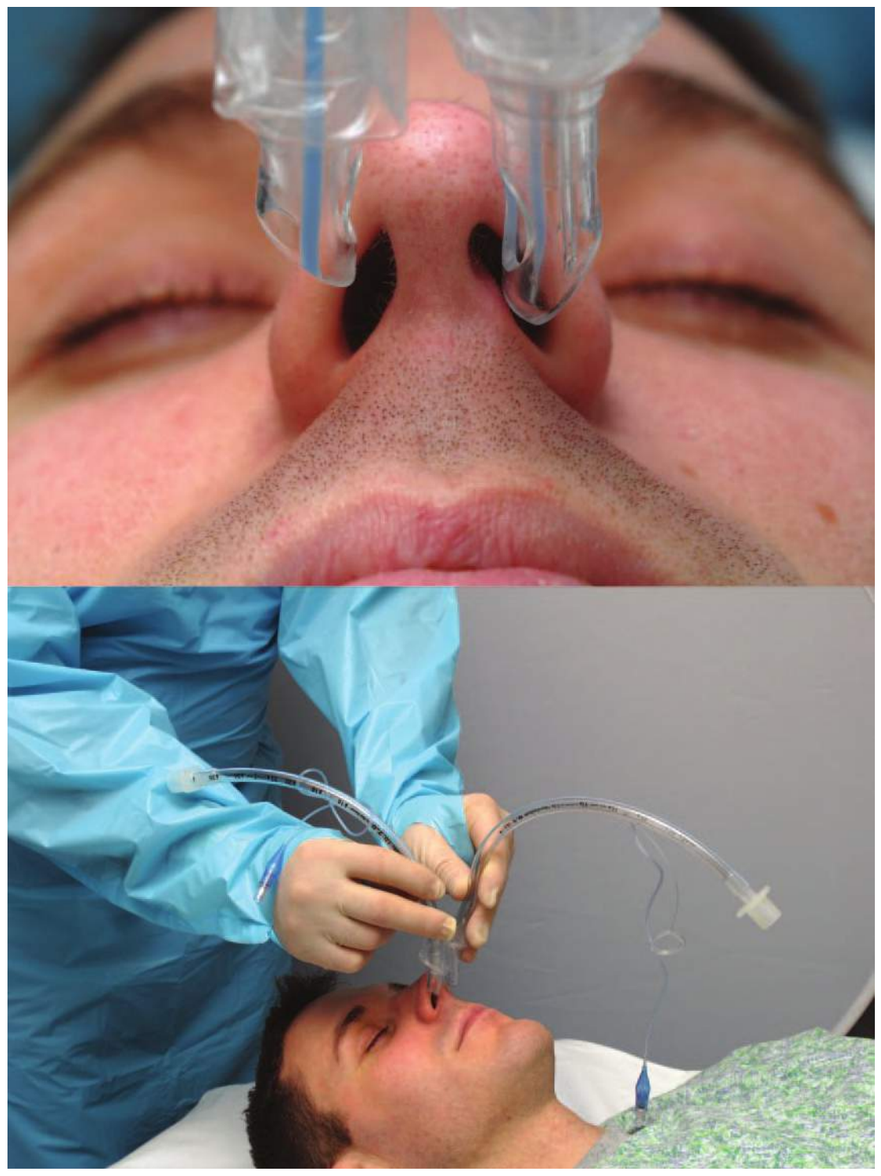


FIGURE 22.68 ■ **Bevel Placement for NT Intubation.** When inserted into the nose, the bevel of the tube should face the turbinates to minimize the trauma to these structures. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.69 ■ **Maneuvering for Maximized Breath Sounds.** Once the ETT is in close proximity to the glottic opening, the intubator must rotate the tube left or right till maximum air movement is heard, then advance the tube. (Photo contributor: Lawrence B. Stack, MD.)

anterior displacement of the tip if the Endotrol tube is used. If such a tube is not available, anterior displacement can be produced by partially inflating the ETT cuff.

The audible feedback to determine tube tip position can be enhanced by the attachment of the Beck Airway Airflow Monitor (BAAM), which creates an amplified sound with air movement, much like the sound of a tea kettle. Alternatively, one can remove the diaphragm/bell from a stethoscope and insert the distal end of the stethoscope tubing into the proximal end of the ETT. In addition to amplifying transmitted sounds, this trick also can spare the intubator from a sputum ear wash. Positional feedback can also be enhanced by use of a quantitative waveform capnometer/capnograph.

Pearls

1. Blind nasotracheal intubation offers a viable option in the event that oral intubation is rendered impossible in the short term, such as a patient who has a wired jaw or large subglossal hematoma.
2. The blind nasotracheal intubation technique requires maintenance of spontaneous respirations because the intubator relies on audible variation in transmitted breath sounds via the ETT.
3. The TrachLight lightwand can be used for blind nasal tracheal (NT) intubation, with the stiffening wire removed. Tube tip position is determined identically to that with orotracheal transillumination, with identical repositioning maneuvers, except that rotational maneuvers are likely to be more extreme as noted above.

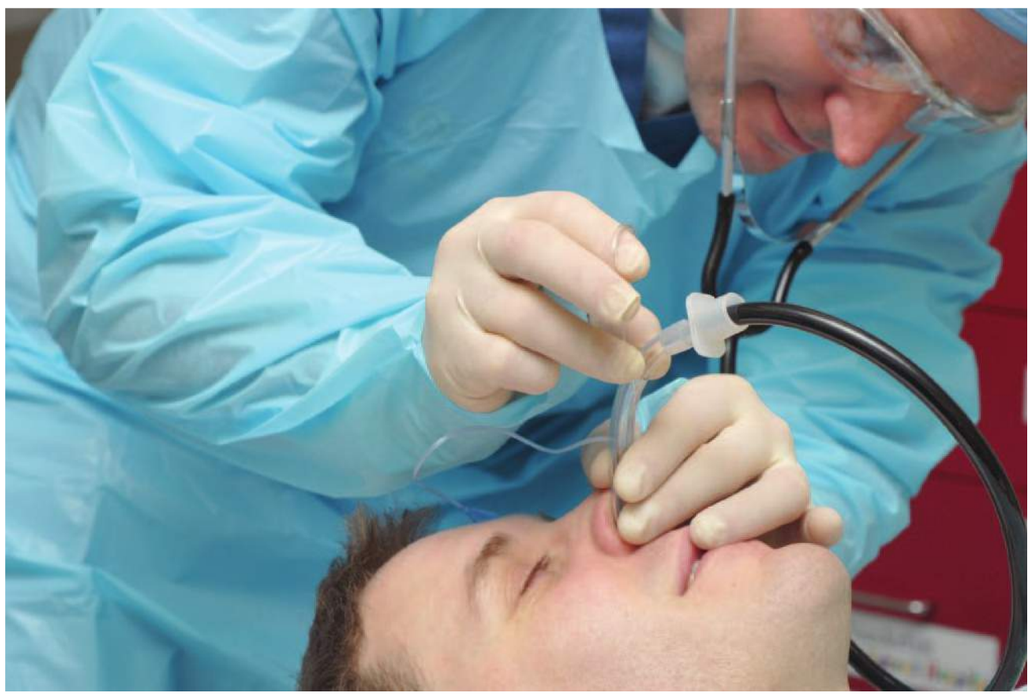


FIGURE 22.71 ■ **Stethoscope Airflow Monitor.** Removing the diaphragm/bell from a stethoscope and placing it in the end of the ETT will enhance identify the location of maximum air movement. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.70 ■ **Beck Airway Airflow Monitor (BAAM).** The BAAM makes an amplified sound to help identify location of maximized air movement. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.72 ■ **TrachLight Augmentation of NT Intubation.** Remove the stiffening wire, and place the stylet into the ETT and use conventional landmarks to guide maneuvering decisions for proper tube placement. (Photo contributor: Lawrence B. Stack, MD.)

There are two methods of nasal flexible fiberoptic technique: (1) scope-first and (2) tube-first.

Patient Preparation

For both methods, the nose is prepared as described above under blind nasotracheal intubation.

Technique: Scope-First

The largest possible ETT, preferably 7.0 mm ID, is inserted over the endoscope to the most proximal position of the endoscope. The fiberoptic endoscope is then inserted through the selected naris. Under direct visual guidance, the endoscope is advanced along the floor of the nose, navigating under the inferior turbinate. The endoscope tip is manipulated by a combination of trigger deflection (up/down) and scope rotation (left/right) to keep the lumen in the center of the optical field. As the tube is advanced to the posterior nasopharynx, the scope tip is deflected downward to reveal the epiglottis and laryngeal inlet. The scope is advanced further, with the operator maneuvering the scope tip to keep the glottic opening in the center of the optical field. With the endoscope tip just above the cords, 2 mL of 1% lidocaine should be injected through the injection port of the fiberscope to provide vocal cord anesthesia prior to tube passage. The endoscope advancement continues until the endoscope tip is approximately 3 to 5 cm above the carina. The tube is then gently advanced over the fiberoptic sheath into the naris and to the trachea. If resistance is encountered at 14 to 16 cm, the tube may be



FIGURE 22.73 ■ **Scope-First Method.** The ETT is placed at the proximal extreme. The endoscope is then guided to the glottic opening and the tube is then advanced over the endoscope. (Photo contributor: Lawrence B. Stack, MD.)

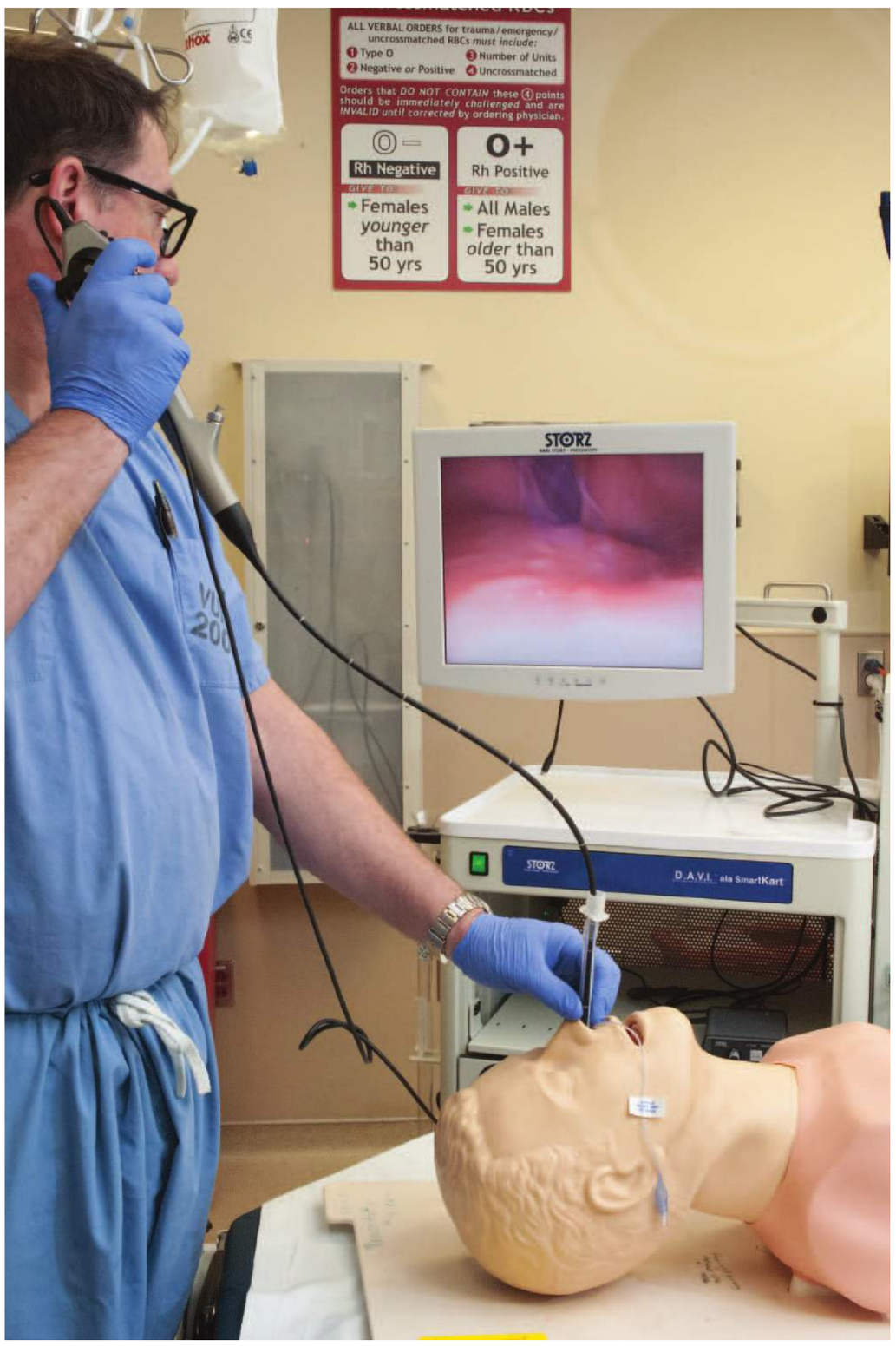


FIGURE 22.74 ■ **Tube-First Method.** The ETT is advanced to the hypopharynx. The endoscope is then advanced through the tube and maneuvered to the glottic opening. The tube is then advanced over the endoscope. (Photo contributor: Lawrence B. Stack, MD.)

impacting upon the right arytenoid. As discussed previously, this can be corrected by a severe counterclockwise rotation. The tube tip should be verified to be the correct distance from the carina.

If, during endoscope navigation through the nose, the view becomes blurry because of adherent secretions, the tip can be cleared by gently deflecting the tip into "pink" mucosa. If one becomes lost or disoriented during scope advancement or if there is no obvious lumen through which to advance, the endoscope should be withdrawn slightly and the tip maneuvered to provide a recognizable view. The endoscope should not be advanced if a lumen is not readily apparent.

Technique: Tube-First

To avoid some of the technique difficulty inherent with nasal navigation, the ETT can be passed through the naris and advanced into the hypopharynx first, before scope insertion. The endoscopist then inserts the intubating fiberscope through the ETT, and advances it to and through the glottic opening to the appropriate distance above the carina. Tube advancement proceeds as described above. The tube-first method may also facilitate endoscopic intubation in the event of epistaxis.

CRICOTHYROTOMY (SELDINGER TECHNIQUE)

Equipment

The Melker kit manufactured by Cook Medical includes all the equipment necessary: catheter, thin-walled needle, Seldinger wire, cuffed tracheostomy tube 5.0 mm ID, and tapered introducer.

Technique

1. Identify the thyroid cartilage and the cricothyroid membrane directly caudally.
2. Prep the neck with suitable antiseptic; anesthetize the skin overlying the cricothyroid membrane with 1% lidocaine.
3. Isolate the trachea between the thumb and third finger of the sterilely gloved nondominant hand, using the index finger to palpate the cricothyroid membrane landmark. With a syringe attached, preferably containing sterile water or remaining lidocaine, insert the catheter through the cricothyroid membrane at a 30-degree angle to the horizontal, aiming toward the feet. Once through the skin, begin aspirating as the catheter is advanced. Air and bubbles entering the syringe confirm entry into the trachea.
4. Carefully slide the catheter off of the needle and into the trachea.
5. Reattach syringe and reconfirm tracheal position.
6. Thread Seldinger wire through catheter into trachea; lack of resistance to wire passage confirms correct position.
7. Remove catheter and, with the included scalpel, make a small incision at the wire-skin entry site.
8. Insert tracheostomy tube with dilator/introducer over wire into trachea. Expect significant resistance. Inability to pass dilator may indicate an inadequate skin incision
9. Withdraw dilator/introducer and wire as one unit, inflate cuff and initiate ventilation.
10. Secure tracheostomy tube with tracheostomy ties



FIGURE 22.76 ■ **Identify Landmarks.** Palpate thyroid cartilage and cricothyroid cartilage caudally. Angle needle 30 degrees caudad to enter the trachea. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.75 ■ **Melker Cricothyrotomy Kit.** Components of the kit produced by Cook Inc. (www.cookmedical.com). (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.77 ■ **Bubbles during Aspiration.** Aspiration of bubbles during needle advancement confirms tracheal placement. (Photo contributor: Lawrence B. Stack, MD.)

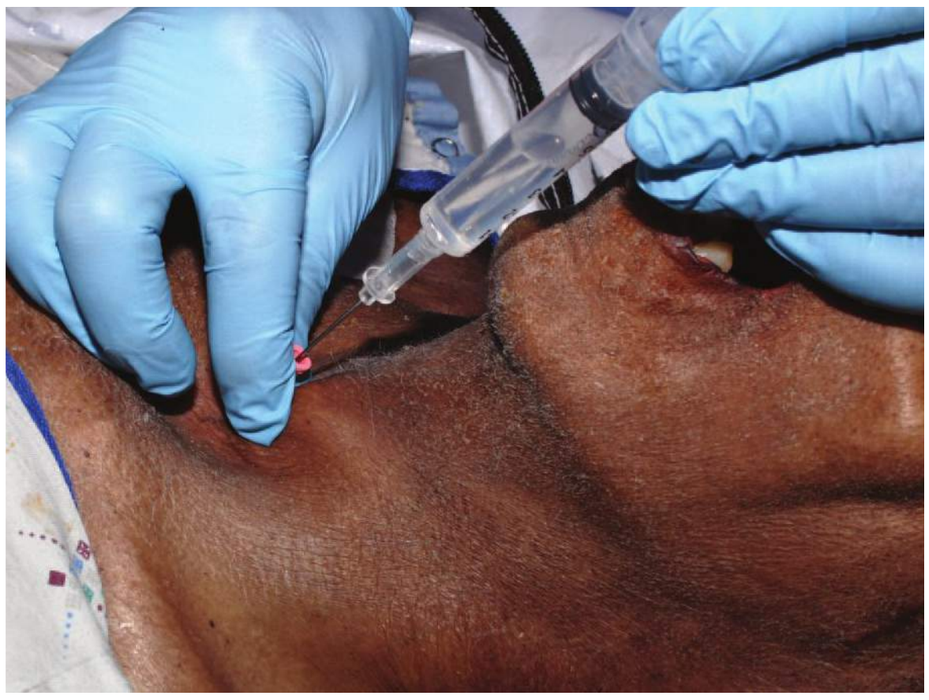


FIGURE 22.78 ■ **Slide Catheter Into Trachea.** Carefully slide the catheter off the needle into the trachea. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.79 ■ **Confirm Tracheal Placement.** Again, aspirate bubbles from the catheter to confirm tracheal placement. (Photo contributor: Lawrence B. Stack, MD.)

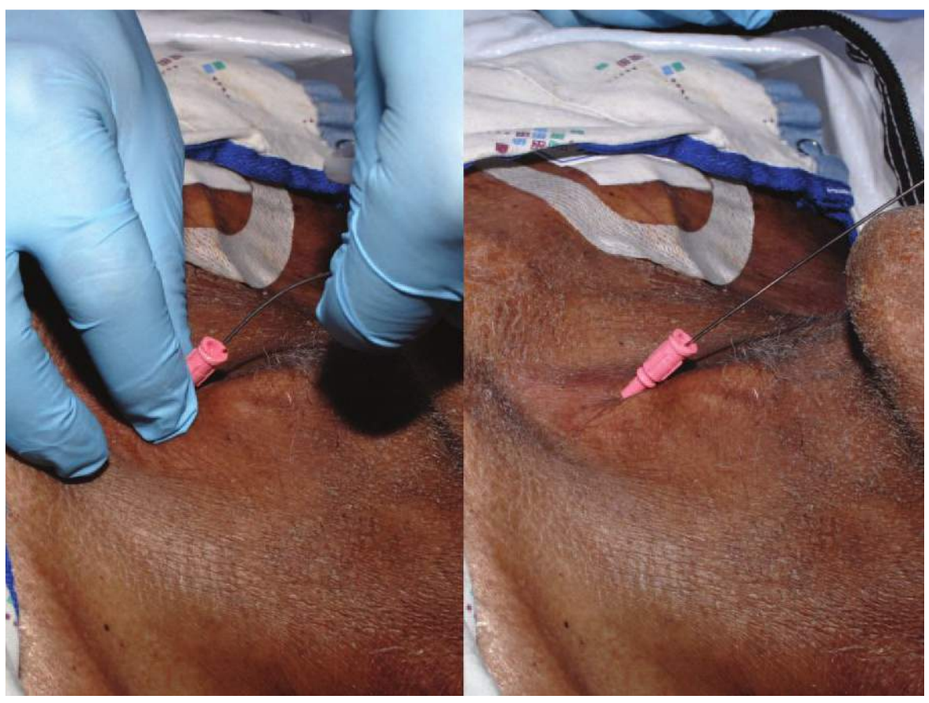


FIGURE 22.80 ■ **Thread Wire.** Lack of resistance during threading of Seldinger wire confirms tracheal placement. (Photo contributor: Lawrence B. Stack, MD.)

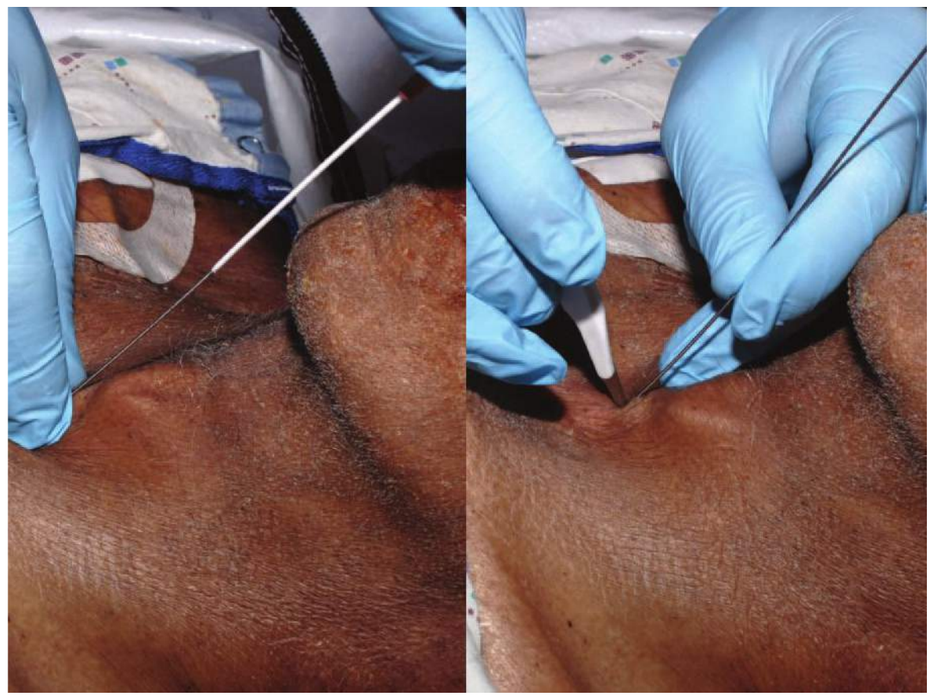


FIGURE 22.81 ■ **Incision at Wire Entry Site.** Remove catheter and make incision at the wire entry site. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.82 ■ **Insert Tracheostomy Tube/Introducer.** Insert both devices over the wire and into the trachea. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.83 ■ **Remove Wire and Introducer.** Remove these devices together as a single unit. (Photo contributor: Lawrence B. Stack, MD.)

Technique

1. Identify the thyroid cartilage and the cricothyroid membrane directly caudally.
2. Cleanse skin surface with appropriate antibacterial medication.
3. Anesthetize skin surface with 1% lidocaine.
4. While stabilizing the trachea with the gloved nondominant hand, make a vertical incision through the skin overlying the cricothyroid membrane.
5. Dissect the tissues over the cricothyroid membrane in a horizontal direction, until the cricothyroid membrane is exposed.
6. Incise the cricothyroid membrane horizontally and insert a tracheal hook to stabilize and control the trachea.
7. If a tracheal dilator is available, insert it into the trachea to dilate and control the trachea to facilitate tube insertion.
8. Insert a cuffed tracheostomy tube, or a 6.0 ID ETT.
9. Inflate cuff and initiate ventilation.

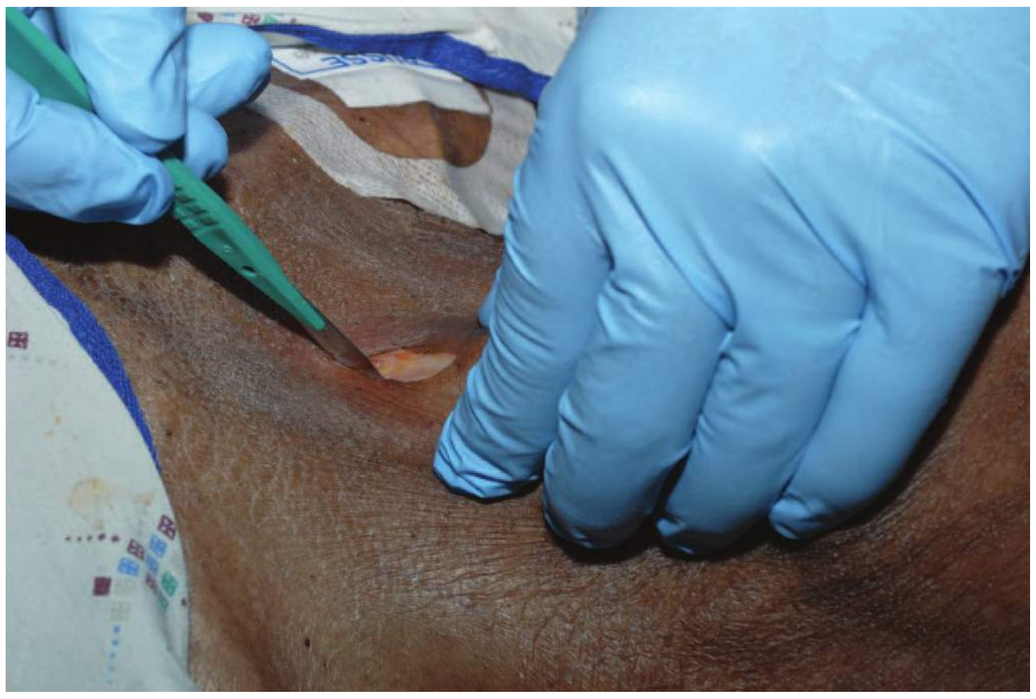


FIGURE 22.84 ■ **Incision over Cricothyroid Membrane.** Stabilize the proximal trachea, make a vertical incision over the cricothyroid membrane. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.85 ■ **Horizontal Incision through Cricothyroid Membrane.** Insert a tracheal hook through the incision to gain proximal control of the trachea. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 22.86 ■ **Insert Tracheal Dilator.** Rotate dilator cephalad. (Photo contributor: Lawrence B. Stack, MD.)

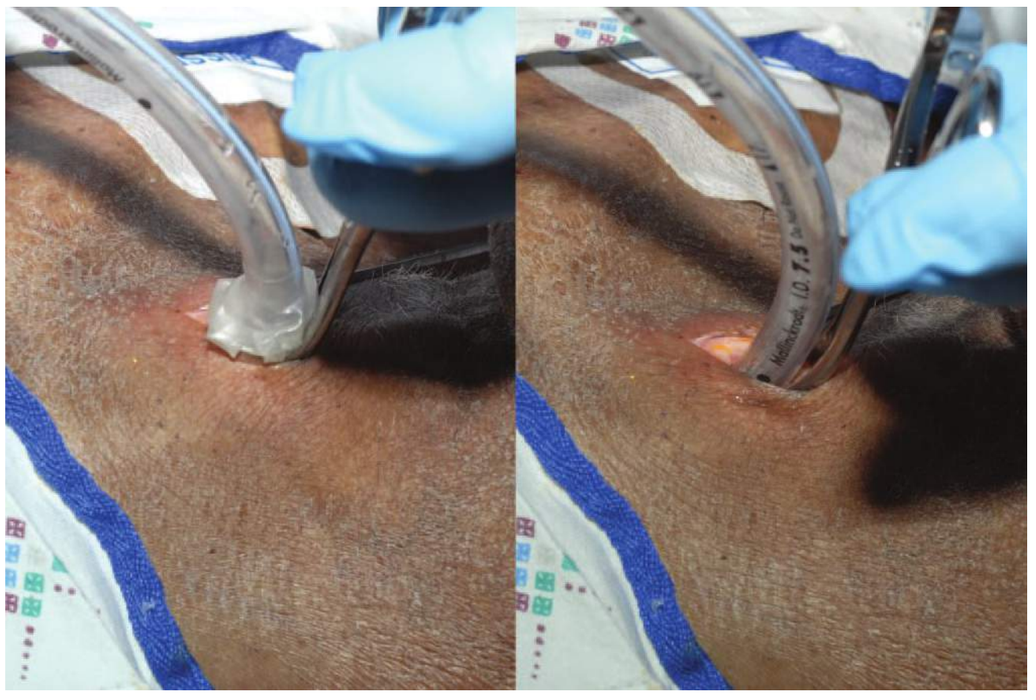
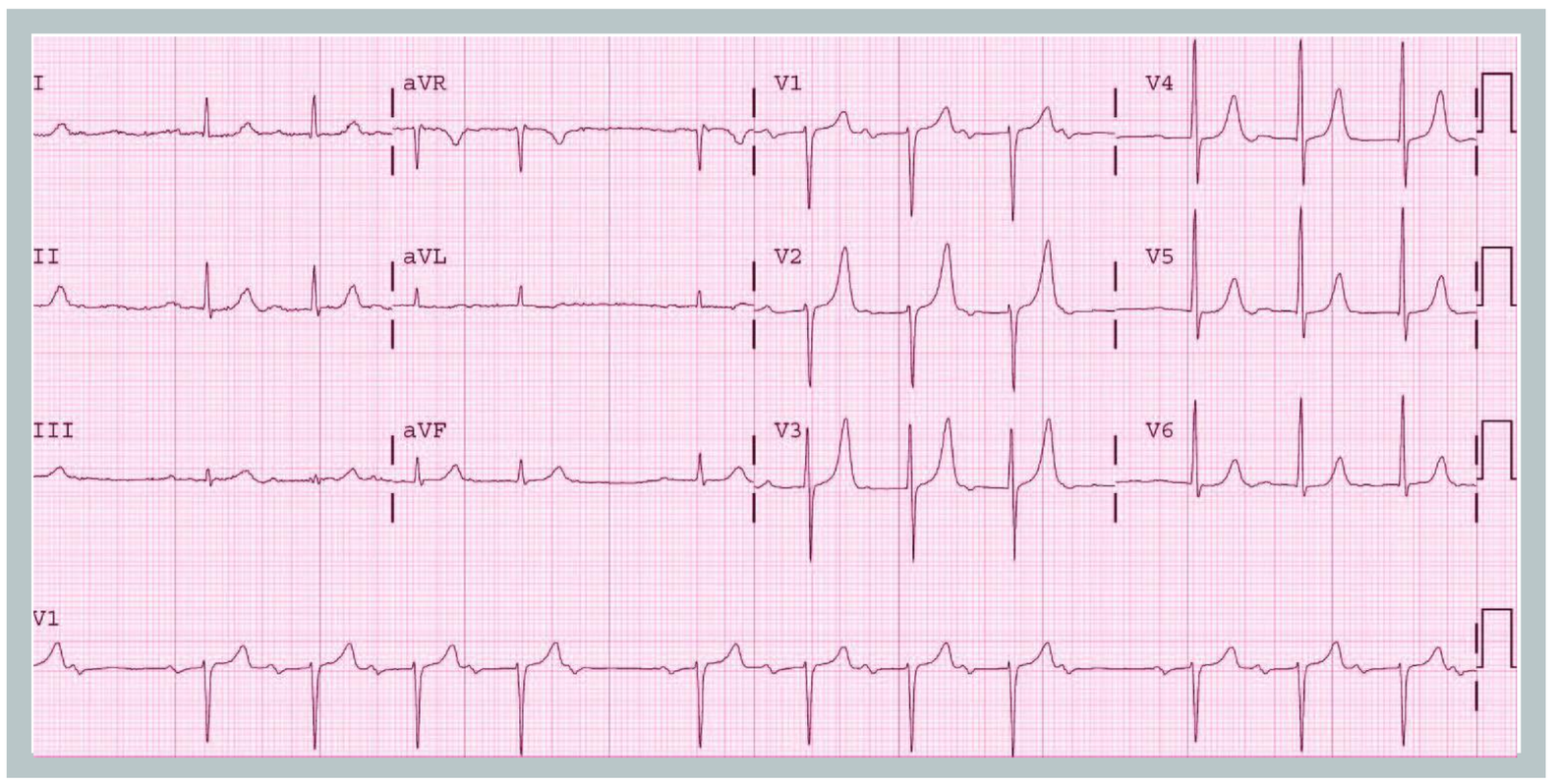


FIGURE 22.87 ■ **Insert ET or Tracheostomy Tube.** Insert a cuffed ETT or tracheostomy tube and remove dilator. (Photo contributor: Lawrence B. Stack, MD.)

Chapter 23

ECG ABNORMALITIES

James V. Ritchie
Michael L. Juliano
R. Jason Thurman



Second-Degree AV Block (Mobitz I, Wenckebach). The PR interval gradually increases until a P wave is not followed by a QRS and a beat is "dropped." The process then recurs. P waves occur at regular intervals, though they may be hidden by T waves. (ECG contributor: James Paul Brewer, MD.)

ACUTE ANTERIOR MYOCARDIAL INFARCTION

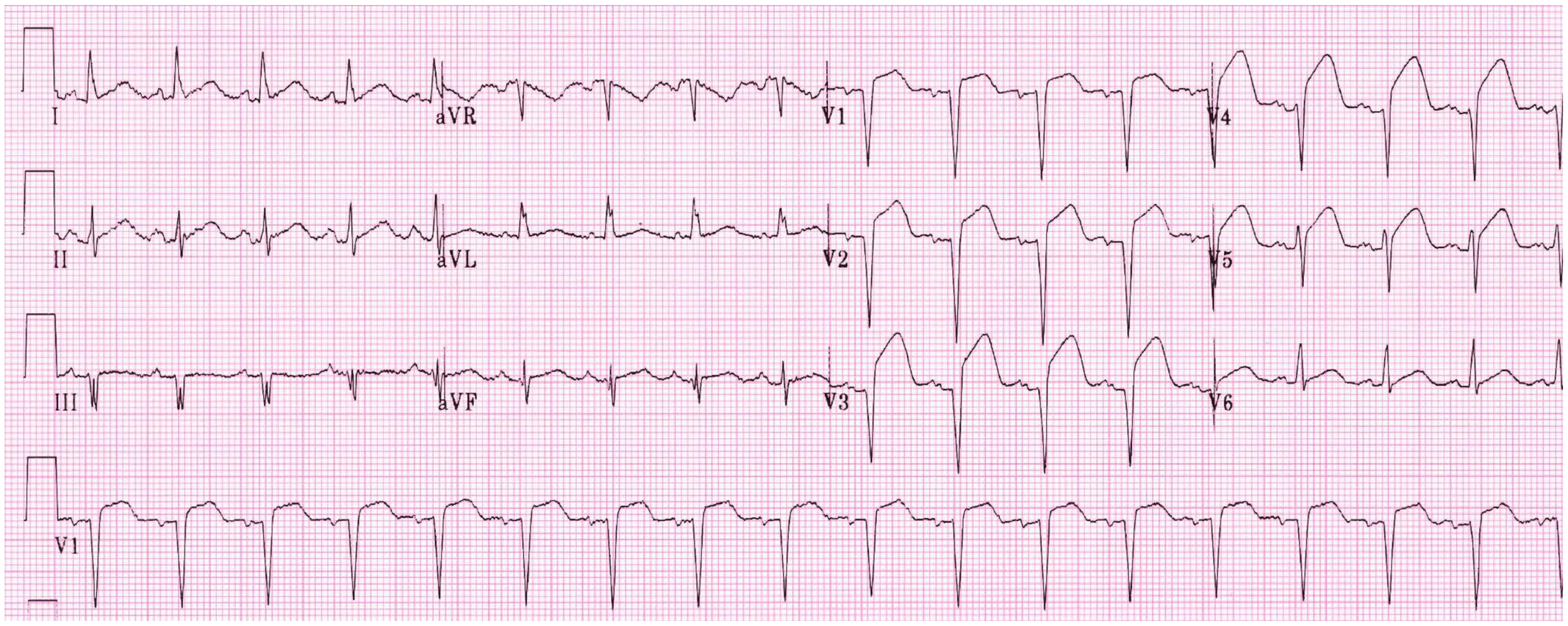


FIGURE 23.1A ■ **Acute Anteroseptal Myocardial Infarction.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- ST segment elevation in the anterior precordial leads.
- V_1-V_4: Anteroseptal injury.
- V_3-V_4: Anterior injury.
- V_3-V_6: Anterolateral injury. Leads I and aVL may also be involved, especially if the circumflex artery is affected (high lateral injury).
- Reciprocal ST segment depressions are often present in the inferior leads (II, III, aVF).

Pearls

1. The left anterior descending artery supplies blood to the anterior and lateral left ventricle and ventricular septum.
2. Normal R-wave progression (increasing upward amplitude with R wave > S wave at V_3 or V_4) may be interrupted.
3. The development of pathologic Q waves in any of the V leads other than V_1 strongly suggests that the injury has progressed to an infarction, as seen in this example.



FIGURE 23.1B ■ Pathologic ST-segment elevation beyond 1 mm (double arrow) with pathologic Q waves (arrow) in lead V_3. The ST segment demonstrates a convex upward, or "tombstone," morphology.

ACUTE INFERIOR MYOCARDIAL INFARCTION

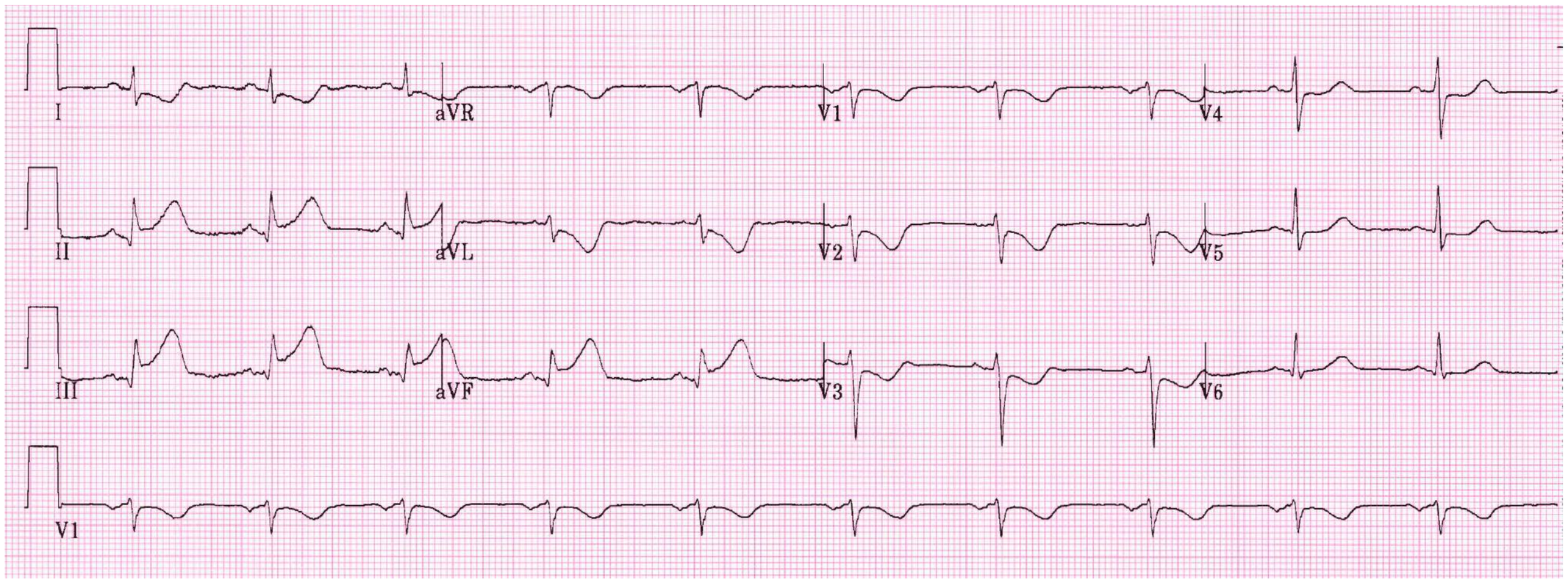


FIGURE 23.2A ■ **Acute Inferior-Posterior Myocardial Infarction.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- ST segment elevation in inferior leads (II, III, aVF)
- ST segment depressions in the anterior leads (V_1-V_3) and possibly high lateral leads (I, aVL)

Pearls

1. The right coronary artery supplies blood to the right ventricle, the sinoatrial (SA) node, the inferior portions of the left ventricle, and usually to the posterior portion of the left ventricle and the atrioventricular (AV) node.
2. Infarctions involving the SA node may produce sinus dysrhythmias including tachycardias, bradycardias, and sinus arrest.
3. Infarctions involving the AV node may produce AV blocks.
4. In the presence of acute inferior injury, a right-sided ECG should be obtained to look for right ventricular involvement. The administration of nitroglycerin in the presence of acute right ventricular infarction can precipitate profound hypotension, as these patients are preload-dependent.
5. Since the right coronary artery so often supplies the posterior left ventricle, look for evidence of a posterior infarction (as present in the example) and consider obtaining an ECG with posterior leads.

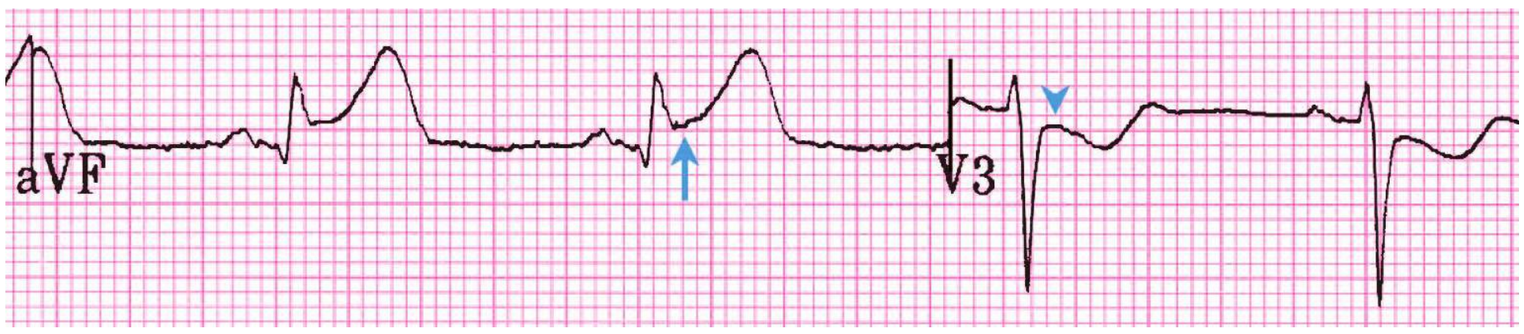


FIGURE 23.2B ■ ST-segment elevation is present in the inferior leads (II, III, aVF) (arrow), with reciprocal ST depression in the anterior leads (V_2-V_4) (arrowhead) and high lateral leads (I, aVL).

ACUTE RIGHT VENTRICULAR MYOCARDIAL INFARCTION

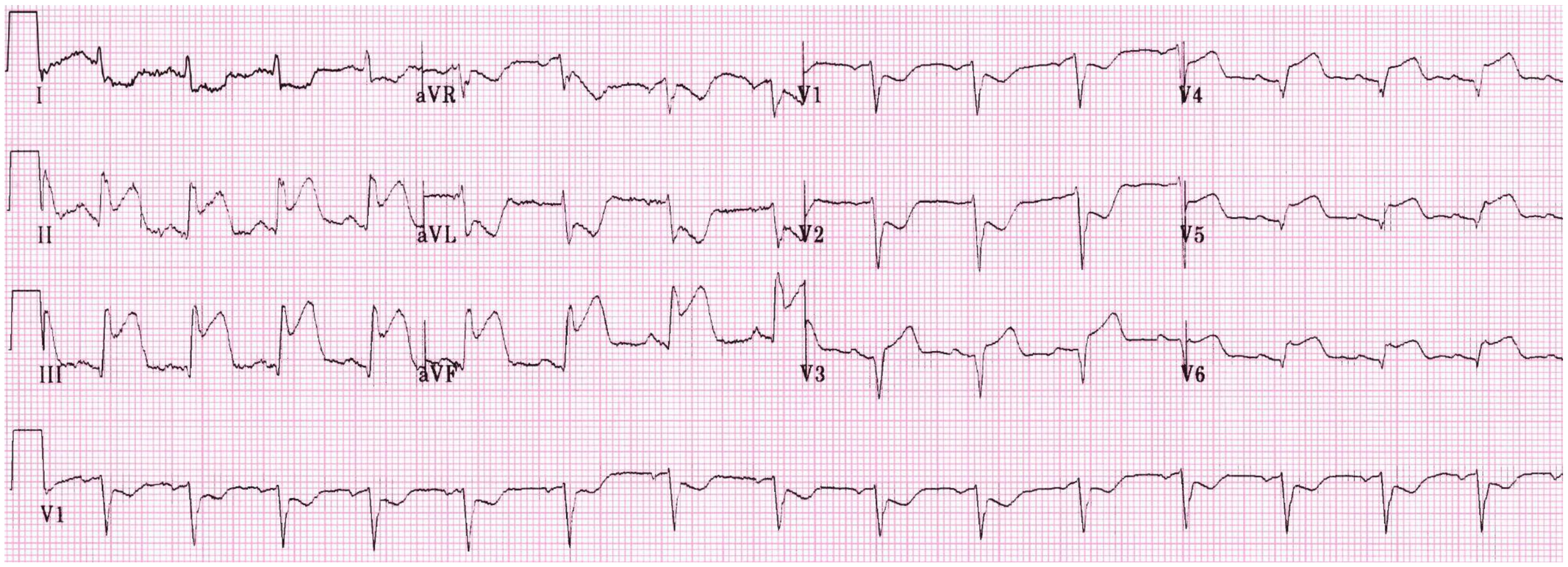


FIGURE 23.3A ■ **Right Ventricular Myocardial Infarction.** This ECG was obtained with right-sided lead placement. (ECG contributor: Thomas Bottoni, MD.)

ECG Findings

- ST elevation in right-sided V leads (V_4R, V_5R).
- ST elevation greater in lead III than lead II suggests RV MI.
- ST elevation in the normally obtained V_1 also strongly suggests RV MI.
- Often associated with inferior MI and/or posterior MI.

Pearls

1. The smaller muscle mass of the right ventricle produces a less intense injury pattern that is overwhelmed by the left ventricle in the normally obtained ECG. Placement of right-sided V leads, with V_1-V_6 in mirror-image locations on the right side of the chest, is important in detecting right ventricular injury.
2. The heart with an injured right ventricle is very preload-dependent. Beware of lowering preload with nitrates in any patient with suspected RV MI as severe hypotension may occur. Treat hypotension with volume.
3. Obtain a right-sided ECG in any patient with inferior or posterior MI, and in any patient with a significant hypotensive response to nitrates.

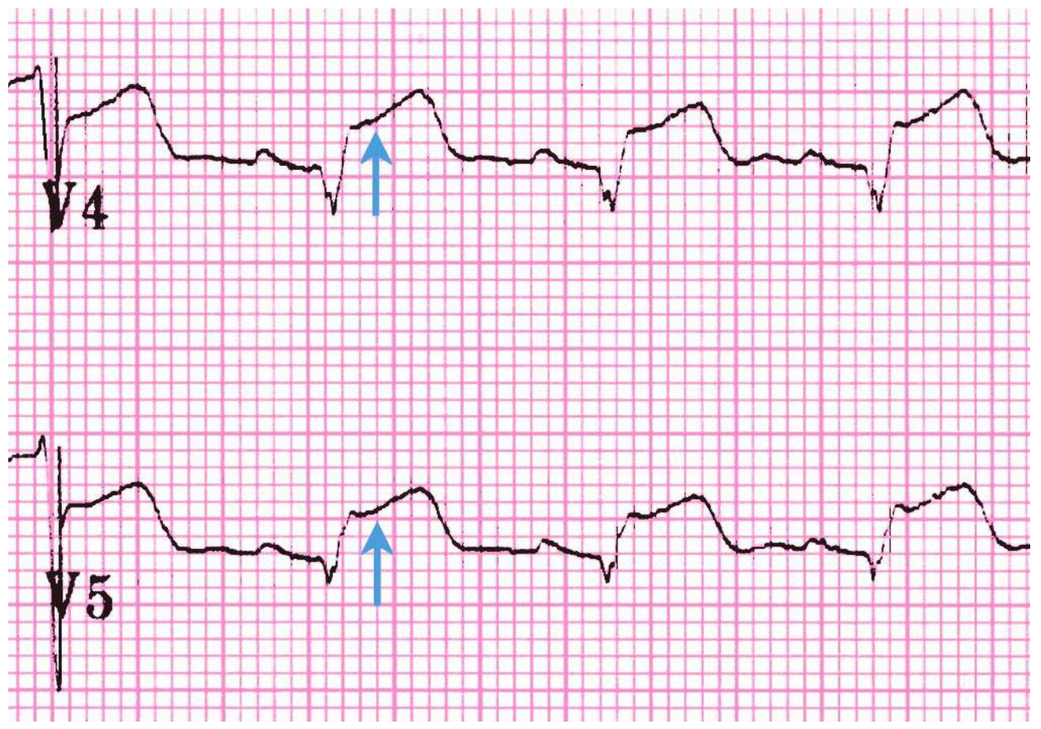


FIGURE 23.3B ■ ST elevation in V_4R and V_5R (arrows), with the V_4 and V_5 leads placed in their mirror-image locations on the right side of the chest. Any ST elevation seen in the right-sided precordial leads is significant.

ACUTE POSTERIOR MYOCARDIAL INFARCTION

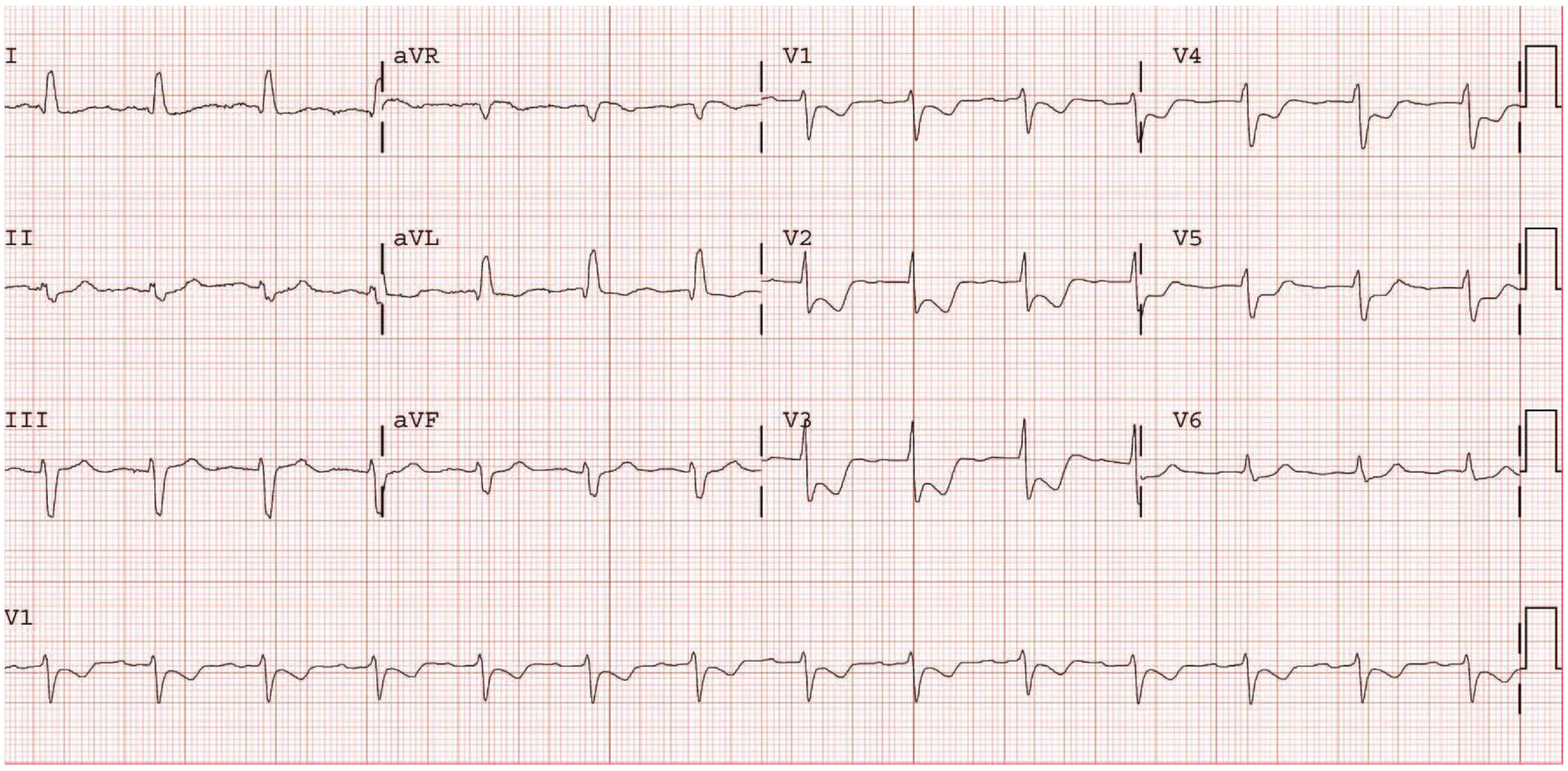


FIGURE 23.4A ■ **Acute Posterior Myocardial Infarction.** (ECG contributor: R. Jason Thurman, MD.)

ECG Findings

- With acute injury pattern—ST segment depression in lead V_1 and/or V_2 with acute injury pattern
- With infarction pattern—Small S wave and large R wave greater than 4 ms duration in lead V_1 or V_2 with infarction
- With infarction pattern—R-wave/S-wave ratio greater than 1 in lead V_1 or V_2 with infarction

Pearls

1. The posterior portion of the left ventricle has no EKG electrodes directly overlying it and is the last portion of ventricle to depolarize. It receives its blood supply from either the right coronary artery (in 85% of individuals) or the circumflex artery (in 15% of individuals).
2. V_1 and V_2 are primarily affected as the most anterior leads and indirectly assess the posterior left ventricle, though in an "inverted" orientation. Instead of observing downgoing Q waves and ST elevation, one expects to see large upgoing R waves and ST depression. By holding the EKG up to a backlight upside down and horizontally flipped, the more classic injury pattern can be observed by looking through the EKG paper (see Fig. 23.4C).
3. Posterior involvement may be confirmed with posterior leads. V_8 is located at inferior tip of left scapula; V_9 is positioned between V_8 and the spine at the same level.
4. Frequently, an inferior MI is also present with a posterior MI, since the right coronary artery serves both areas. In the above example, there is subtle ST elevation in the lateral leads, indicating posterior-lateral injury.

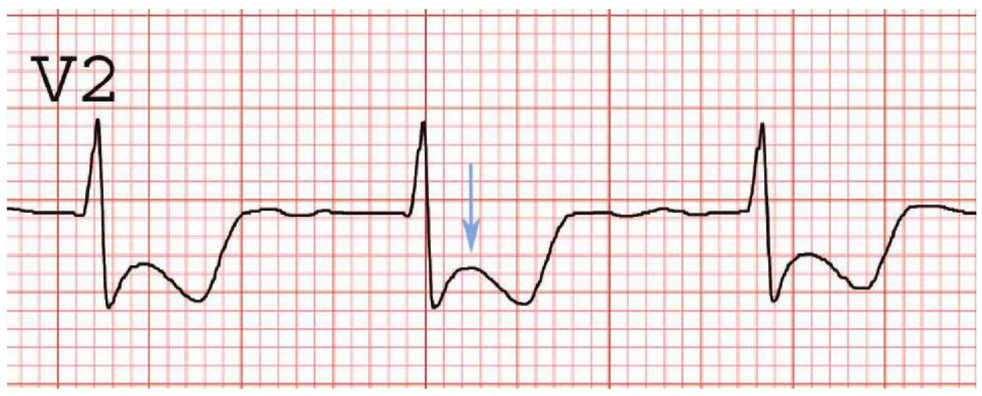


FIGURE 23.4B ■ This tracing demonstrates injury in the posterior LV, manifesting as acute ST depression in V_2 (arrow).

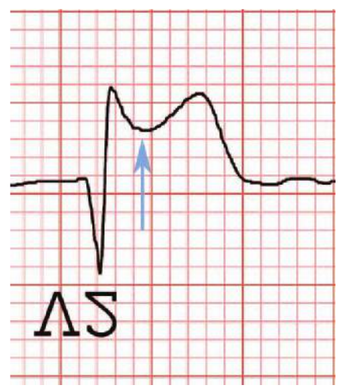


FIGURE 23.4C ■ By inverting and rotating the EKG, the characteristic ST-elevation injury pattern is easily seen (arrow). This can be done in practice by flipping the EKG upside down and looking through the printed EKG with a backlight.



FIGURE 23.4D ■ The ST depression is subtle and downsloping. However, the R-wave amplitude approximates that of the S wave, and the R-wave duration is significant (>4 ms). This is actually an "inverted Q wave" from a posterior infarction that has evolved since the initial tracing.

LEFT MAIN LESION

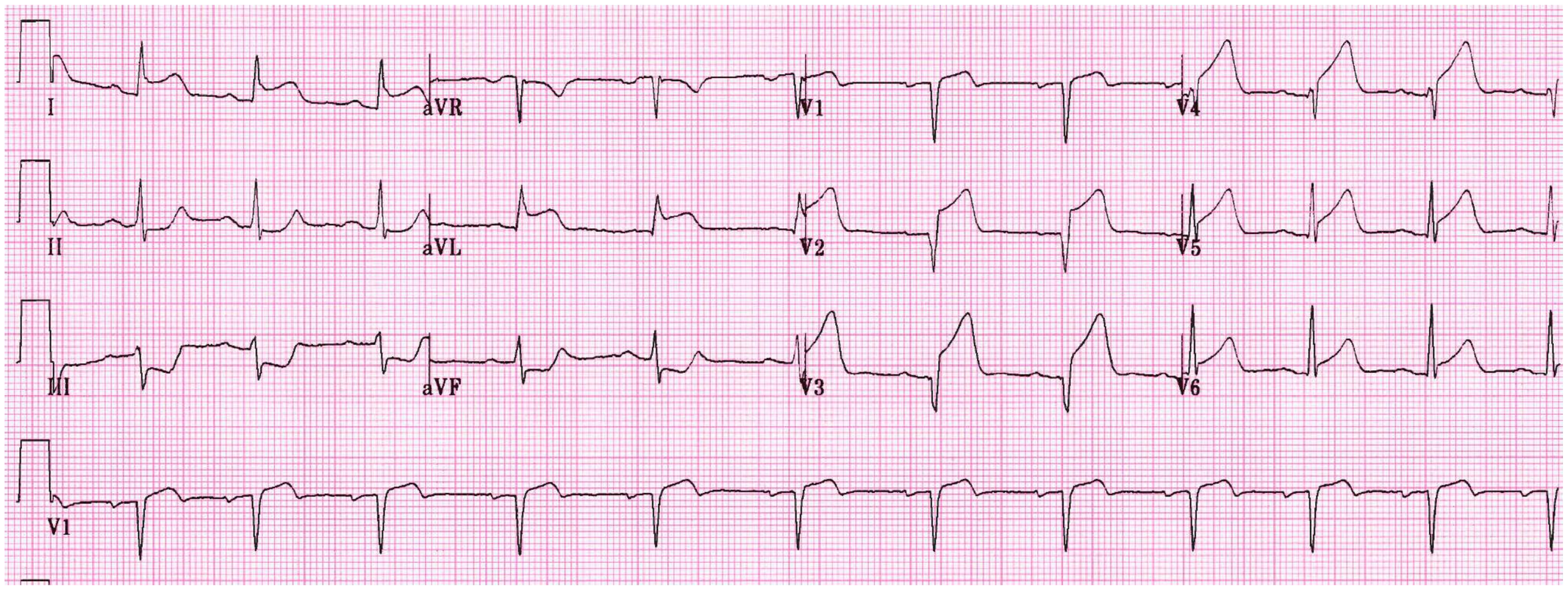


FIGURE 23.5A ■ **Left Main Coronary Artery Lesion (Widowmaker).** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- ST segment elevation in the precordial leads (V_2-V_6) and high lateral leads (I, aVL).
- Reciprocal ST segment depressions in the inferior leads (II, III, aVF).
- ST segment elevation in AVR coupled with reciprocal ST depressions may indicate AMI from left main disease.

Pearls

1. The left main coronary artery branches into the left anterior descending artery and the circumflex artery. It supplies blood to the ventricular septum and the anterior and lateral aspects of the left ventricle, usually sparing the posterior and inferior portion, which is most often served by the right coronary artery.
2. Normal R-wave progression (increasing R-wave amplitude across the precordial leads) may be interrupted.
3. Risk of cardiogenic shock is high since so much of the left ventricle is served by the left main coronary artery.
4. A left main coronary thrombosis is also known as the "widowmaker lesion."
5. Isolated ST segment elevation in lead aVR may also indicate acute myocardial ischemia from left main coronary artery disease.



FIGURE 23.5B ■ Significant ST elevation is present in the precordial leads (V_2-V_6) and high lateral leads (I and aVL) (arrows). In this example, significant Q waves have appeared, signaling infarction (arrowhead).

SGARBOSSA CRITERIA FOR AMI IN SETTING OF LBBB

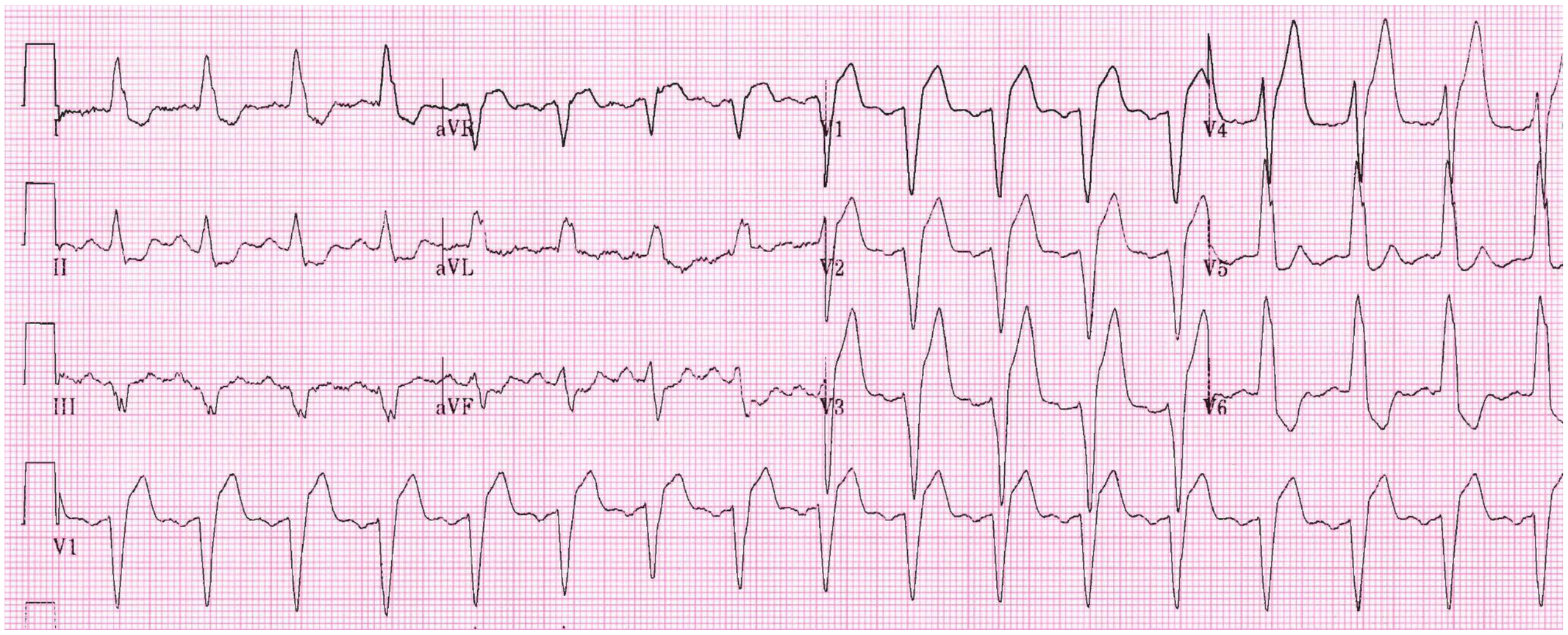


FIGURE 23.6A ■ **Acute Myocardial Infarction by Sgarbossa Criteria in the Setting of Underlying LBBB.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- ST elevation greater than or equal to 1 mm concordant with QRS deflection (score = 5)
- ST depression greater than or equal to 1 mm in leads V_1, V_2, V_3 concordant with QRS deflection (score = 3)
- ST elevation greater than or equal to 5 mm discordant with QRS deflection (score = 2)

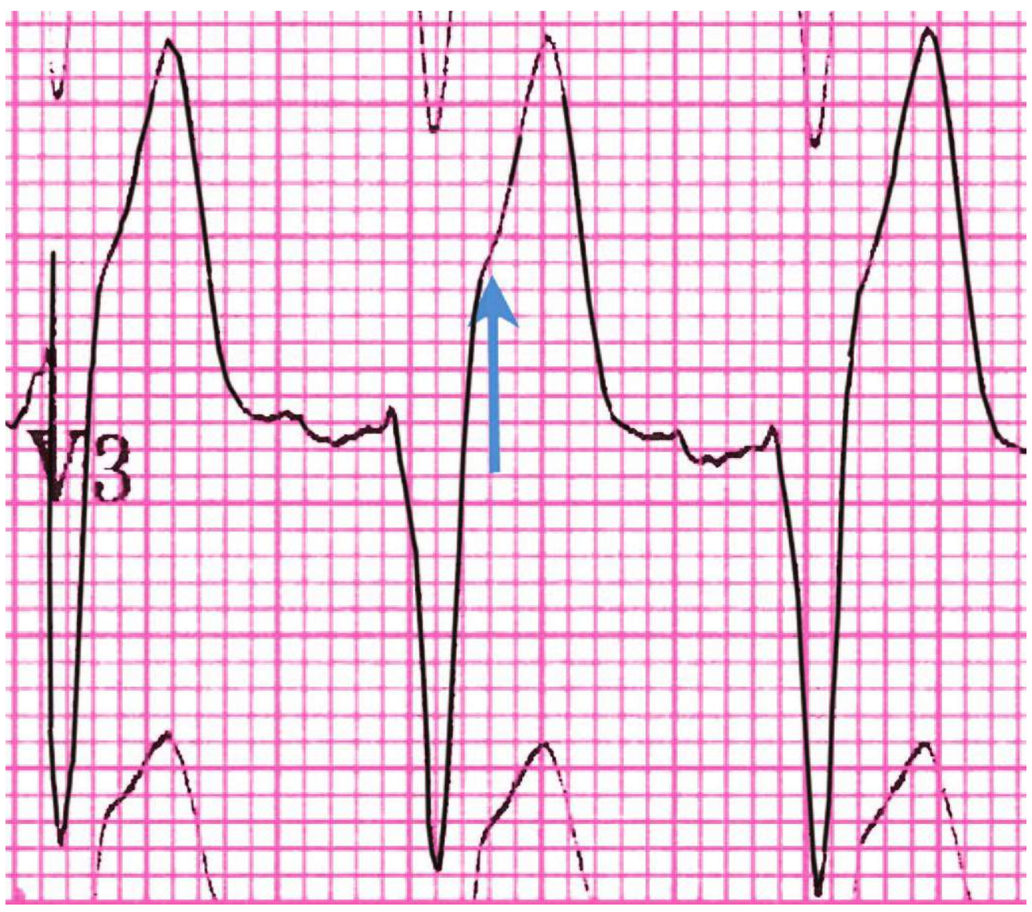


FIGURE 23.6B ■ The ST elevation is greater than 5 mm discordant from the primary QRS deflection (arrow).

Pearls

1. These scored criteria may be used to diagnose acute myocardial infarction in the setting of a left bundle branch block (LBBB). However, most myocardial ischemia in the setting of LBBB does not produce these changes. An absence of these findings should not be used as evidence against acute coronary syndrome.
2. Score of greater than or equal to 3 gives a specificity for myocardial infarction of 90%.
3. The first and third criteria listed above may also be used in ECGs with wide QRS complexes resulting from a pacemaker or idioventricular rhythm.



FIGURE 23.6C ■ The ST depression is greater than 1 mm concordant to the primary QRS deflection (arrow).

SUBENDOCARDIAL ISCHEMIA

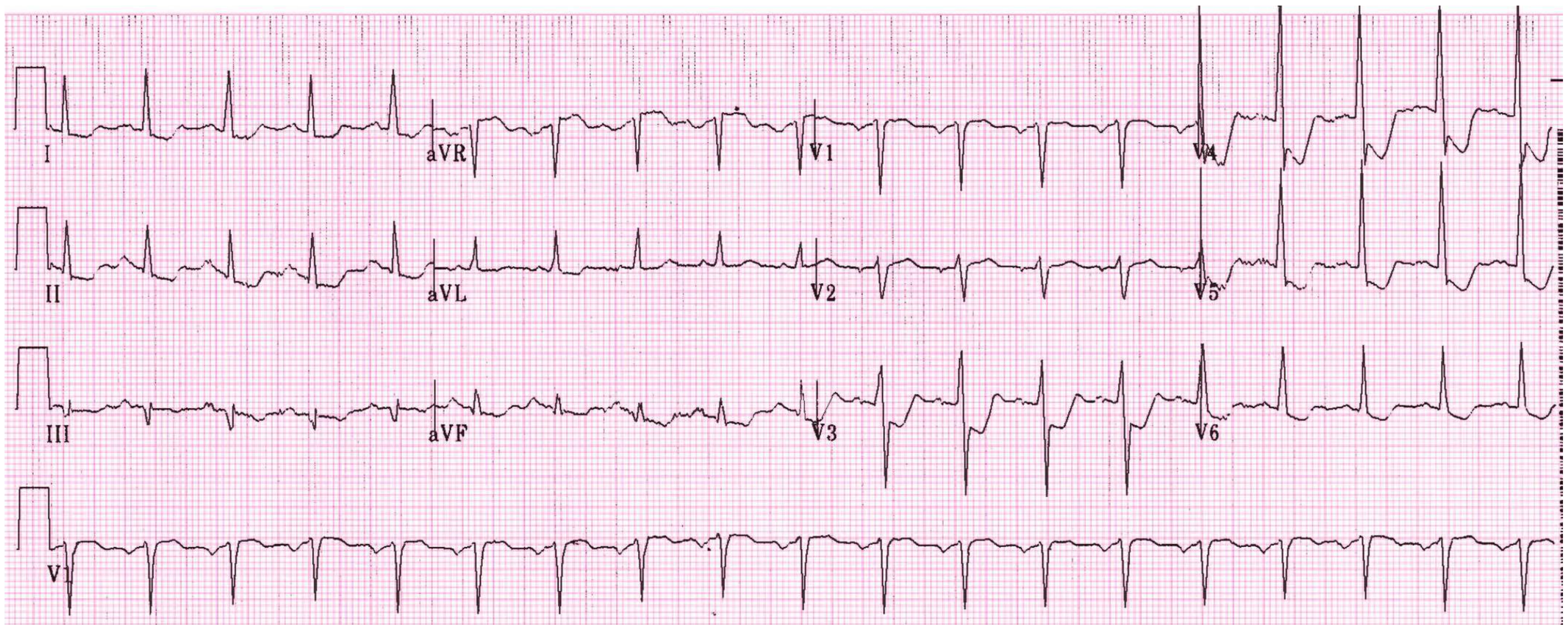


FIGURE 23.7A ■ **Subendocardial Ischemia.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- ST segment depression greater than or equal to 1 mm in anatomically adjoining leads.
- ST segments may be horizontal or downsloping with acute ischemia.

Pearls

1. Some ST depression in the lateral precordial leads (V_4-V_6) is common at higher heart rates, and is commonly seen during exercise treadmill tests, but such depression should not be downsloping unless ischemia is also present.
2. ST elevation in other leads suggests that the depression may represent reciprocal changes from acute injury rather than subendocardial ischemia.
3. Downsloping ST depression may also be seen in left ventricular hypertrophy (LVH), but this depression should not be dynamic, and should be stable with serial ECGs. ST depression from ischemia will be dynamic, changing with time on serial ECGs.
4. Isolated ST depression in leads V_1 and V_2 may represent posterior ischemia.

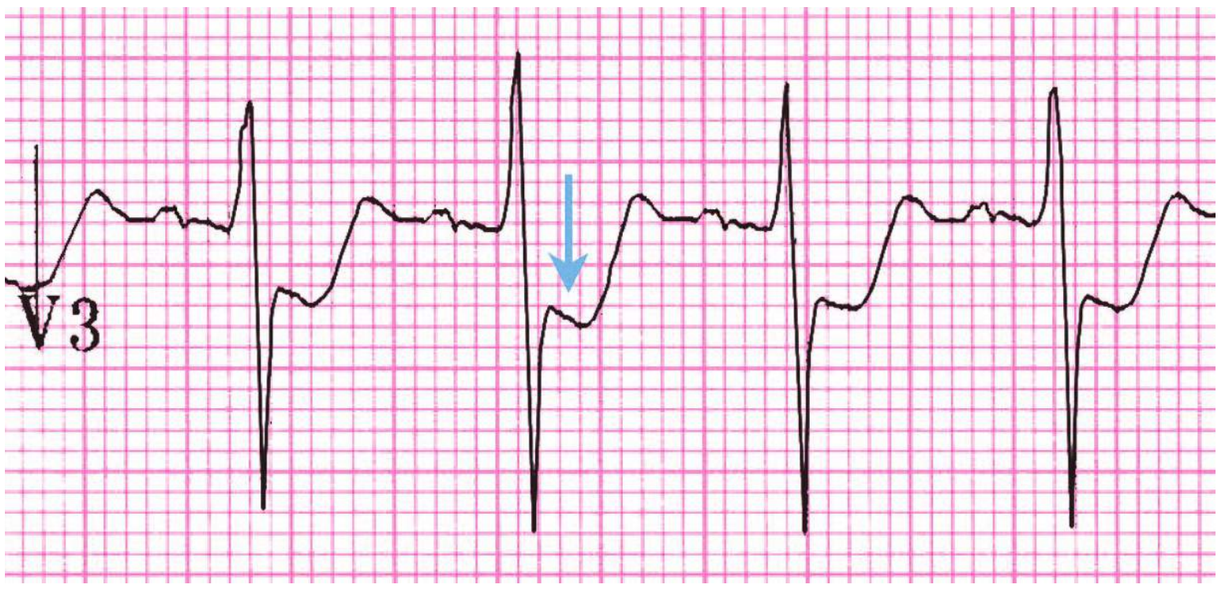


FIGURE 23.7B ■ Downsloping ST segments depressed greater than 1 mm (arrow). These changes were dynamic over time. The patient sustained a nontransmural myocardial infarction.

HYPERACUTE T WAVES

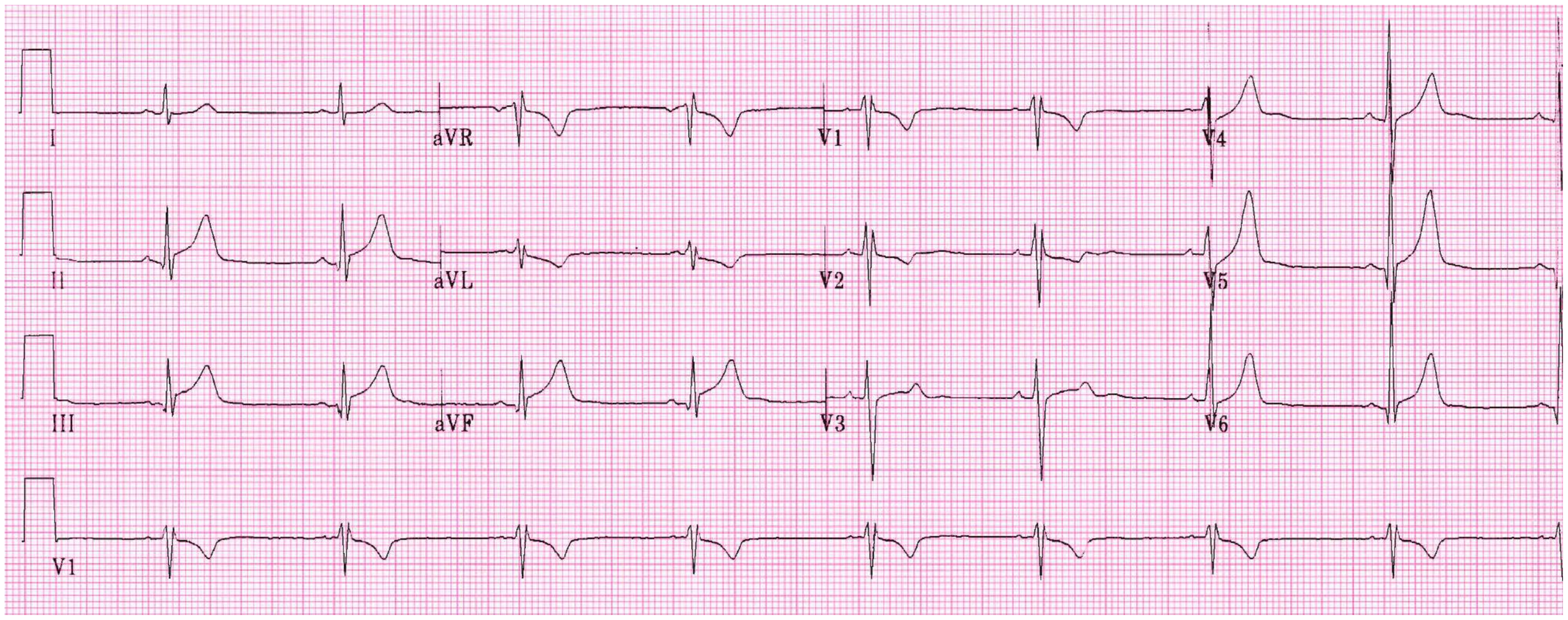


FIGURE 23.8A ■ **Hyperacute T Waves.** T waves in a patient with acute myocardial ischemia. (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- T-wave amplitude/QRS amplitude greater than 75%.
- T waves greater than 5 mV in the limb leads.
- T waves greater than 10 mV in the precordial leads.
- T waves have asymmetric appearance.



FIGURE 23.8B ■ T-wave height is greater than 10 mm in V_5 (double arrow) and is asymmetric. This height was transient and was significantly diminished in a tracing obtained 15 minutes later. Note also in the 12-lead ECG example above the presence of inferior ST elevation.

Pearls

1. Hyperacute T waves occur very early (within minutes) during myocardial injury and are transient.
2. The term "hyperacute T waves" is reserved for the early stages of myocardial infarction. "Prominent T waves" can also be seen with LVH, early repolarization, or with hyperkalemia.
3. Serial ECGs are useful in distinguishing transient hyperacute T waves from other causes of tall, peaked T waves.

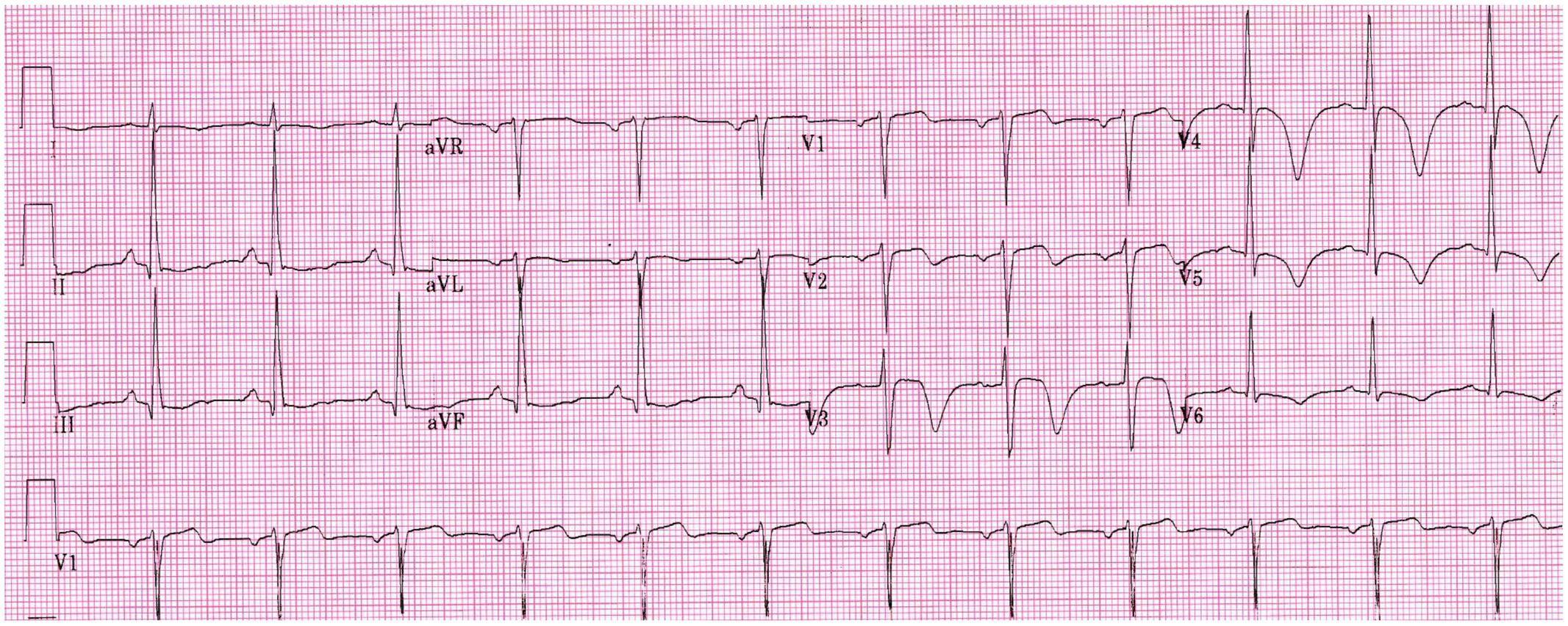


FIGURE 23.9A ■ **Wellens Waves.** Wellens waves are present and are indicative of a high-grade LAD lesion. (ECG contributor: James V. Ritchie, MD.)

ECG Findings (Two Classic Types)

- Biphasic T waves in anterior and/or lateral leads
- Deeply inverted, symmetrical T waves in the same leads

Pearls

1. These characteristic patterns of T-wave changes are closely associated with critical left anterior descending artery stenosis.
2. The changes are classically apparent on ECG after resolution of chest pain.
3. These changes are transient, and often are not associated with cardiac enzyme elevations.
4. Wellens waves are not associated with changes in R-wave progression.
5. Serial electrocardiograms may assist in differentiating Wellens waves from stable, nonspecific findings.

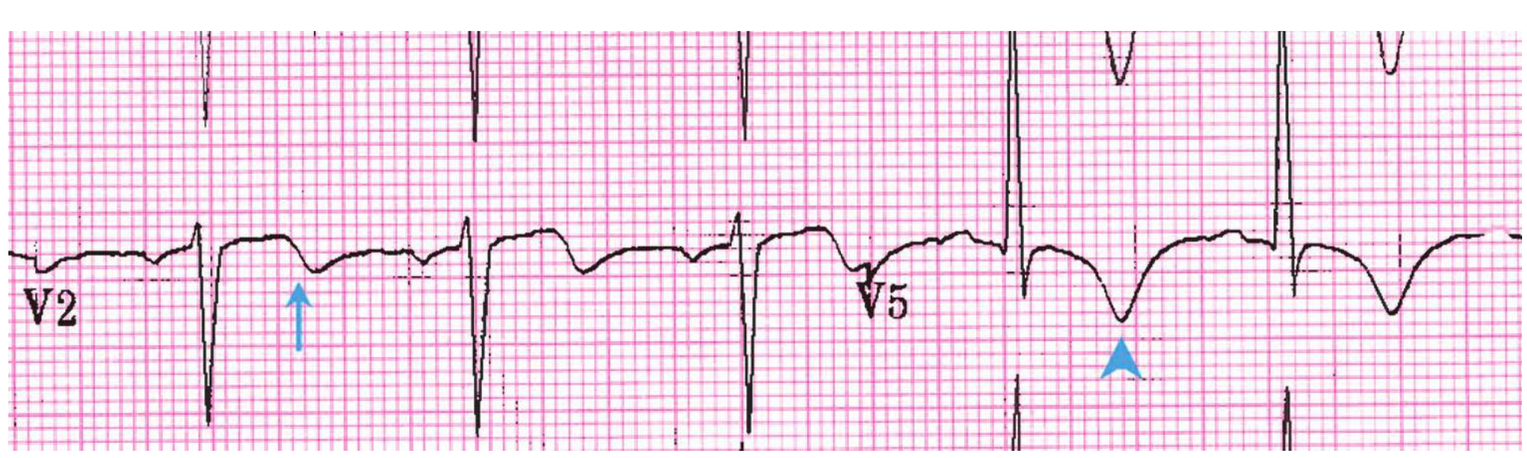


FIGURE 23.9B ■ Biphasic T waves with the later segment inverted, as in V_2 above (arrow), or deep symmetric inverted T waves, as in V_5 above (arrowhead). These findings in the precordial leads, in the setting of suspected ACS, strongly suggest an underlying high-grade LAD lesion.

"CEREBRAL" T WAVES

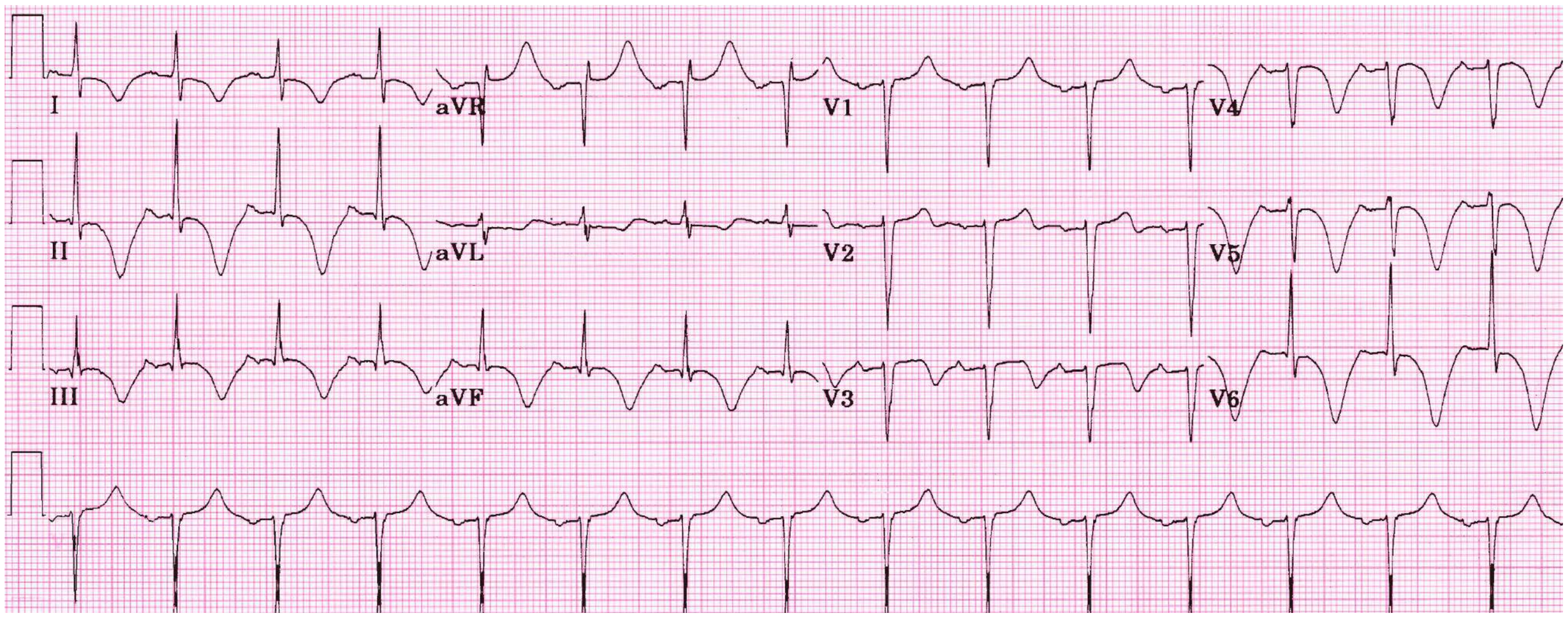


FIGURE 23.10A ■ **Cerebral T Waves.** This ECG was obtained on a patient with a severe acute hemorrhagic CVA. (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Inverted, wide T waves are most notable in precordial leads (can be seen in any lead).
- QT interval prolongation.

Pearls

1. These are associated with acute cerebral disease, most notably an ischemic cerebrovascular event or subarachnoid hemorrhage.
2. They may be accompanied by ST segment changes, U waves, and/or any rhythm abnormality.
3. Differential diagnosis includes extensive myocardial ischemia.
4. Strongly suspect an intracranial etiology in a patient with altered mental status and these electrocardiographic findings.

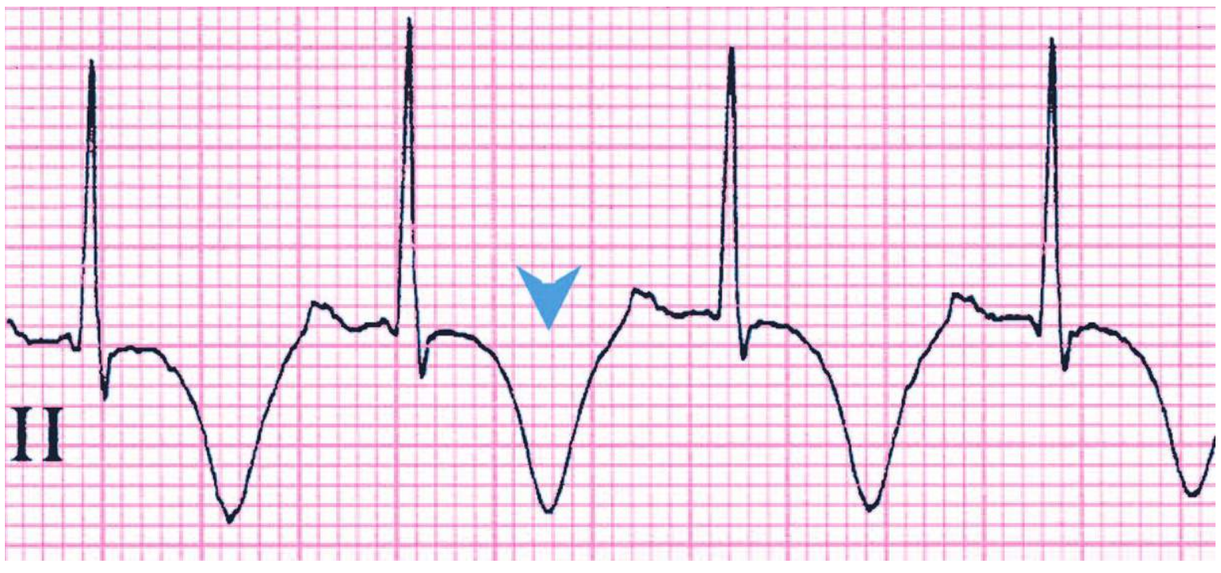


FIGURE 23.10B ■ Deep, symmetrical, inverted T waves (arrowhead) with a prolonged QT interval.

EARLY REPOLARIZATION

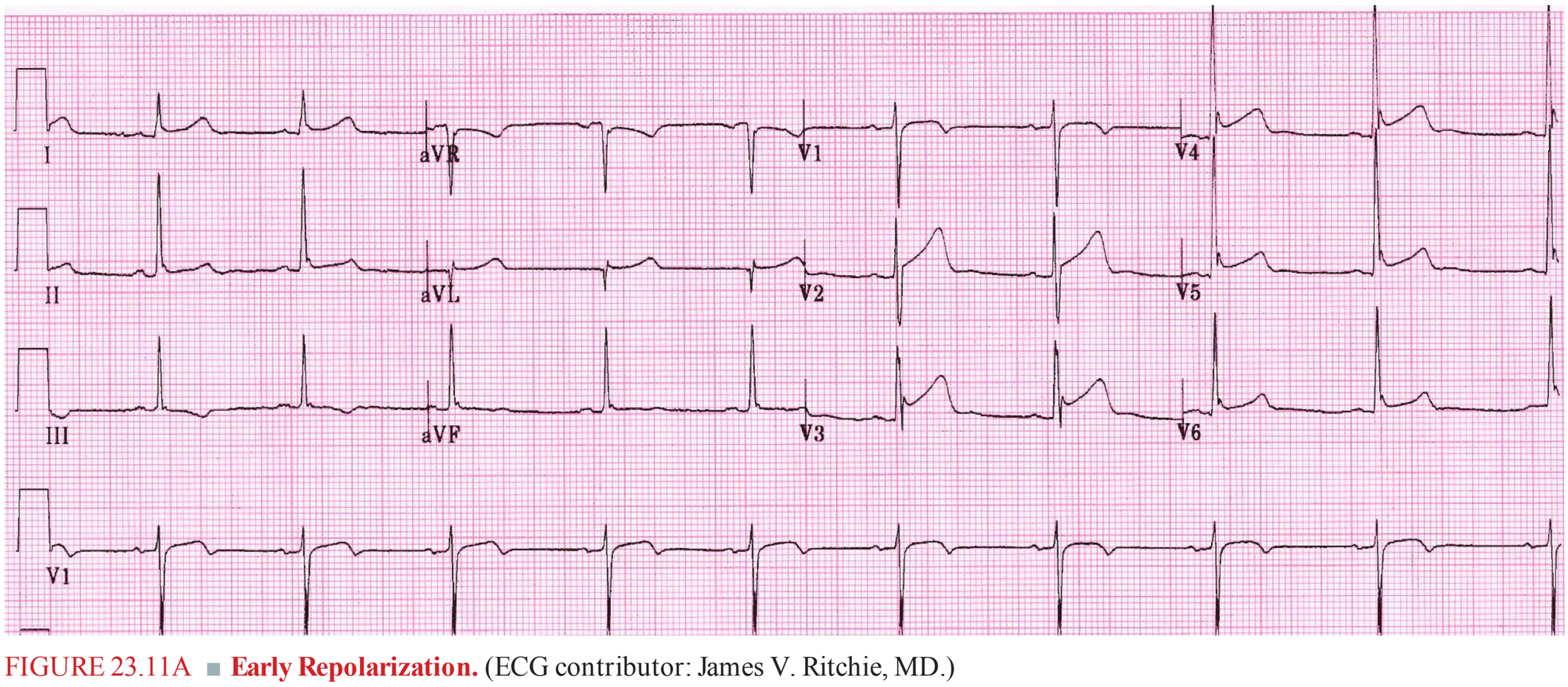


FIGURE 23.11A ■ **Early Repolarization.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- ST elevation, usually in the anterior leads.
- J-point elevation, but usually less than one-third the total height of the T wave.
- ST segment is "concave upward," or "holds water," or "is smiling at you."
- J-point notch strongly suggests early repolarization, but is not always present.

Pearls

1. This is a normal variant and is especially common in young healthy males, but also may be present in other groups. However, any clinical suspicion for ongoing myocardial ischemia should prompt further investigation
2. Q waves and reciprocal ST-segment depression in other leads should not accompany early repolarization. If present, they strongly suggest ischemia as the cause for the ST elevation.

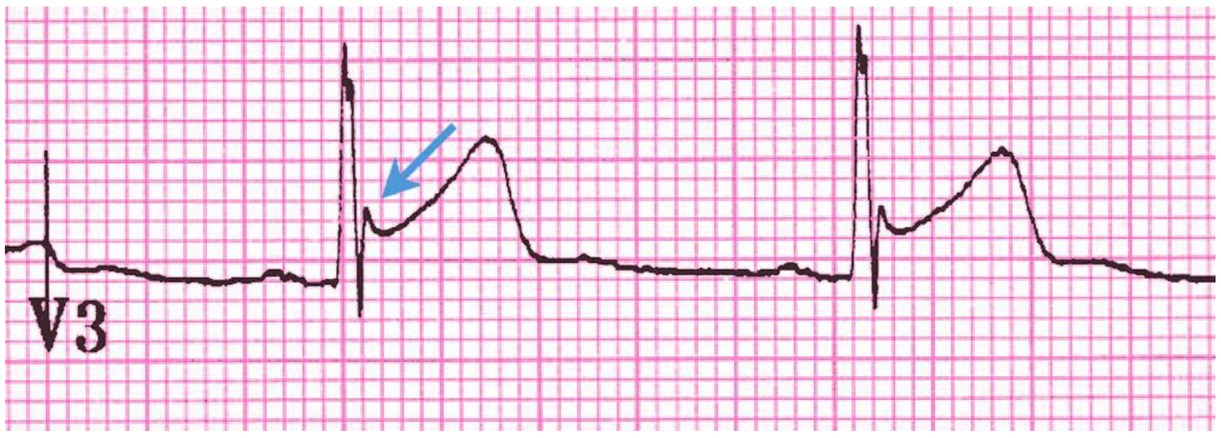


FIGURE 23.11B ■ ST elevation in precordial leads, with a concave-upward ST segment and a J-point notch (arrow).

LEFT VENTRICULAR ANEURYSM

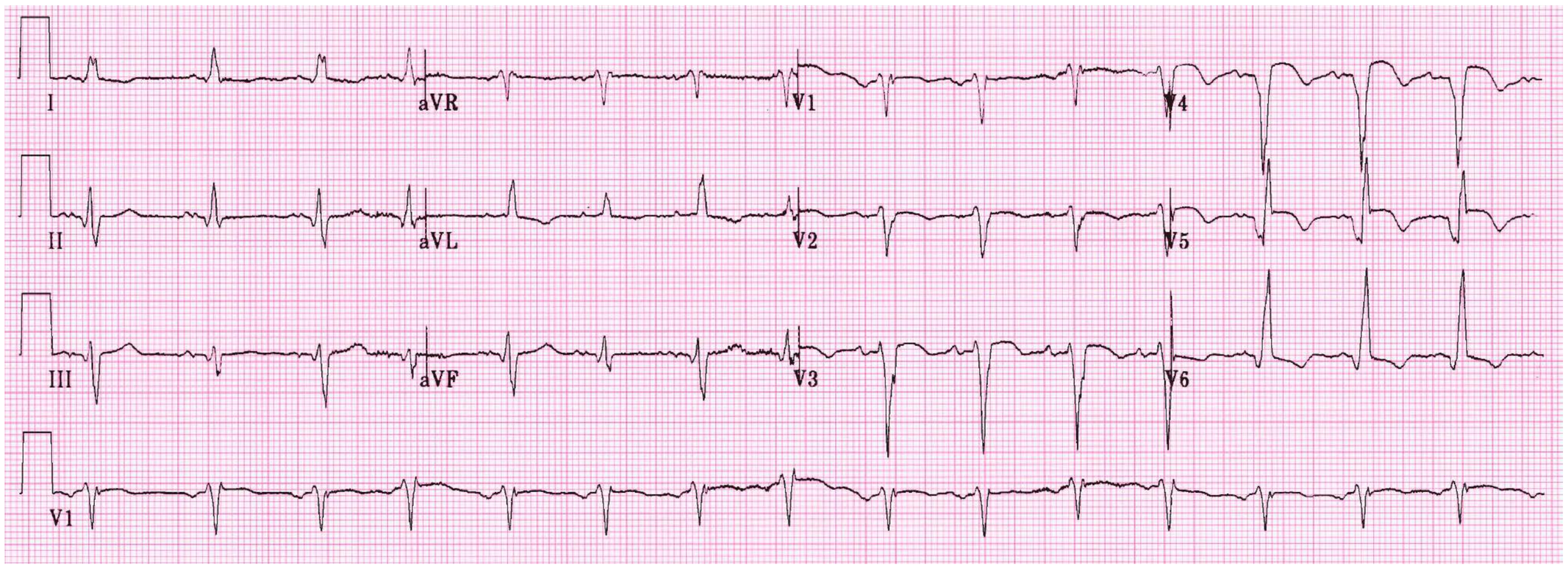


FIGURE 23.12A ■ **Left Ventricular Aneurysm.** This ECG was obtained on an asymptomatic patient with history of MI 2 years prior. (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- ST elevation in anterior contiguous leads
- Deep pathologic Q waves in anterior leads

Pearls

1. ST segment elevation which occurs in the setting of a myocardial infarction should resolve within days under normal circumstances.
2. Persistent ST-segment elevation occurring for weeks or longer after a myocardial infarction is suspicious for ventricular aneurysm.
3. Ventricular aneurysms may follow a large myocardial infarction in the anterior portion of the heart.
4. The aneurysm consists of scarred myocardium, which does not contract but bulges outward during systole; complications include congestive heart failure, myocardial rupture, arrhythmias, and thrombus formation.
5. Suspect an LV aneurysm when these findings appear in the ECG of a patient who does not demonstrate symptoms suggesting ACS. However, one should also be vigilant for the presence of "silent" ACS.



FIGURE 23.12B ■ Persistent ST elevations (arrow) and deep, pathologic Q waves (arrowhead) in an asymptomatic patient with a history of anterior myocardial infarction 2 years earlier.

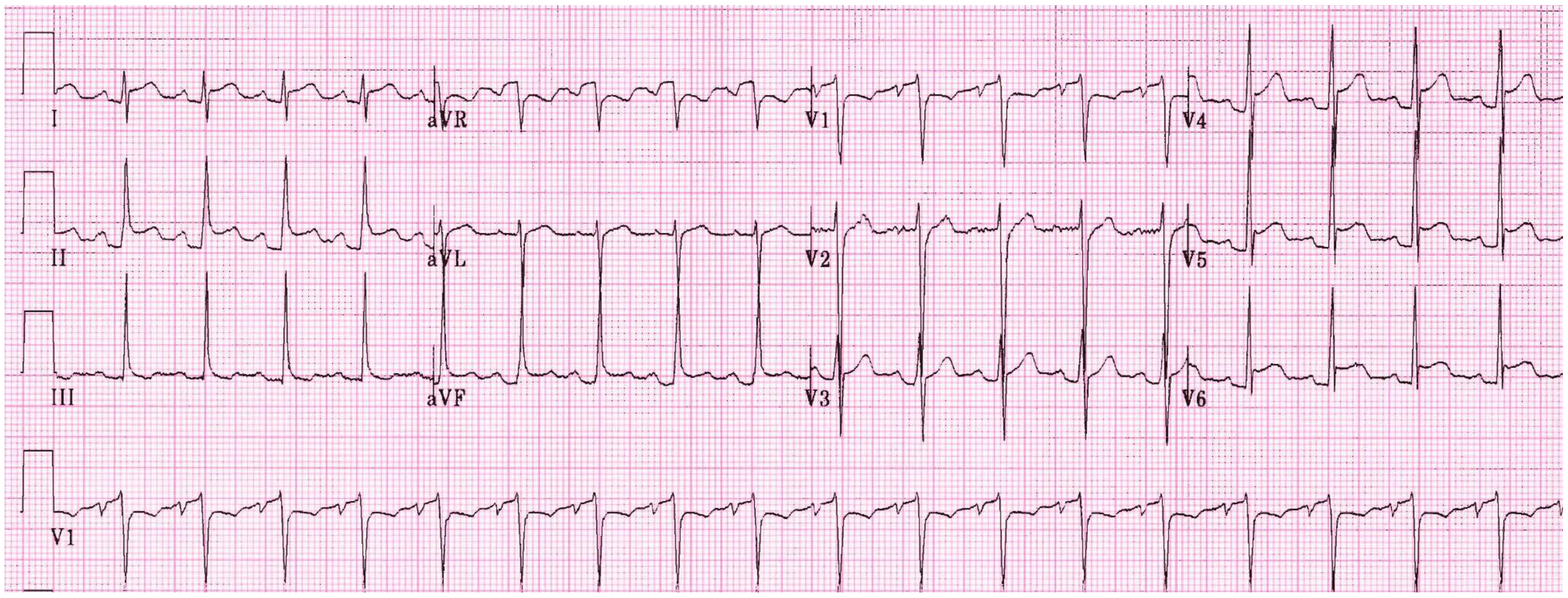


FIGURE 23.13A ■ **Acute Pericarditis.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Diffuse ST elevation in noncontiguous leads
- PR depression
- T-wave flattening or inversion
- PR elevation in lead aVR

Pearls

1. Pericarditis may produce inflammation of the epicardium. This is most often demonstrated on the ECG as a widespread injury pattern.
2. Pericarditis does not produce abnormal Q waves. The presence of abnormal Q waves must prompt consideration of acute or old coronary syndrome, including Dressler syndrome or postinfarct pericarditis.
3. Pericarditis may be focal, resulting in regional rather than diffuse EKG changes.
4. Benign early repolarization and myocarditis may also appear as ST elevation in many noncontiguous leads.

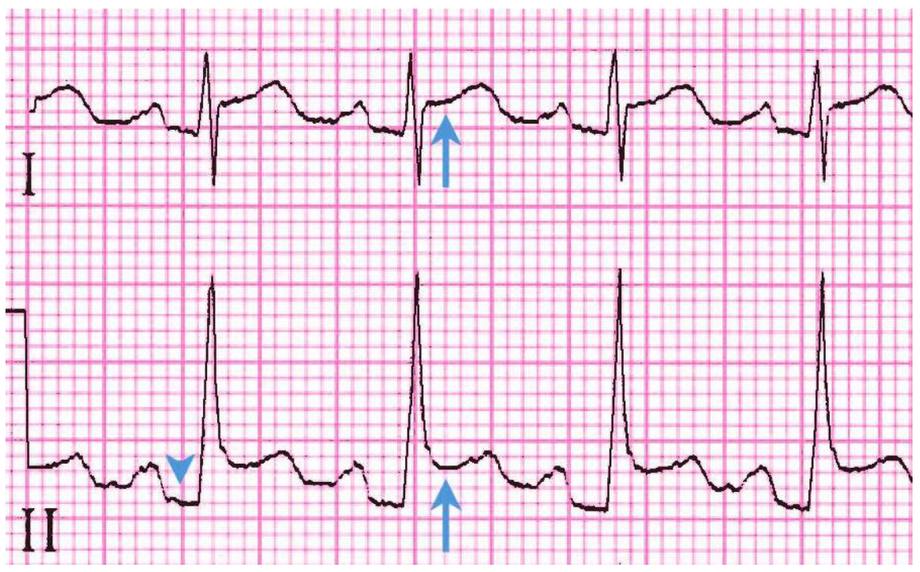


FIGURE 23.13B ■ ST elevation in noncontiguous leads I and II (arrows) with PR depression (arrowhead). No pathologic Q waves or reciprocal changes are present.

FIRST-DEGREE AV BLOCK

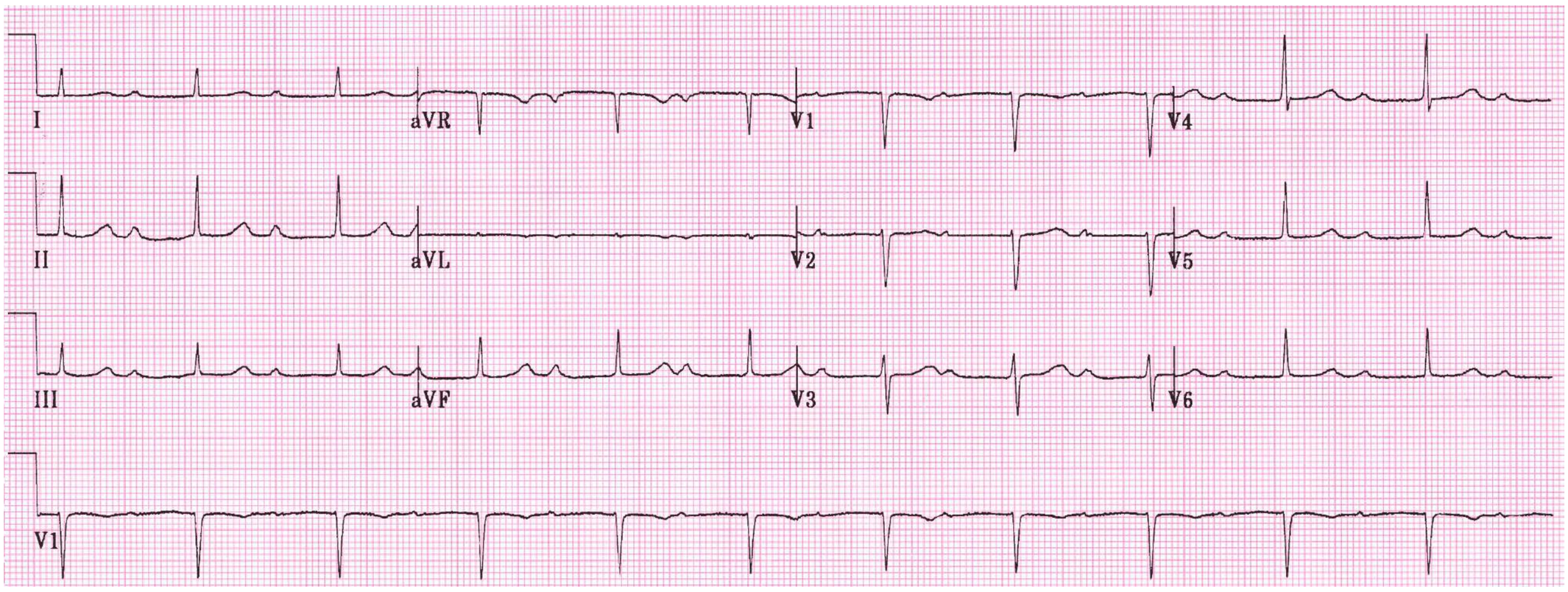


FIGURE 23.14A ■ **First-Degree AV Block.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- A PR interval greater than 200 ms (normal 120-200 ms) with no significant variation in PR intervals between beats.
- Each P wave is followed by a QRS complex.

Pearls

1. This type of heart block usually does not affect heart function and can be considered nonpathologic (especially in athletes or patients with higher vagal tone).
2. First-degree block may also be due to heart disease (myocarditis, rheumatic fever) or drugs (digoxin, amiodarone, β-blockers, calcium channel blockers).

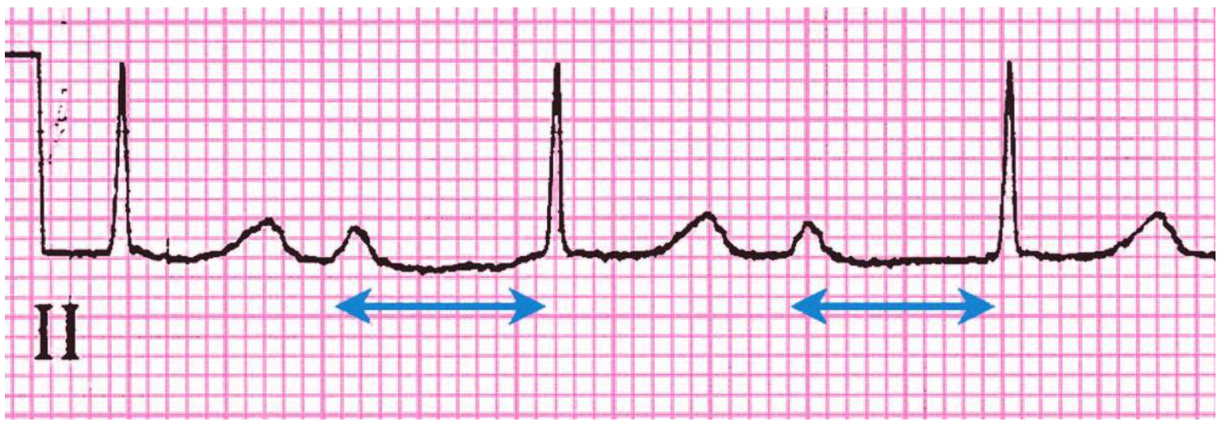


FIGURE 23.14B ■ The PR interval is fixed (double arrows) and is longer than 0.2 seconds, or five small blocks.

TYPE 1 SECOND-DEGREE AV BLOCK (MOBITZ I, WENCKEBACH)

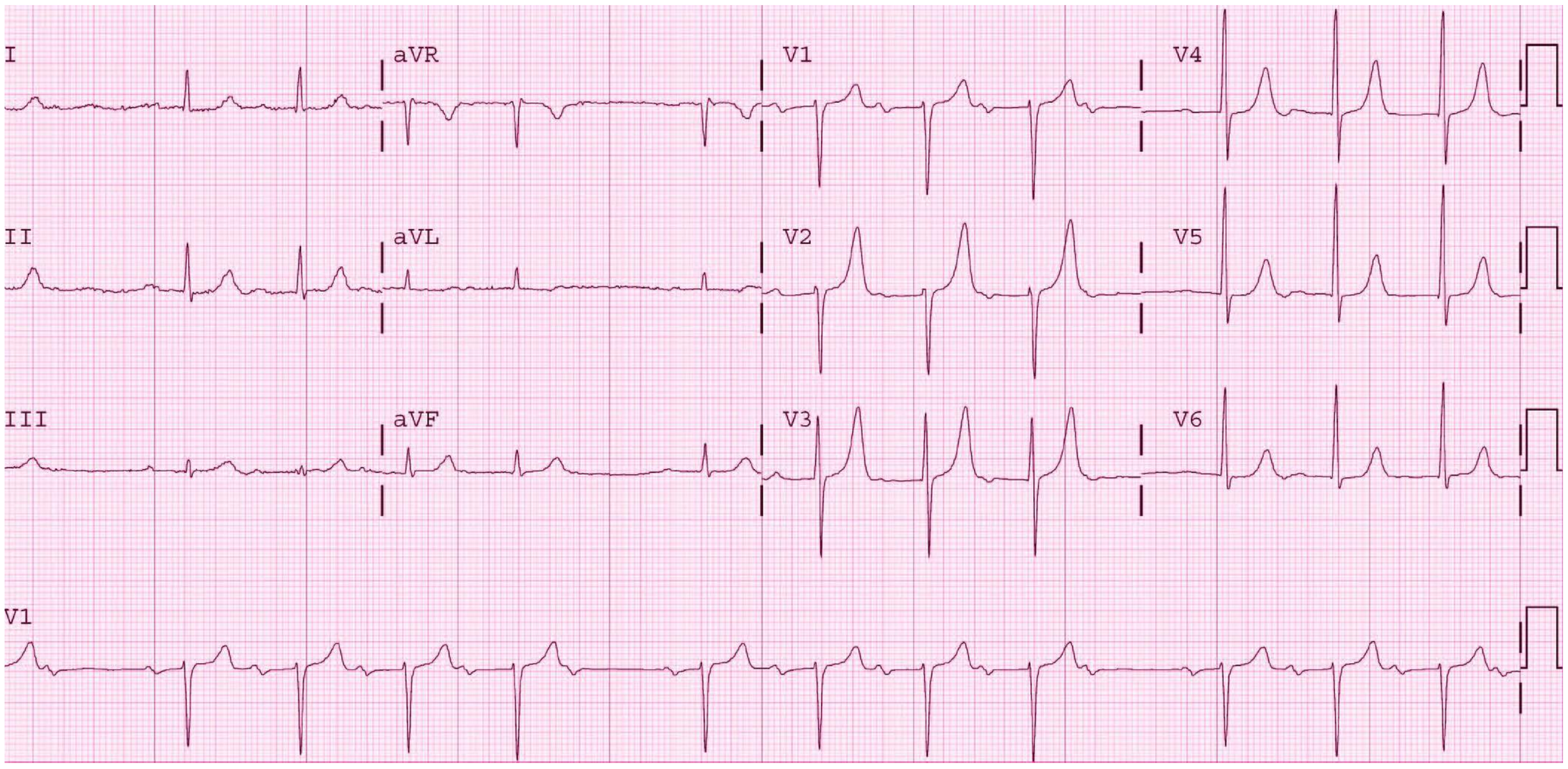


FIGURE 23.15A ■ **Second-Degree AV Block (Mobitz I, Wenckebach).** (ECG contributor: James Paul Brewer, MD.)

ECG Findings

- Progressive PR-interval prolongation throughout the cardiac cycle until a P wave occurs without a QRS complex ("dropped" beat).
- After the dropped QRS complex, the cycle continues again with the PR interval of the first beat in the cycle always shorter than the PR interval of the last beat in the previous cycle.
- P wave may be hidden by the preceding T wave.

Pearls

1. The number of P-QRS complexes prior to the "dropped" beat may vary.
2. A clue to the diagnosis of Mobitz type I heart block can be found in the appearance of grouped QRS complexes.
3. This type of block is normally asymptomatic, and may be seen in athletes.
4. These patients have low risk of progression to complete heart block, and usually do not require a pacemaker. However, Mobitz type I heart block may be caused by inferior myocardial infarction or drugs (digoxin, amiodarone, β-blockers, calcium channel blockers).

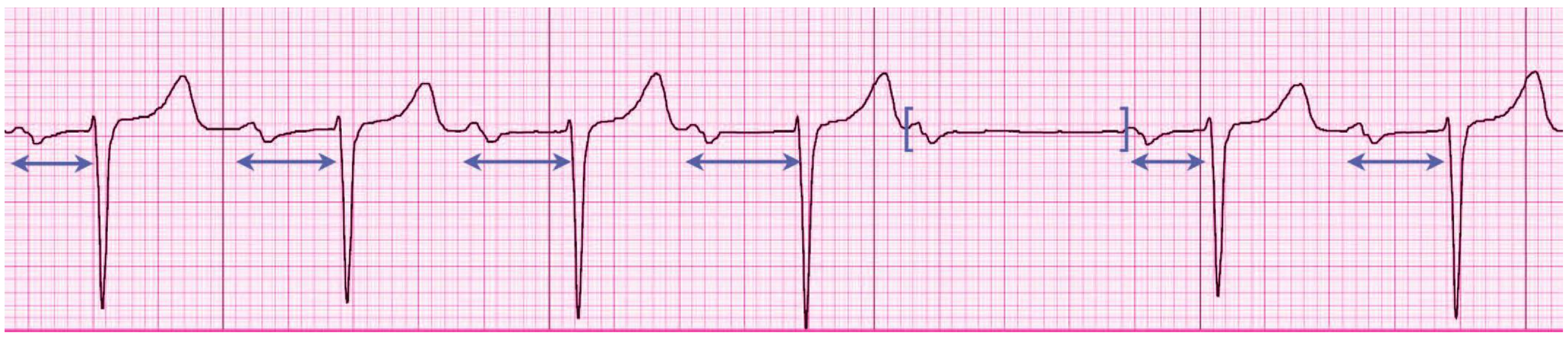


FIGURE 23.15B ■ The PR interval gradually increases (double arrows) until a P wave is not followed by a QRS and a beat is "dropped" (brackets). The process then recurs. P waves occur at regular intervals, though they may be hidden by T waves.

TYPE 2 SECOND-DEGREE AV BLOCK (MOBITZ II)

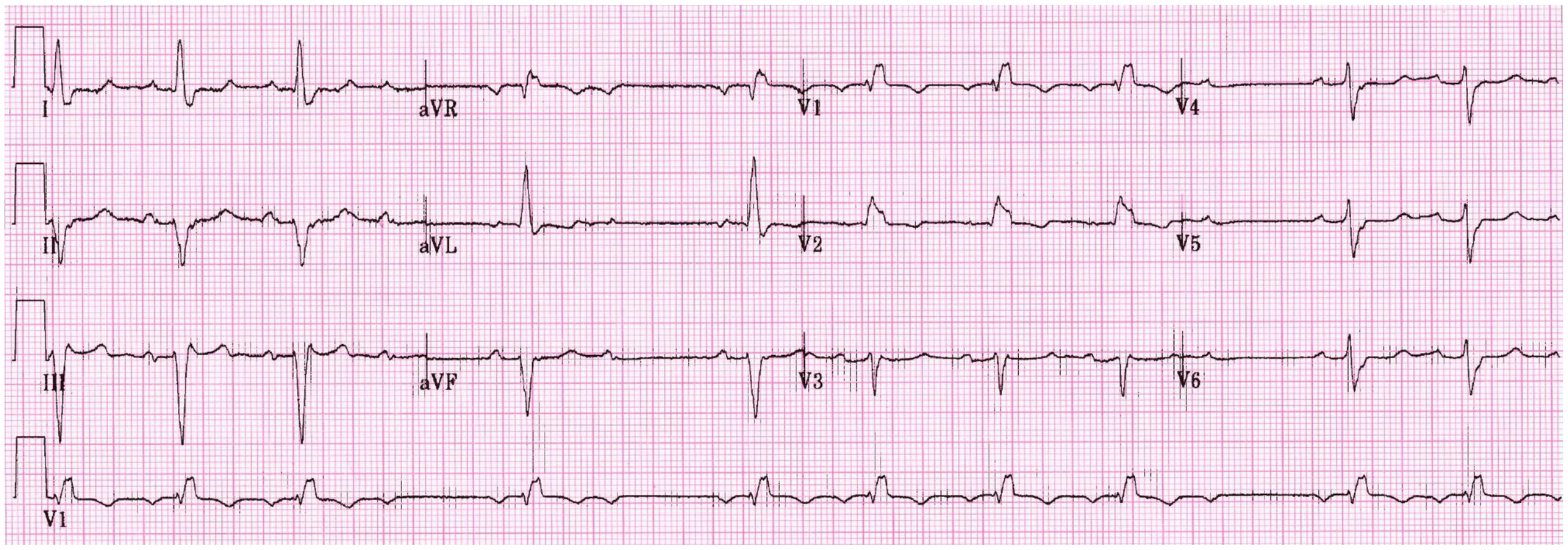


FIGURE 23.16A ■ **Type II Second-Degree AV Block (Mobitz II).** (ECG contributor: Michael L. Juliano, MD.)

ECG Findings

- The PR interval remains constant and does not increase (as seen with Mobitz type I) with each cardiac cycle prior to the "dropped" QRS complex.
- P-P interval is constant, and R-R interval is constant until the dropped beat.
- R-R interval encompassing the "dropped" QRS should be roughly equal to two P-P intervals.

Pearls

1. This type of heart block is associated with disease of the conduction system distal to the AV node. A pacemaker is usually indicated.
2. Mobitz type II block can accompany myocardial infarction and has a high chance of progression to a complete heart block.



FIGURE 23.16B ■ The PR interval is constant (double arrows) until the dropped beat (brackets).

THIRD-DEGREE (COMPLETE) AV BLOCK

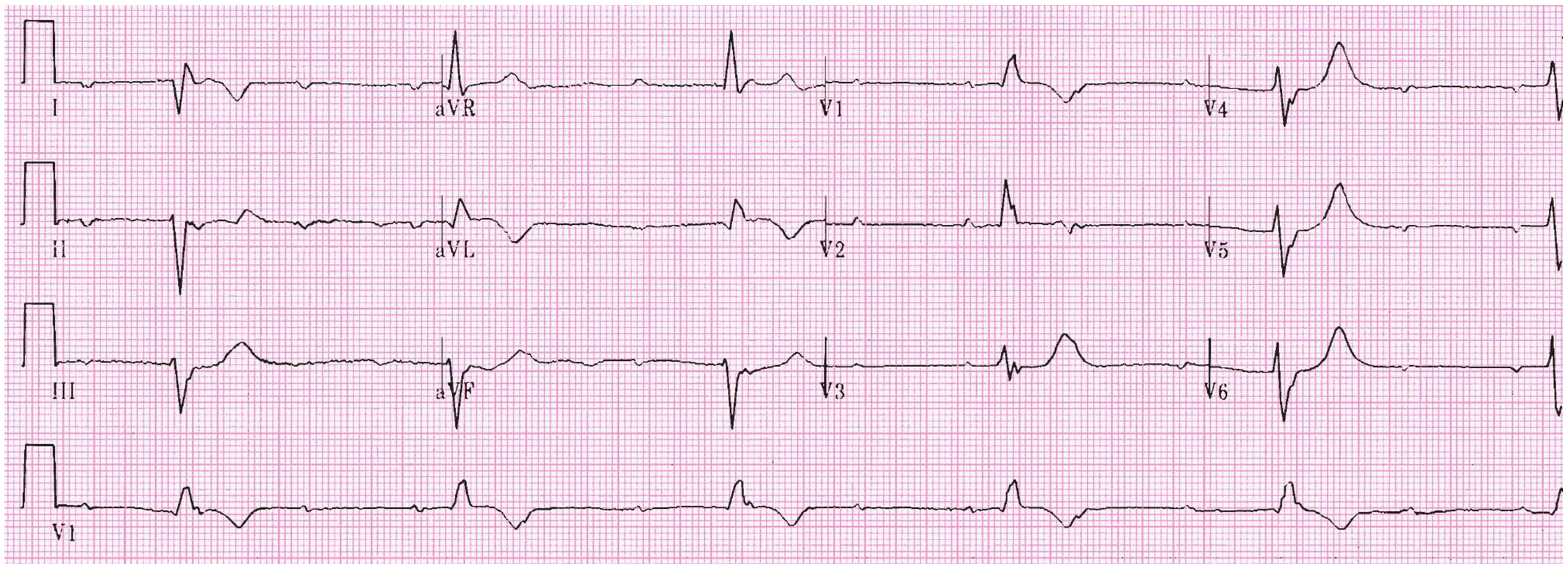


FIGURE 23.17A ■ **Third-Degree AV Block (Complete Heart Block).** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Atrial and ventricular electrical activities are entirely disassociated.
- The P-P and R-R intervals remain constant.
- P waves may be hidden in the QRS complex or may distort the shape of the T wave.
- The atrial rate is usually faster than the ventricular rate.

Pearls

1. Third-degree block is also called complete heart block because no impulses are conducted from the atria to the ventricles.
2. AV rate and QRS morphology depend upon the location of the escape pacemaker.
3. A node escape rate is typically 40 to 60 bpm, with a narrow QRS complex.
4. Ventricular escape rate is usually 20 to 40 bpm, with a widened QRS complex.
5. Complete heart block may be caused by myocardial infarction, conduction system disease, or drugs such as digoxin.
6. Complete heart block may dramatically decrease cardiac output, cardiac pacing is often required.

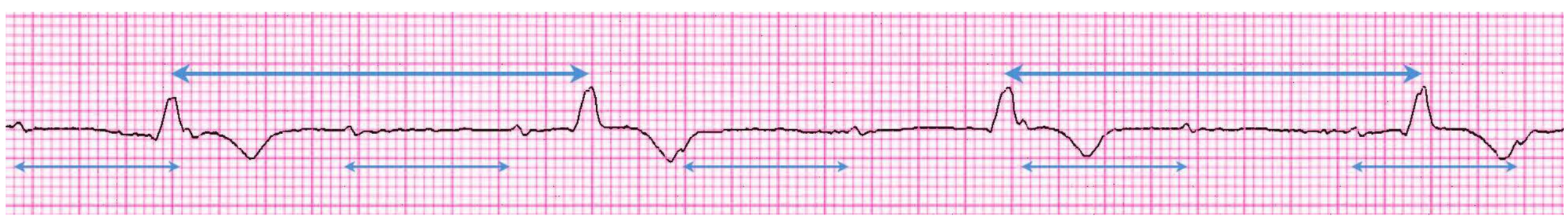


FIGURE 23.17B ■ The P-P interval is uniform (lower double arrows) and the R-R interval is uniform (upper double arrows), but the P waves and QRS complexes are disassociated.

QT INTERVAL PROLONGATION

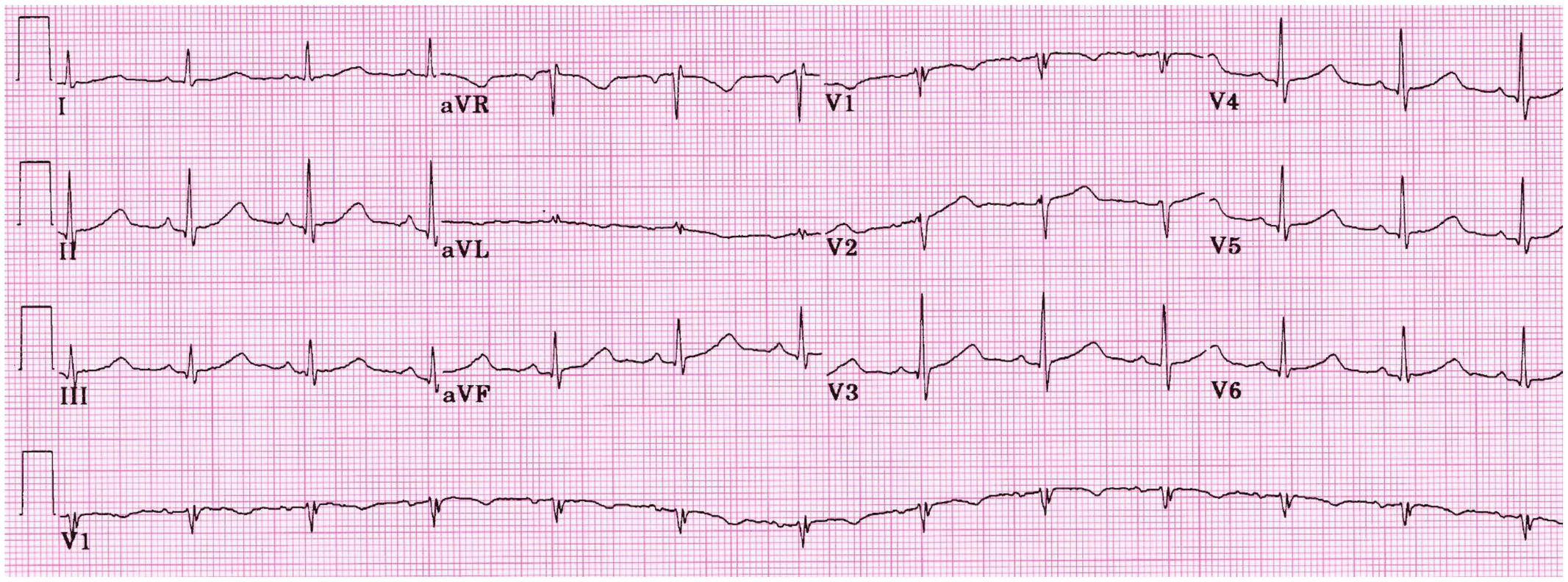


FIGURE 23.18A ■ **Prolonged QT Interval.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Normal QTc interval is less than 440 ms.
- QTc interval greater than 440 ms is considered prolonged.
- QTc interval greater than 550 ms is markedly prolonged.

Pearls

1. The QT interval is measured form the beginning of the QRS complex to the termination of the T wave (or U wave if present) and should be measured in the EKG lead with the longest-appearing QT interval that has a distinct T wave with a clear termination point.
2. The QT interval will increase with bradycardia and decrease with tachycardia, thus it is important to use the corrected QT interval (QTc = QT/μR-R interval) for heart rates other than 60 (in which the QTc = QT).
3. Prolonged QT intervals may be congenital. The vast majority are acquired, usually due to medications or electrolyte abnormalities (hypokalemia, hypomagnesemia, hypocalcemia, and hyperphosphatemia).
4. Numerous medications may prolong the QT interval in therapeutic or toxic doses.
5. Prolongation of the QT interval predisposes to torsades de pointes.
6. Look carefully for prolonged QT intervals in patients who present with syncope.

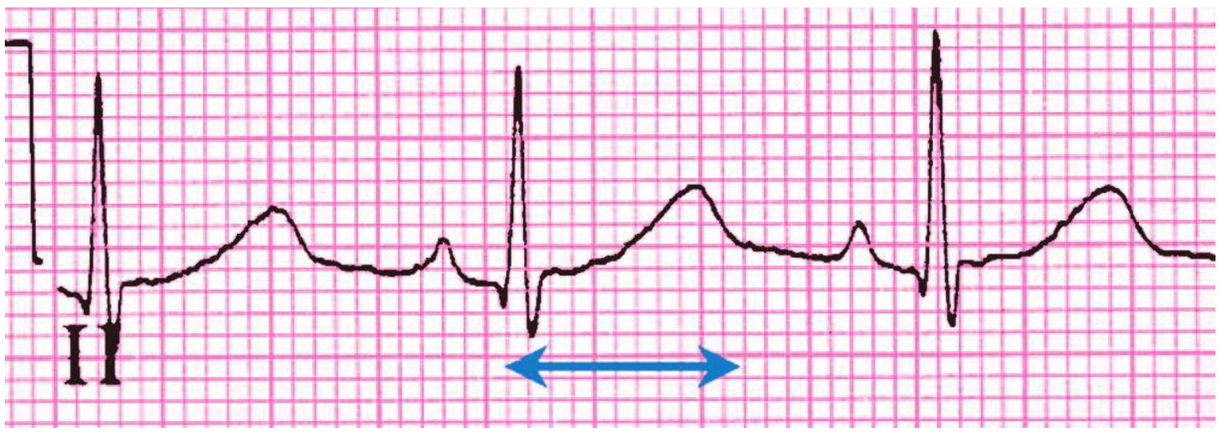


FIGURE 23.18B ■ **QT Interval Prolongation.** QT of 440 ms, QT_c of 498 (double arrow). Note the QT interval is measured from the beginning of the QRS complex to the termination point of the T wave.

RIGHT BUNDLE BRANCH BLOCK

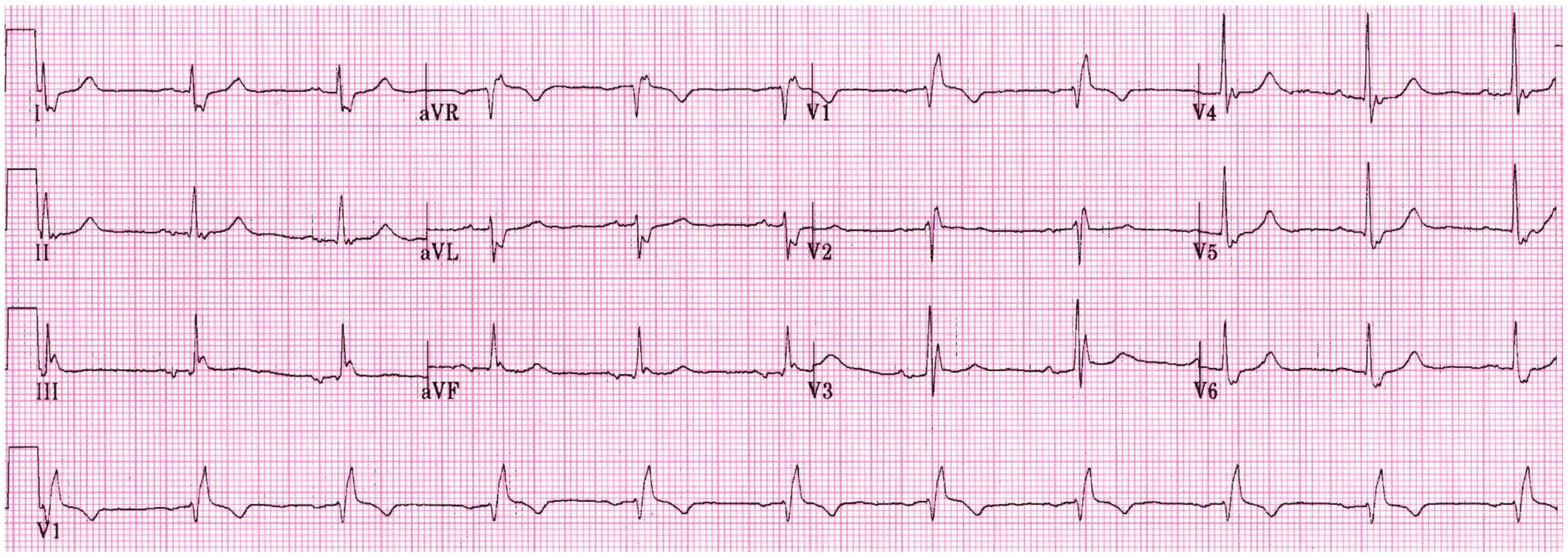


FIGURE 23.19A ■ **Right Bundle Branch Block.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Wide QRS complex, at least 120 ms (three small blocks).
- QRS complex has sR' or rsR' in leads V_1 and V_2.
- Slurred S wave V_6 and I.

Pearls

1. The signal exiting the AV node is carried rapidly to the LV through the intact left bundle, but is delayed into the right ventricle, where depolarization must propagate cell-to-cell. Since the RV myocardial mass is much smaller than that of the LV, this delay in depolarization is best seen in the leads overlying the right ventricle, leads V_1 and V_2.
2. Acute right heart strain, as may occur with pulmonary embolism, may result in new onset right bundle branch block (RBBB).

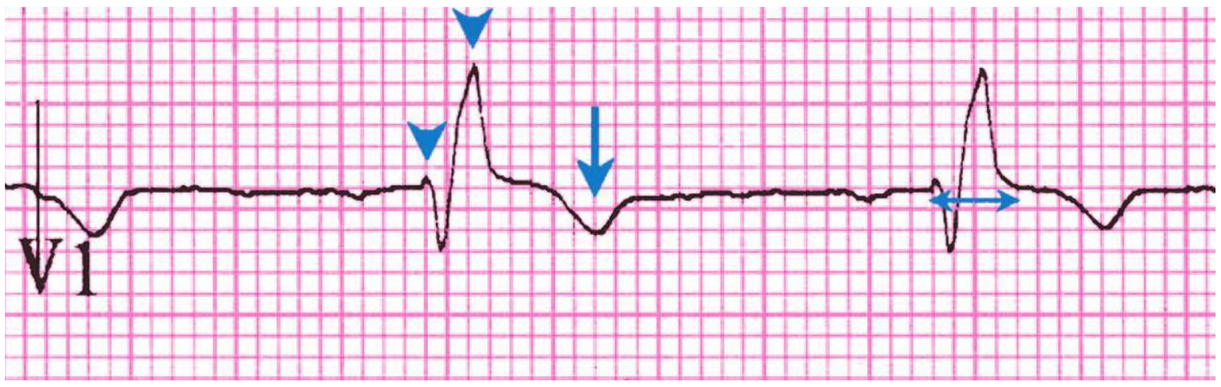


FIGURE 23.19B ■ rsR' pattern in V_1 (arrowheads), with T wave downgoing (arrow). QRS duration greater than 120 ms (double arrow).

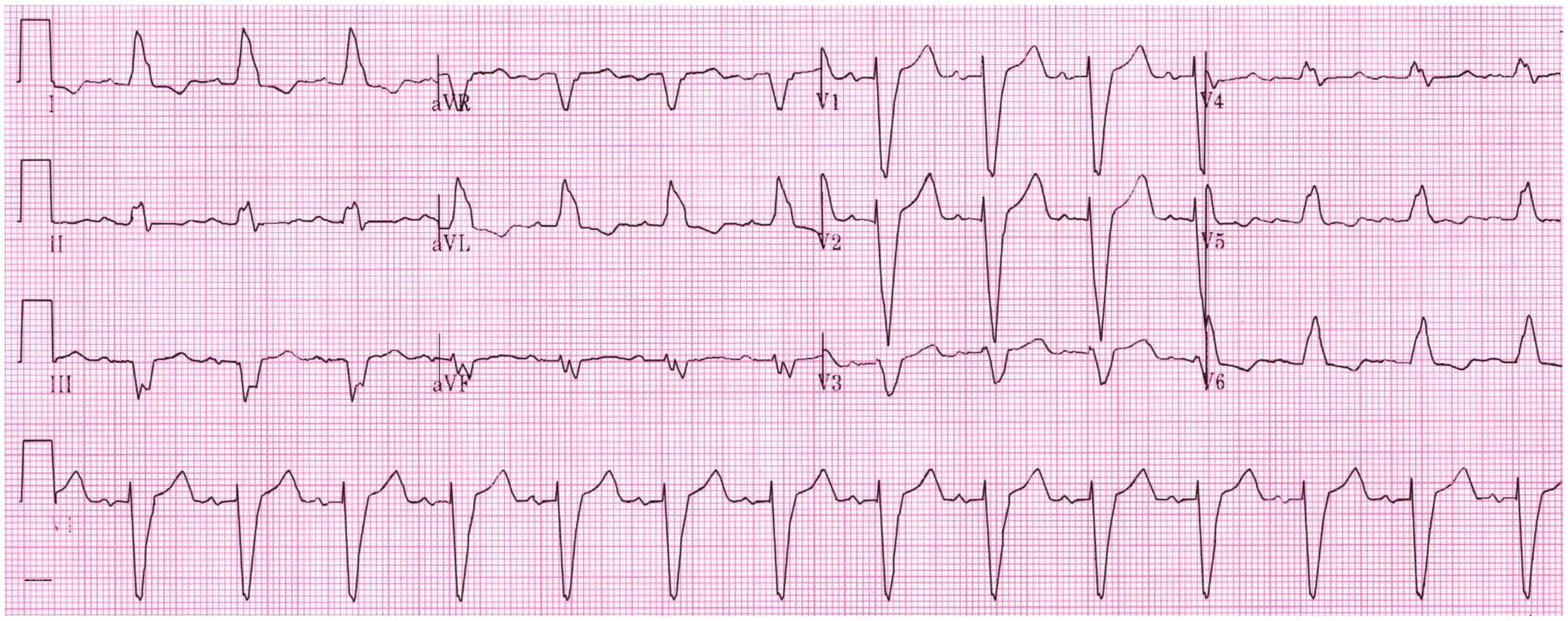


FIGURE 23.20A ■ **Left Bundle Branch Block.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Wide QRS complex, at least 120 ms (three small blocks).
- T wave appears on the opposite side of the baseline from the QRS complex.
- The QRS precordial axis is normal or deviated to the left.
- QRS complex deflection is predominately downward in lead V_1 and upward in lead V_6.

Pearls

1. The signal exiting the AV node does not proceed through the left ventricular conduction system. It must propagate more slowly cell-to-cell through the myocardium, starting in the septum. Therefore, the QRS is wider and the bulk of the depolarization signal is deflected toward the far lateral aspect of the heart.
2. Acute myocardial infarction may produce a new onset LBBB on ECG. Therefore, patients with new onset LBBB with a clinical presentation consistent with acute coronary syndrome should be treated as having an acute ST segment elevation myocardial infarction (STEMI).

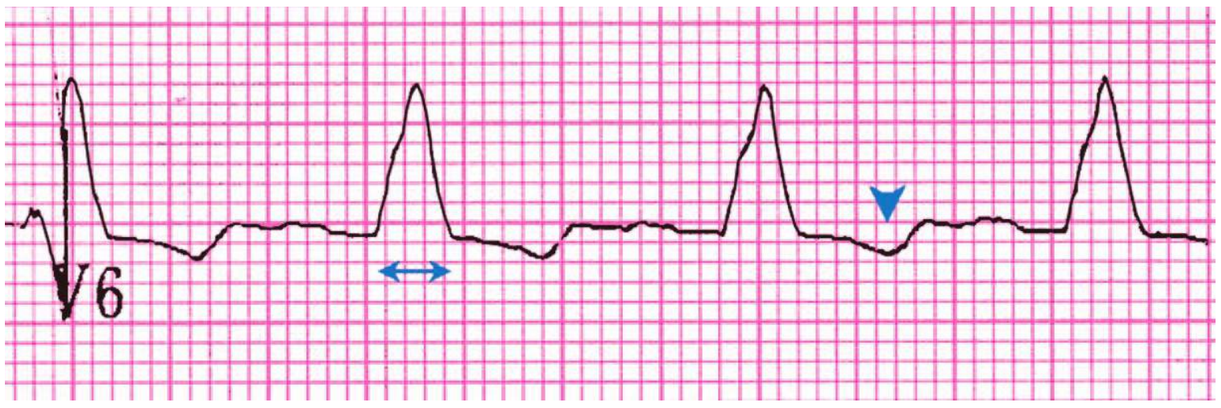


FIGURE 23.20B ■ The QRS is wider than 120 ms (double arrow). The T-wave deflection is in the opposite direction from the QRS deflection (arrowhead).

LEFT ANTERIOR FASCICULAR BLOCK

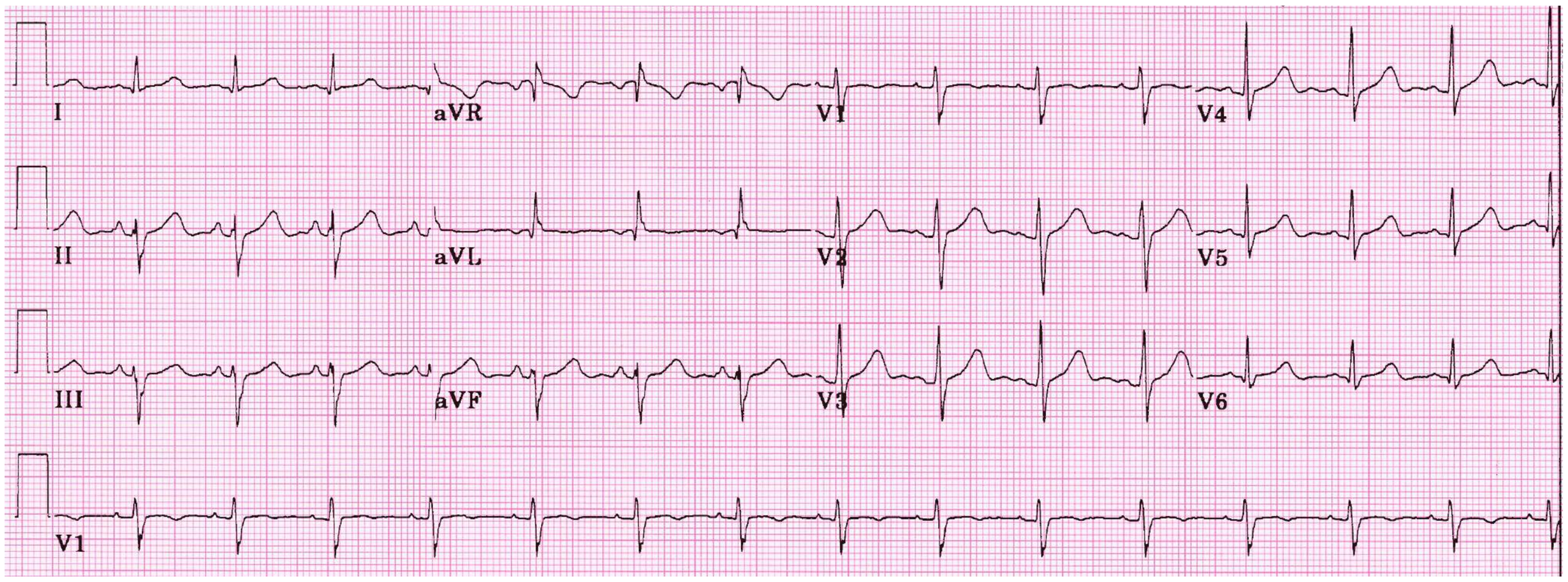


FIGURE 23.21A ■ **Left Anterior Fascicular Block.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- QRS complex widening, usually 90 to 120 ms
- Left axis deviation beyond minus 45 degrees with no other cause (such as inferior myocardial infarction)
- Small R wave and large S wave in the inferior leads
- Slurred S wave in V_5 and V_6

Pearls

1. The signal exiting the AV node is carried rapidly to the inferior aspect of the LV and all of the RV through the intact left posterior fascicle and right bundle, where quick depolarization occurs. However, conduction to the high lateral and upper portions of the left ventricle is slower and must proceed cell-to-cell due to the blocked left anterior fascicle. Therefore, the latter portion of the QRS depolarizes toward the upper lateral myocardium, manifested as strong left axis deviation.
2. Left anterior fascicular block is the most common intraventricular conduction disturbance associated with acute anterior myocardial infarction, with the left anterior descending artery usually involved.

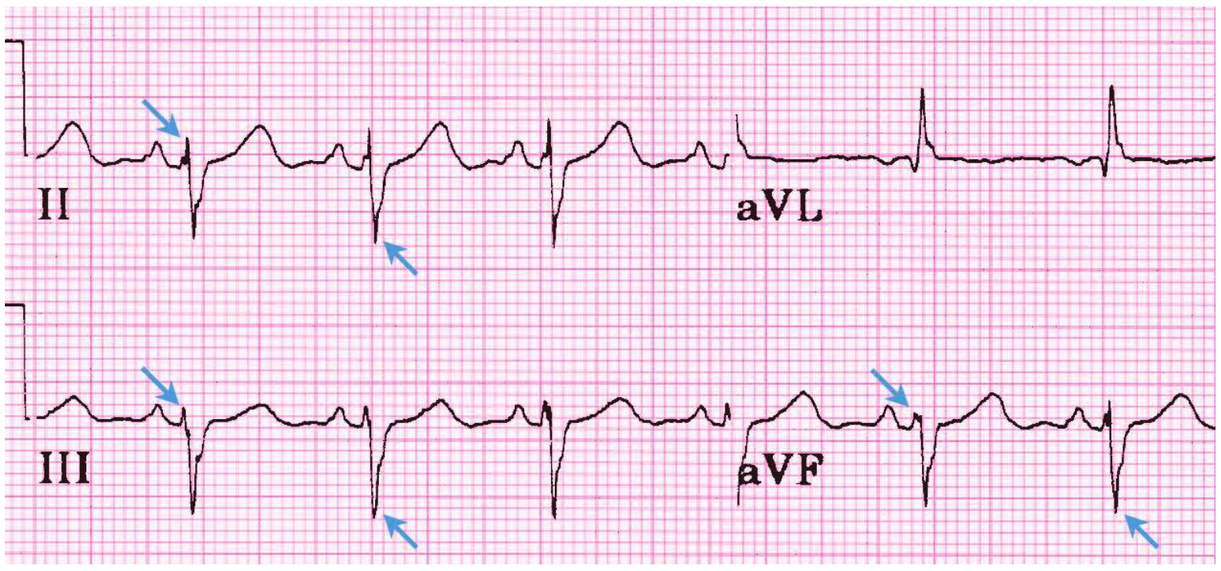


FIGURE 23.21B ■ Small R waves, large S waves in all inferior leads (arrows), with QRS axis deviated left beyond minus 45 degrees.

LEFT POSTERIOR FASCICULAR BLOCK

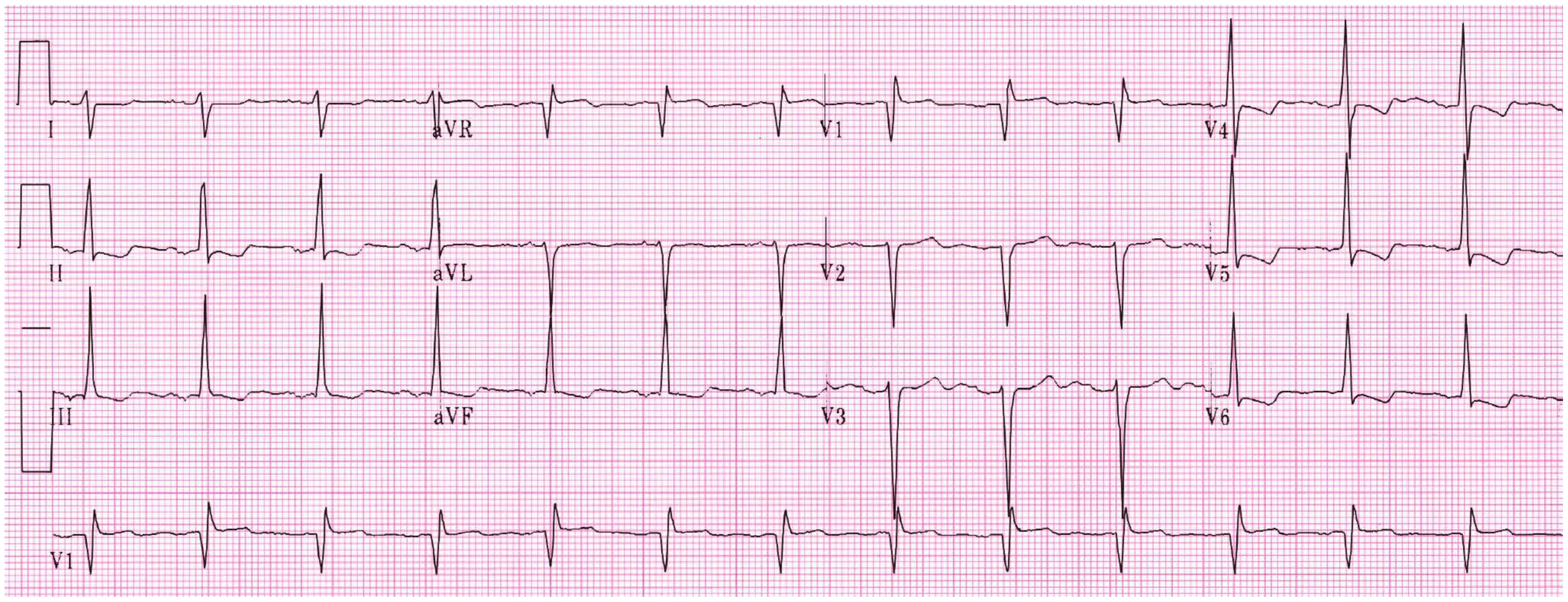


FIGURE 23.22A ■ **Left Posterior Fascicular Block.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- QRS complex widening to 90 to 120 ms.
- Right axis deviation must be beyond 100 degrees and must have no other cause (such as lateral myocardial infarction).
- Small R wave and large S wave in the high lateral leads, I and aVL.
- Slurred S wave in V_5 and V_6.
- This example also contains unrelated ST changes.

Pearls

1. The signal exiting the AV node is carried rapidly to the upper aspect of the LV and all of the RV through the intact left anterior fascicle and right bundle, where depolarization is rapid. However, conduction to the inferior portion of the left ventricle is slower and must proceed cell-to-cell due to the blocked left posterior fascicle. Therefore, the latter portion of the QRS depolarizes toward the inferior myocardium, manifesting as strong right axis deviation.
2. Left posterior fascicular block may be associated with acute inferior myocardial infarction as well as with multiple cardiomyopathic conditions.

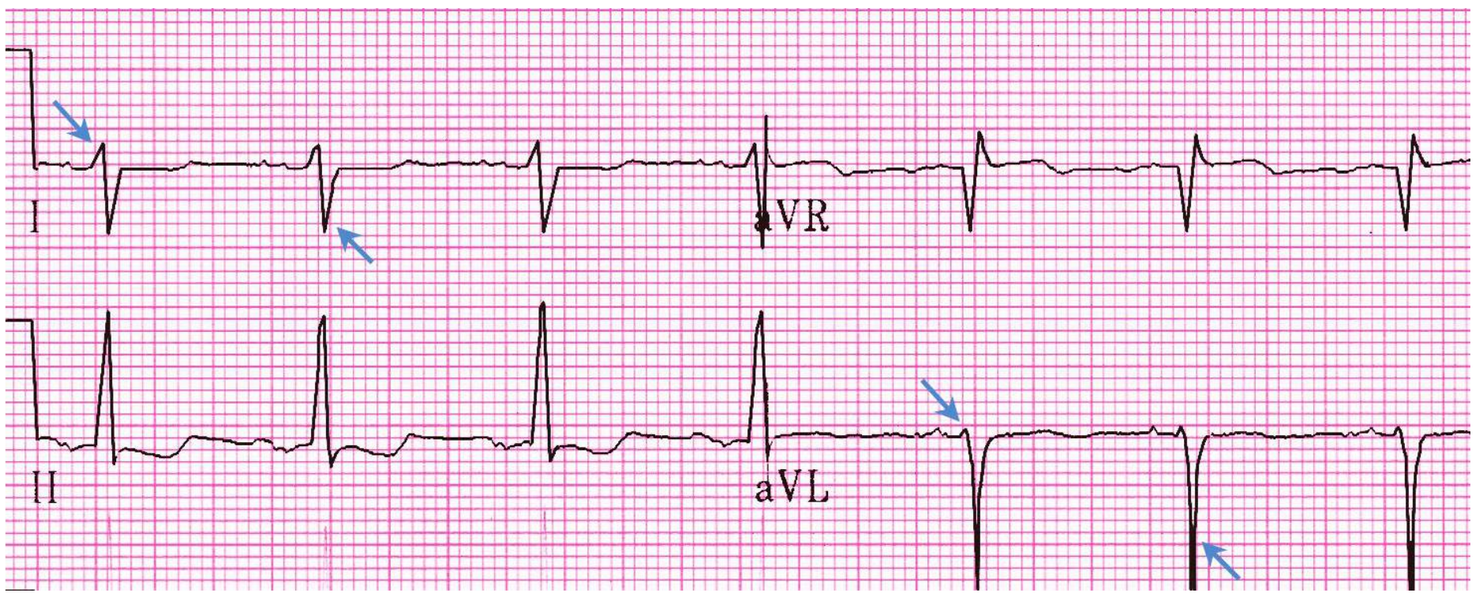


FIGURE 23.22B ■ Small R waves and large S waves in leads I and aVL (arrows).

TRIFASCICULAR BLOCK

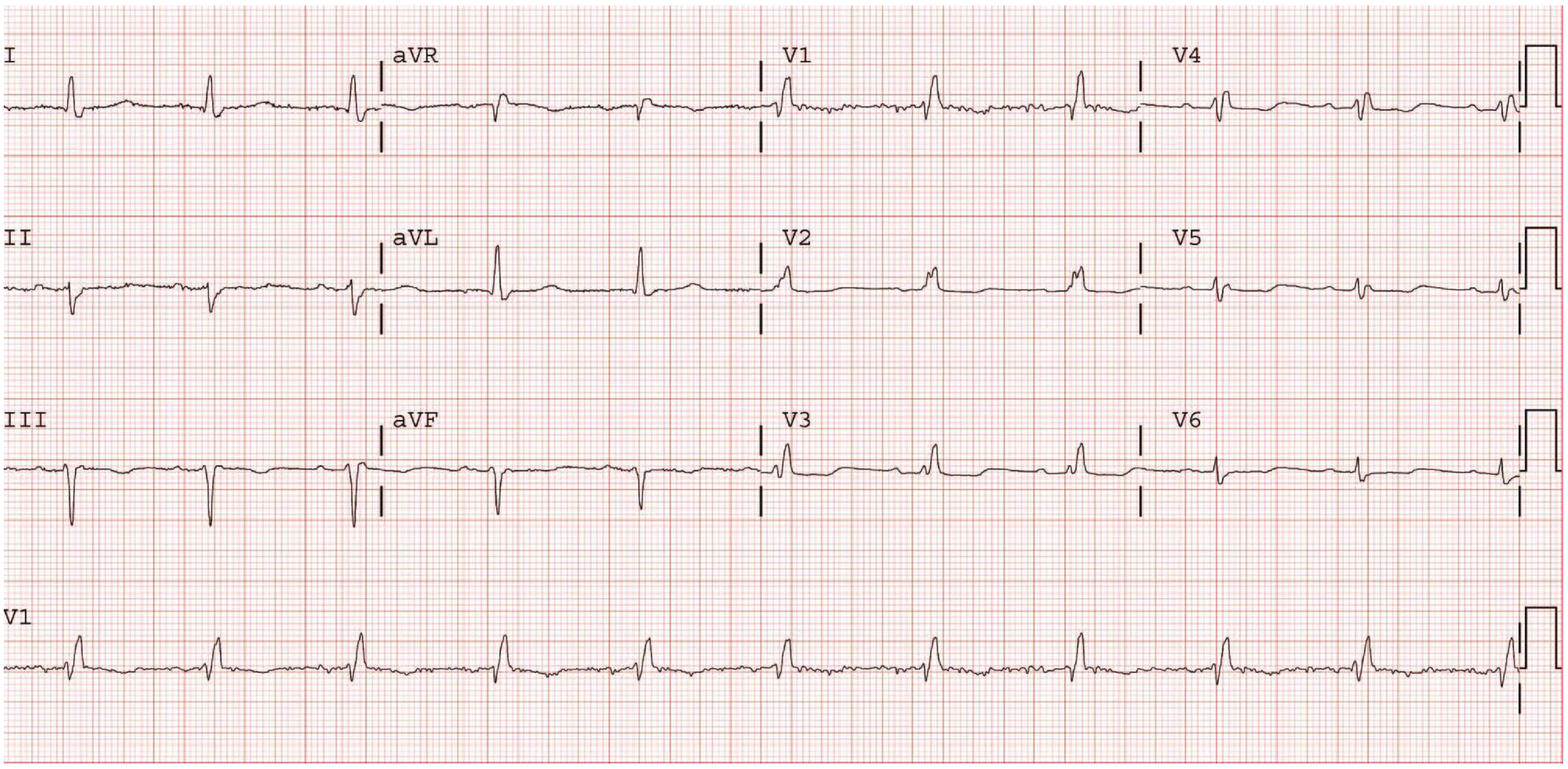


FIGURE 23.23A ■ **Trifascicular Block.** (ECG contributor: R. Jason Thurman, MD.)

ECG Findings

- RBBB with left anterior or posterior hemiblock and first-degree AV block
- Somewhat of a misnomer, not a true blocking of three fascicles: two fascicles blocked coupled with PR interval prolongation
- LBBB plus first-degree AV block sometimes referred to as trifascicular block as well, but again only two fascicles truly blocked in this scenario

Pearls

1. Trifascicular block may be a precursor to complete heart block: Pacemaker evaluation is warranted, especially if symptomatic, but incidence of progression to complete heart block is low.
2. AV blocking agents can potentiate degree of block.
3. Can be due to systemic diseases (Chagas disease) but usually indicative of primary advanced conduction system abnormalities secondary to coronary artery disease.

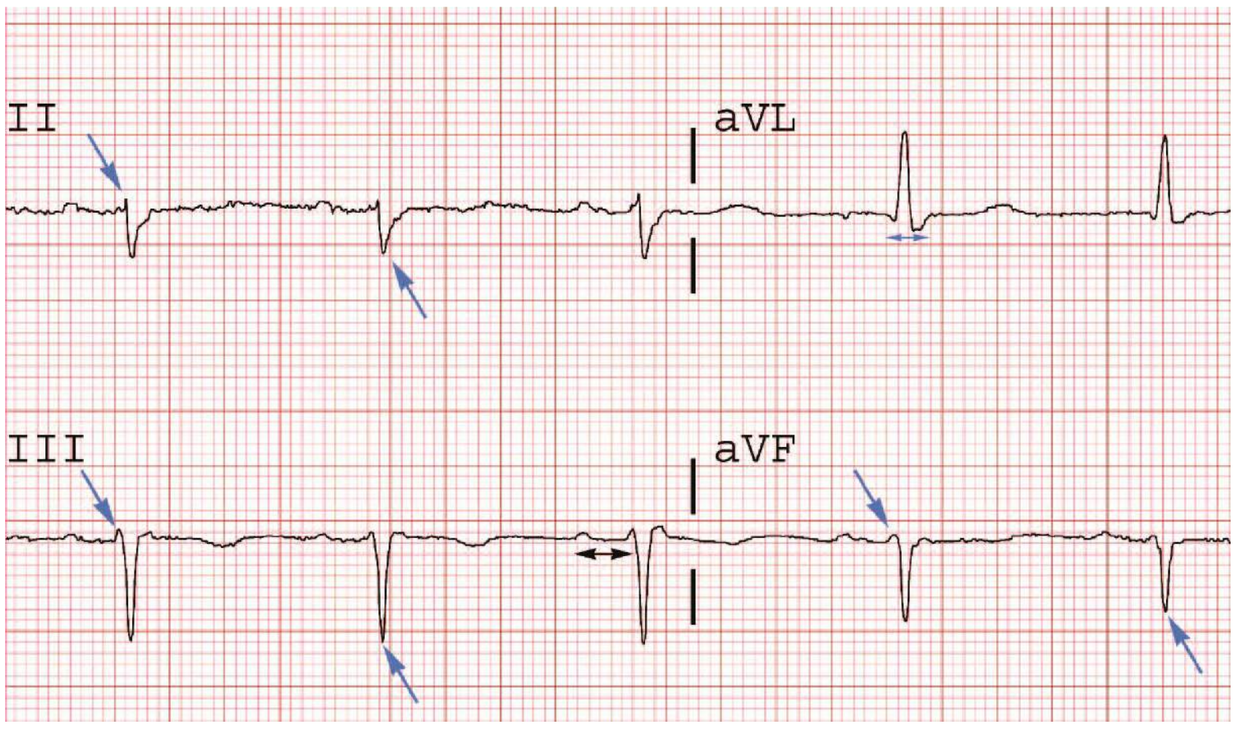


FIGURE 23.23B ■ The QRS is wide with RBBB pattern (blue double arrow). Left posterior fascicular block is present (diagonal arrows) along with a prolonged PR interval and first-degree AV block (black double arrow).

ASHMAN PHENOMENON

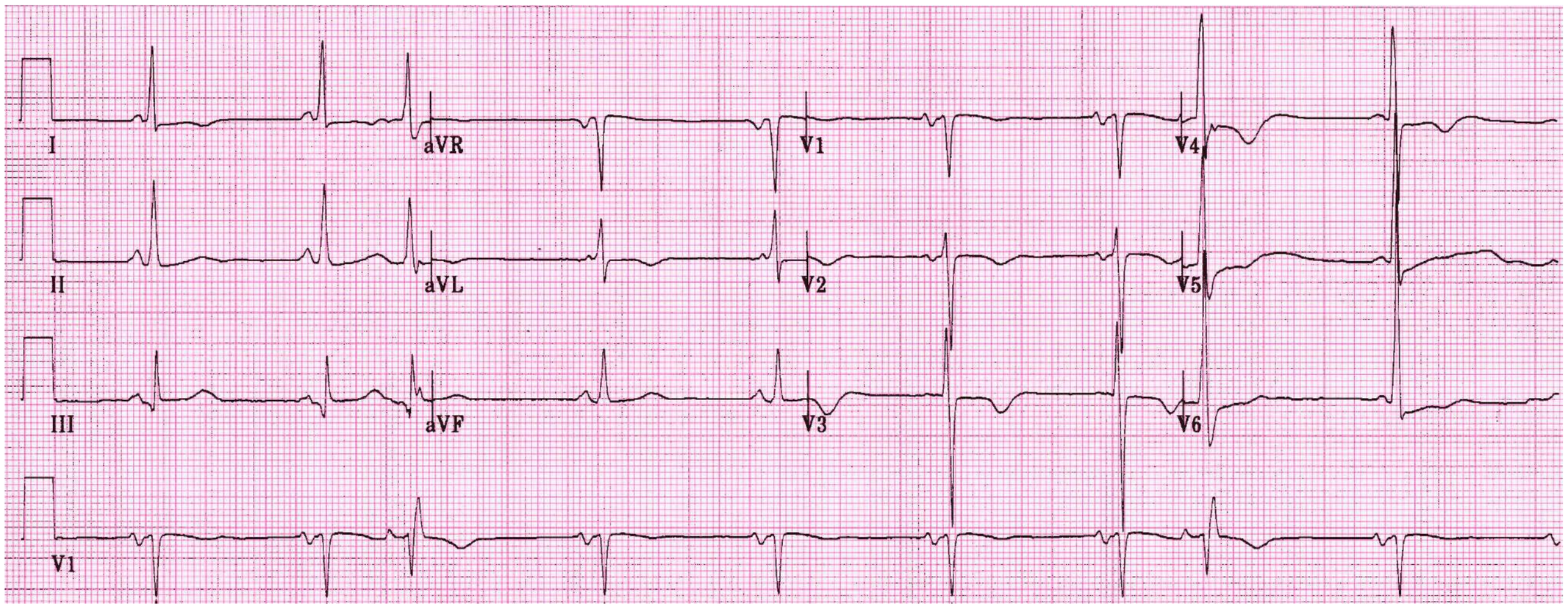


FIGURE 23.24A ■ **Ashman Phenomenon.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Aberrant ventricular conduction, usually with RBBB pattern.
- Altered durations of the refractory period of the bundle branch or ventricular tissue are present, commonly due to atrial fibrillation, atrial ectopy, and atrial tachycardia

Pearls

1. After depolarization, tissue repolarizes during its refractory period. Refractory period changes with the preceding cardiac cycle, with longer R-R intervals producing longer refractory periods and shorter R-R intervals producing shorter refractory periods.
2. A longer R-R interval lengthens the following refractory period. When an early or premature (ectopic) depolarization reaches the ventricular conduction system before it has completely repolarized, aberrant conduction may occur and be manifest on the ECG with a bundle branch block (BBB) pattern.
3. Ashman phenomenon most commonly appears with an RBBB pattern, since the right bundle has a longer refractory period than the left bundle.
4. Ashman phenomenon is often seen in atrial fibrillation, when a long R-R interval is followed by a much shorter R-R interval.
5. In the setting of a premature atrial beat (as seen in this example), the earlier in the cycle the PAC occurs and the longer the preceding R-R interval is, the more likely aberrant conduction of the beat will occur.



FIGURE 23.24B ■ After a relatively long R-R interval (double arrow), a PAC (diagonal arrow) is followed by an aberrantly conducted QRS with RBBB morphology (arrowhead). After a short pause (single arrow), the next beat is conducted normally as it has occurred outside of the refractory period set by the previous beat.

JUNCTIONAL RHYTHM

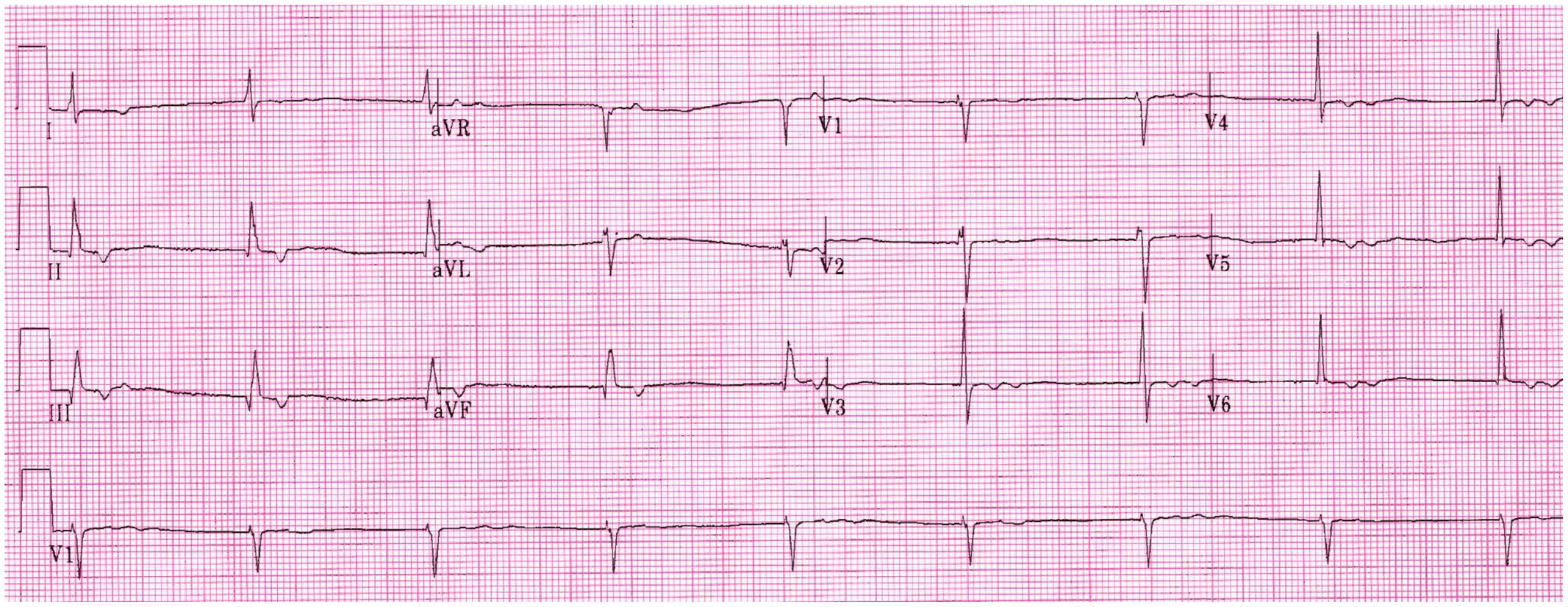


FIGURE 23.25A ■ **Junctional Rhythm with Retrograde P Waves.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- The QRS complex is narrow, with a rate typically between 40 and 60 bpm.
- P waves are absent, retrograde, very slow, or unrelated to the QRS complex.

Pearls

1. When the atria fail to initiate a cardiac rhythm, or when no pacing signal reaches the lower AV node, the AV node or His bundle usually picks up the pacemaking responsibility.
2. P waves may be conducted retrograde and buried in the T wave as seen in this example. In the case of a complete AV block, the P waves have no relation to the QRS complex.
3. If a BBB is also present, the QRS may be wide, and may be difficult to discern from a primary ventricular rhythm.

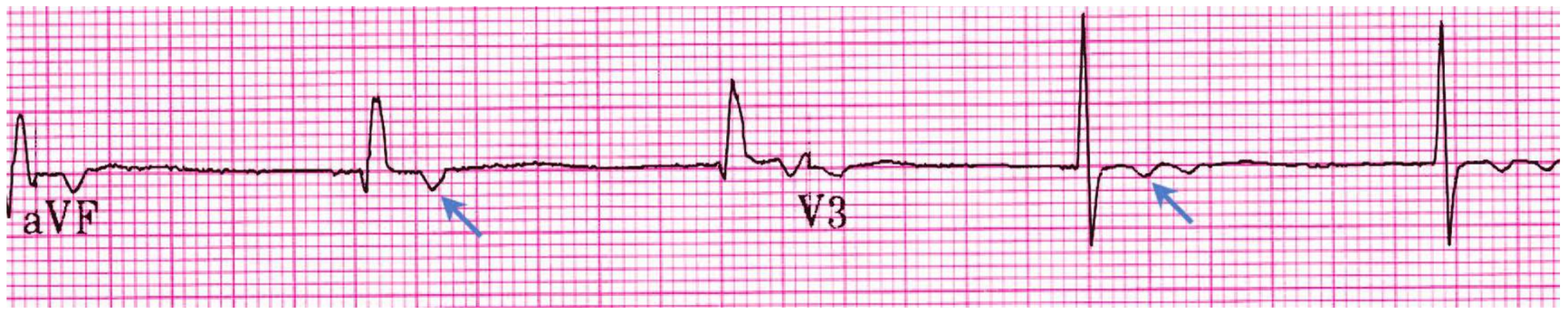


FIGURE 23.25B ■ The QRS is narrow. P waves are not present before the QRS. In this example, the signal which originated in the His bundle is conducted retrograde through the AV node into the atria, and retrograde P waves are apparent in the ST segment (arrows).

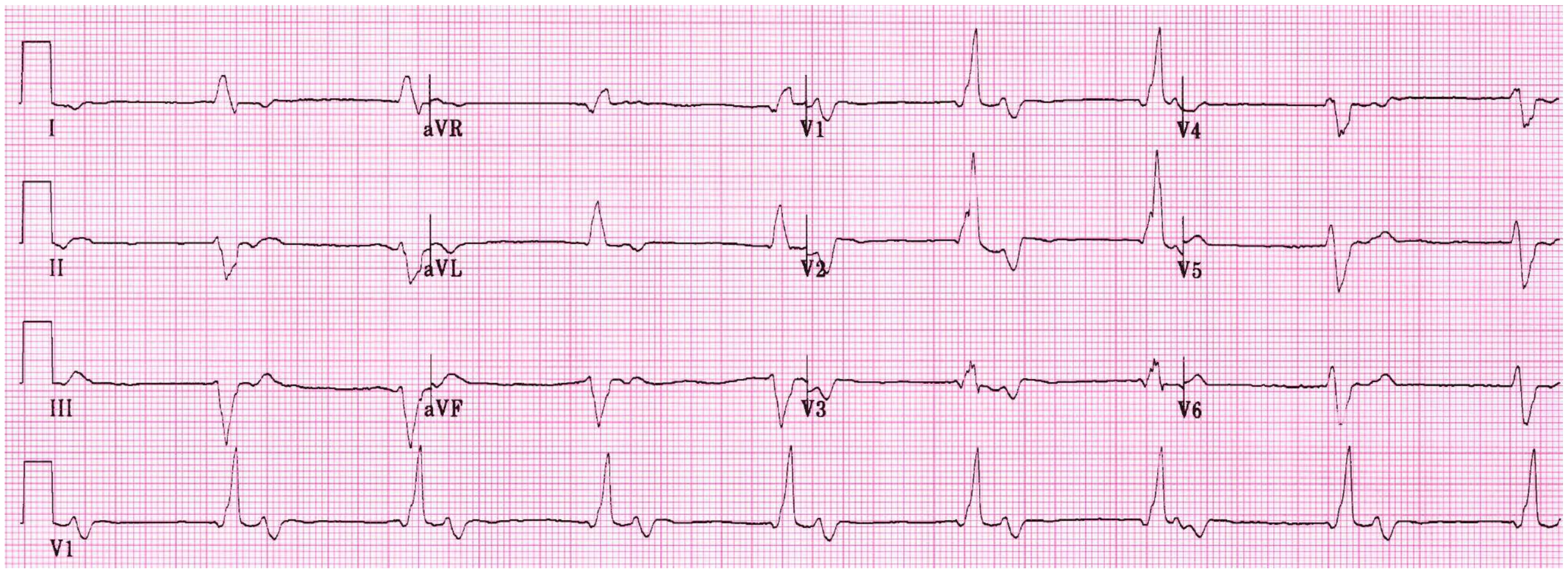


FIGURE 23.26A ■ **Ventricular Rhythm with Retrograde P Waves.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- The QRS complex is wider than 120 ms, with a rate typically between 20 and 40 bpm.
- The T wave is discordant relative to the QRS.

Pearls

1. When the atria and the AV node fail to initiate a cardiac rhythm, or when no pacing signal reaches the ventricle, the ventricular tissue usually picks up the pacemaking responsibility.
2. P waves may also be conducted retrograde and buried in the T wave as seen in this example. In the case of a complete AV block, the P waves have no relation to the QRS complex.



FIGURE 23.26B ■ Wide-complex (double arrow) regular QRS at a rate of approximately 50 bpm. Retrograde P waves are seen in this example (arrow).

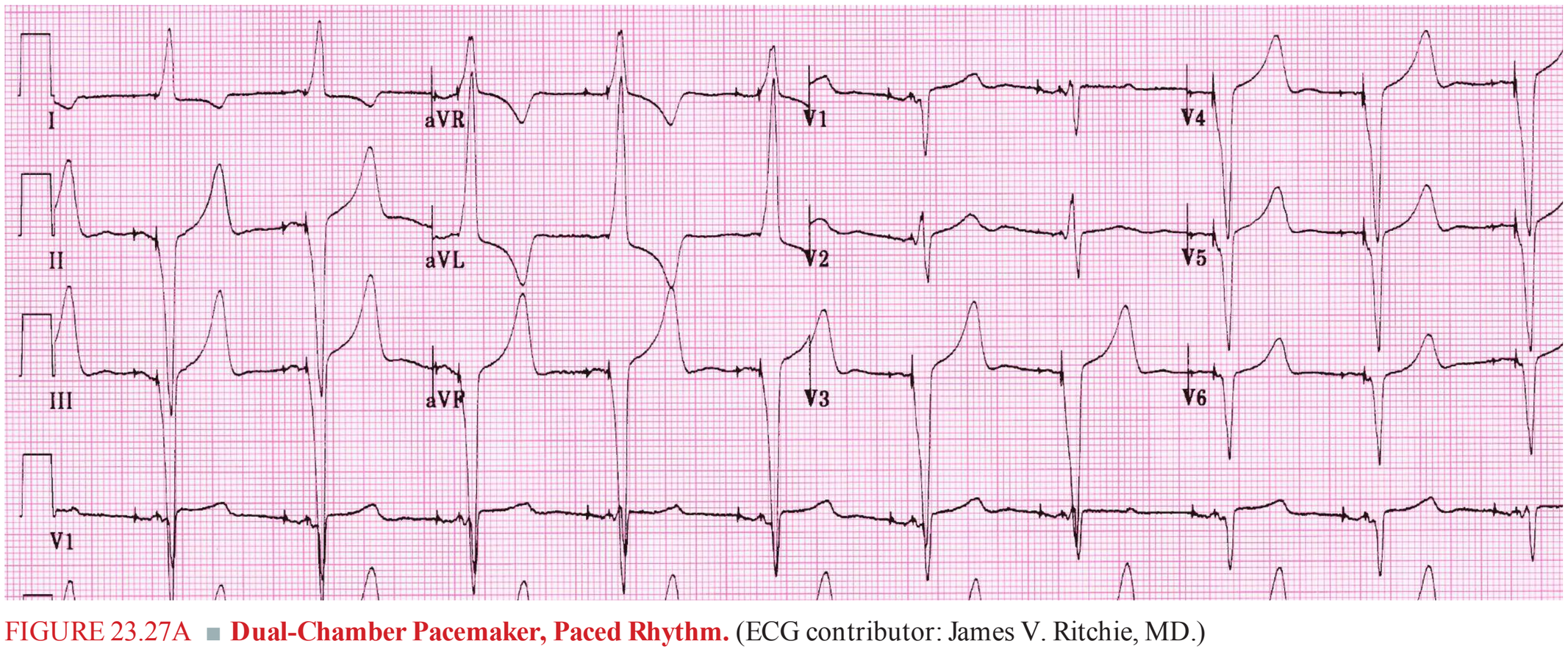


FIGURE 23.27A ■ **Dual-Chamber Pacemaker, Paced Rhythm.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Very narrow signal of no discernable width, immediately followed by a P wave (if an atrial lead) or a QRS complex (if a ventricular lead). The narrow pacer "spike" amplitude varies, and can be larger than the QRS or may be indiscernible.
- A QRS complex initiated by a pacer spike will be wide, with morphology similar to a PVC or idioventricular rhythm. The axis is unlike typical BBBs, because the signal usually originates low in the right ventricle.

Pearls

1. Pacemakers are designated by the "five letter" system. In this system, the letter "A" denotes atrium, "V" denotes ventricle, "D" denotes dual (both chambers), and "O" denotes neither. The first three letters are the most commonly used:
   A. First letter—designates chamber(s) paced
   B. Second letter—designates chamber(s) sensed
   C. Third letter—designates pacemaker response to sensed electrical activity: T: triggered—fires even when beat sensed, I: inhibitory—holds when beat sensed, D: dual—atrial triggered and ventricle inhibited
   D. Fourth letter—extra options: P: programmable, M: multiprogrammable, C: communicating, R: rate adaptation, O: none
   E. Fifth letter—cardioverting options: P: pacing, S: shocking, D: dual (P+S), O: none
2. The two most common pacemaker malfunctions are failure to pace and failure to sense.
3. Some ECG machines perceive small pacer spikes as artifact and do not reproduce them on the printed tracing.

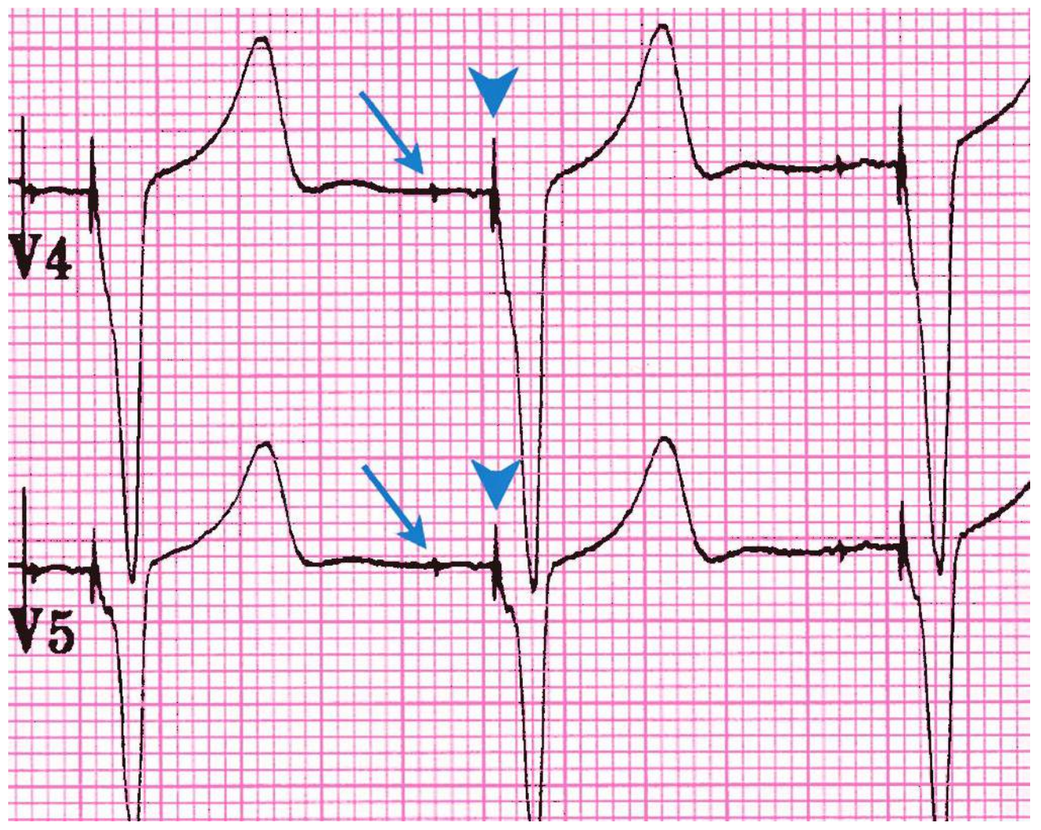


FIGURE 23.27B ■ Tiny pacer spikes (arrows) precede the P waves, and somewhat larger pacer spikes precede the QRS complexes (arrowheads). The QRS complexes are wide, with discordant T waves.

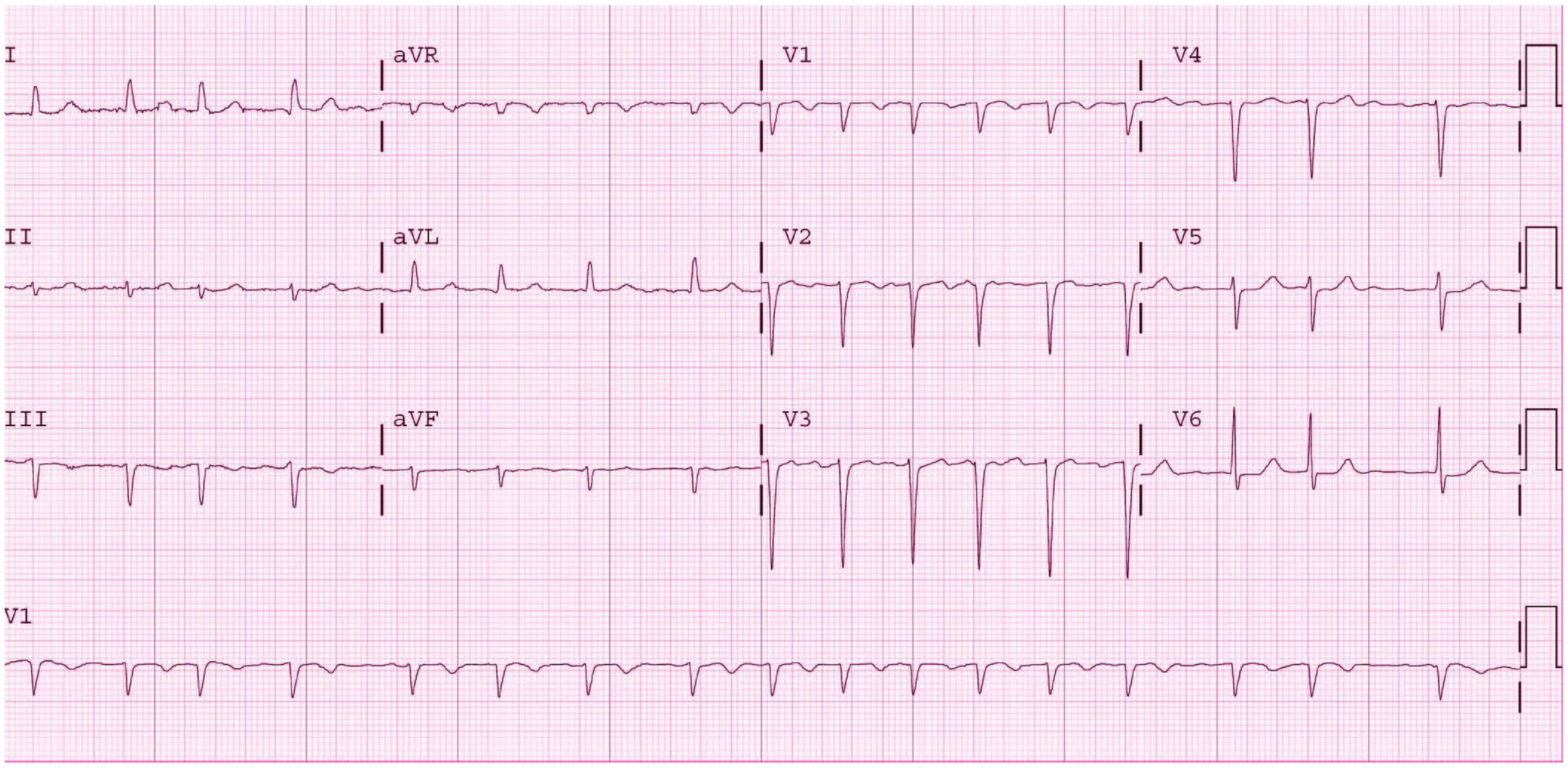


FIGURE 23.28A ■ **Atrial Fibrillation.** (ECG contributor: R. Jason Thurman, MD.)

ECG Findings

- Electrical activity in the atria is chaotic. The ECG baseline, representing the ongoing chaotic atrial activity, is unorganized. The resulting "rumbling" baseline may have large or indiscernibly small amplitude.
- The AV node has a refractory period, and therefore does not conduct every signal it receives from the atria. Thus, ventricles depolarize variably creating varying R-R intervals and an "irregularly irregular" pattern.

Pearls

1. Most "irregularly irregular" rhythms are due to atrial fibrillation, but other rhythms may produce similar findings. These include multifocal atrial tachycardia, atrial flutter with variable AV block, and frequent PJCs.
2. Therapy is geared toward either rate control of the ventricles or rhythm control and cardioversion (chemically or electrically).
3. Synchronized cardioversion may be indicated if a patient is unstable, but the risk of clot embolization must be carefully considered when planning nonemergent electrical cardioversion of atrial fibrillation.



FIGURE 23.28B ■ R-to-R interval varies in an "irregularly irregular" pattern (double arrows). The baseline "rumble," representing "F waves," may be very fine or even indiscernible.



FIGURE 23.28C ■ The baseline "rumble" may be very coarse resembling atrial flutter waves.

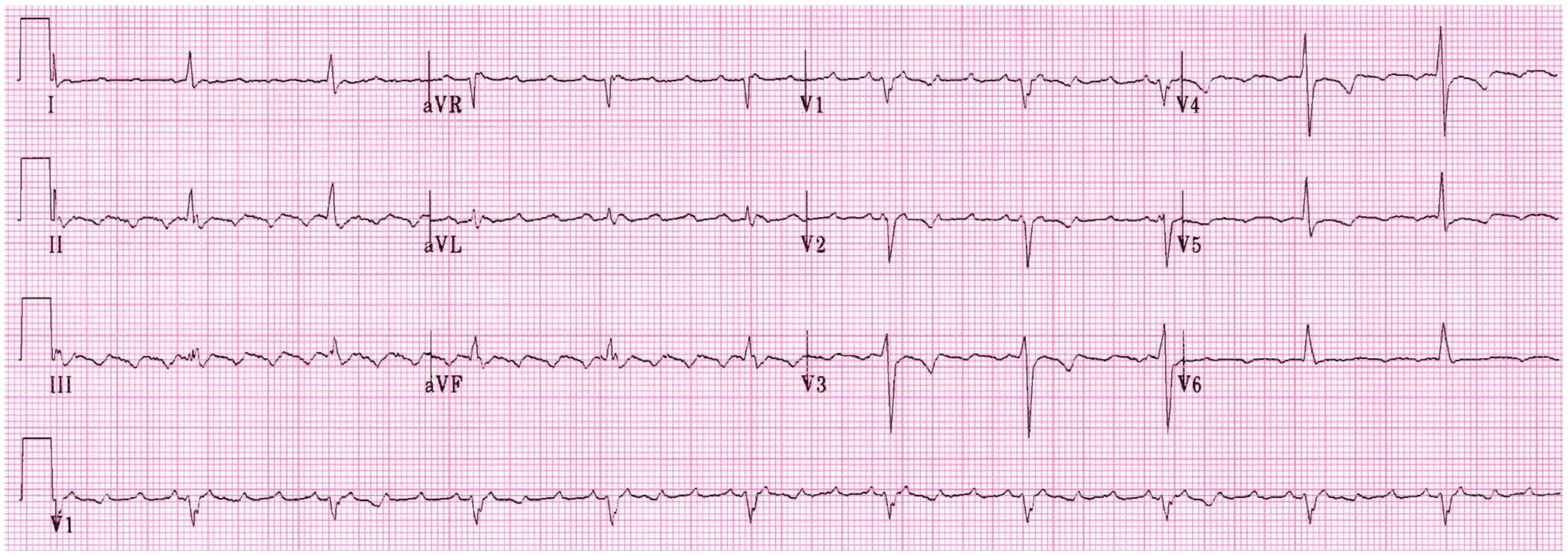


FIGURE 23.29A ■ **Atrial Flutter.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Electrical activity in the atria is ongoing and regular, self-propagating in a roughly circular movement.
- Flutter waves appear in a rapid sine wave or "sawtooth" pattern, usually in the inferior leads.
- Atrial activity in lead V_1 often appears as rapid P waves at a rate approximating 300 bpm.

Pearls

1. The AV node's refractory period prevents 1:1 conduction to the ventricles. Usually, conduction is blocked at a ratio of 1:2 to 1:4. The QRS complexes should appear with regular periodicity. However, AV conduction may be variable from beat-to-beat creating irregular R-to-R intervals.
2. A conduction ratio of 2:1 is usually difficult to discern, because the two flutter peaks between QRS complexes may look like normal P and T waves. A ventricular rate of 140 to 160 bpm should prompt consideration of the possibility of atrial flutter with 2:1 block.
3. Conditions that cause rapid repetitive tremors (such as Parkinson disease, rigors, shivering, or hepatic tremor) may mimic flutter waves on EKG (known as pseudoflutter).

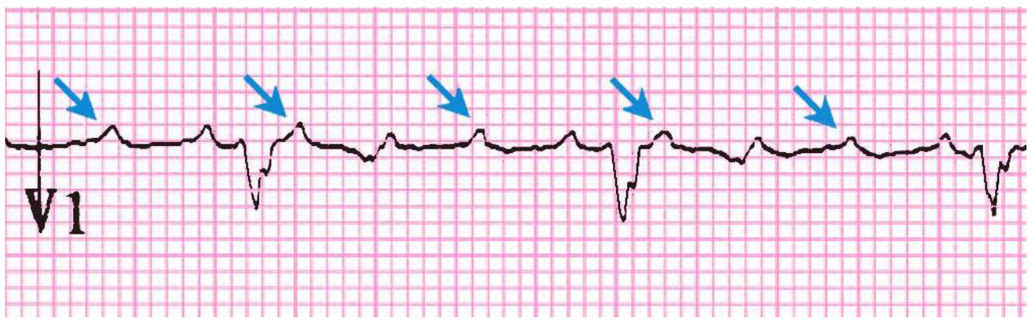


FIGURE 23.29B ■ Atrial flutter with 4:1 block. The flutter waves (arrows marking every other flutter wave) may be mistaken for P and T waves.

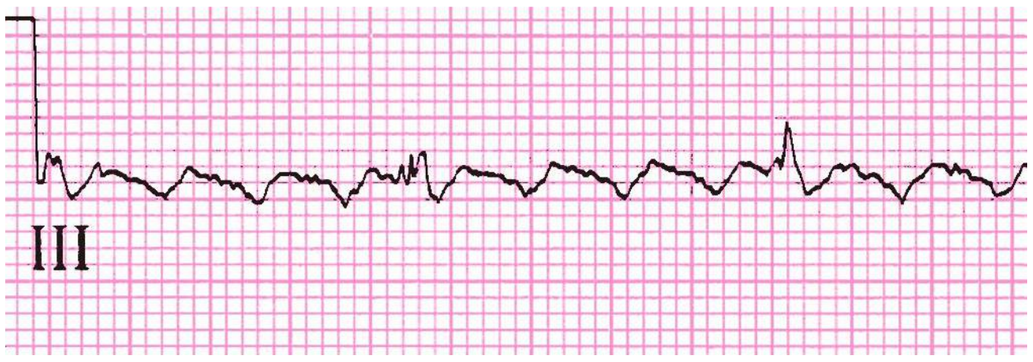


FIGURE 23.29C ■ The "sawtooth" pattern is most apparent in the inferior leads.

MULTIFOCAL ATRIAL TACHYCARDIA

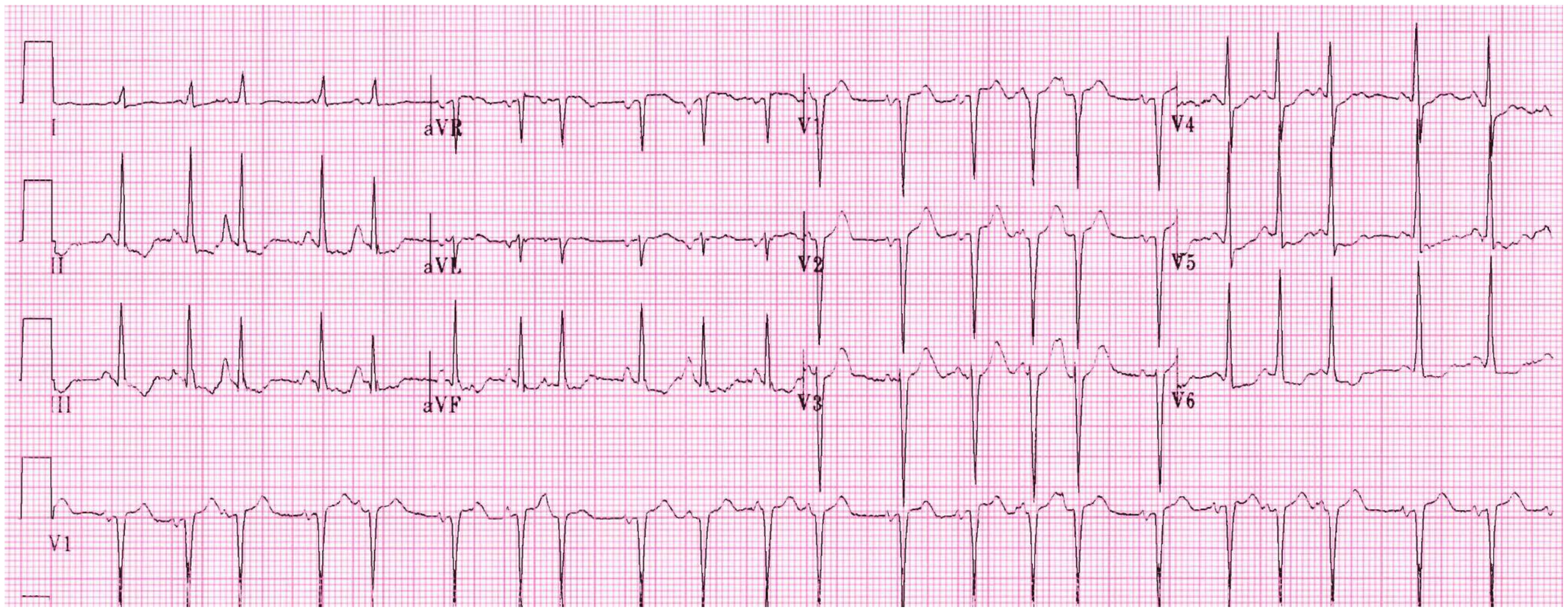


FIGURE 23.30A ■ **Multifocal Atrial Tachycardia.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Multiple P-wave morphologies with heart rate greater than 100 bpm
- A chaotic R-R interval
- Varying PR intervals

Pearls

1. Multiple atrial foci are capable of acting as pacemakers. When irritated by stretching, medications, or certain acute medical conditions, these foci compete in pacing the atria.
2. The different atrial foci produce P waves of different morphologies.
3. Since the atrial foci vary in distance to the AV node, PR intervals vary.
4. Multifocal atrial tachycardia (MAT) usually results from exacerbation of another condition which produces distention or irritation of the atria. The most common cause of MAT is COPD exacerbation. Treatment of the underlying condition should correct the arrhythmia.



FIGURE 23.30B ■ Multiple P morphologies (arrowheads), varying PR intervals (lower double arrows), and varying R-R intervals (upper double arrows) with heart rate greater than 100 bpm.

SUPRAVENTRICULAR TACHYCARDIA (SVT)

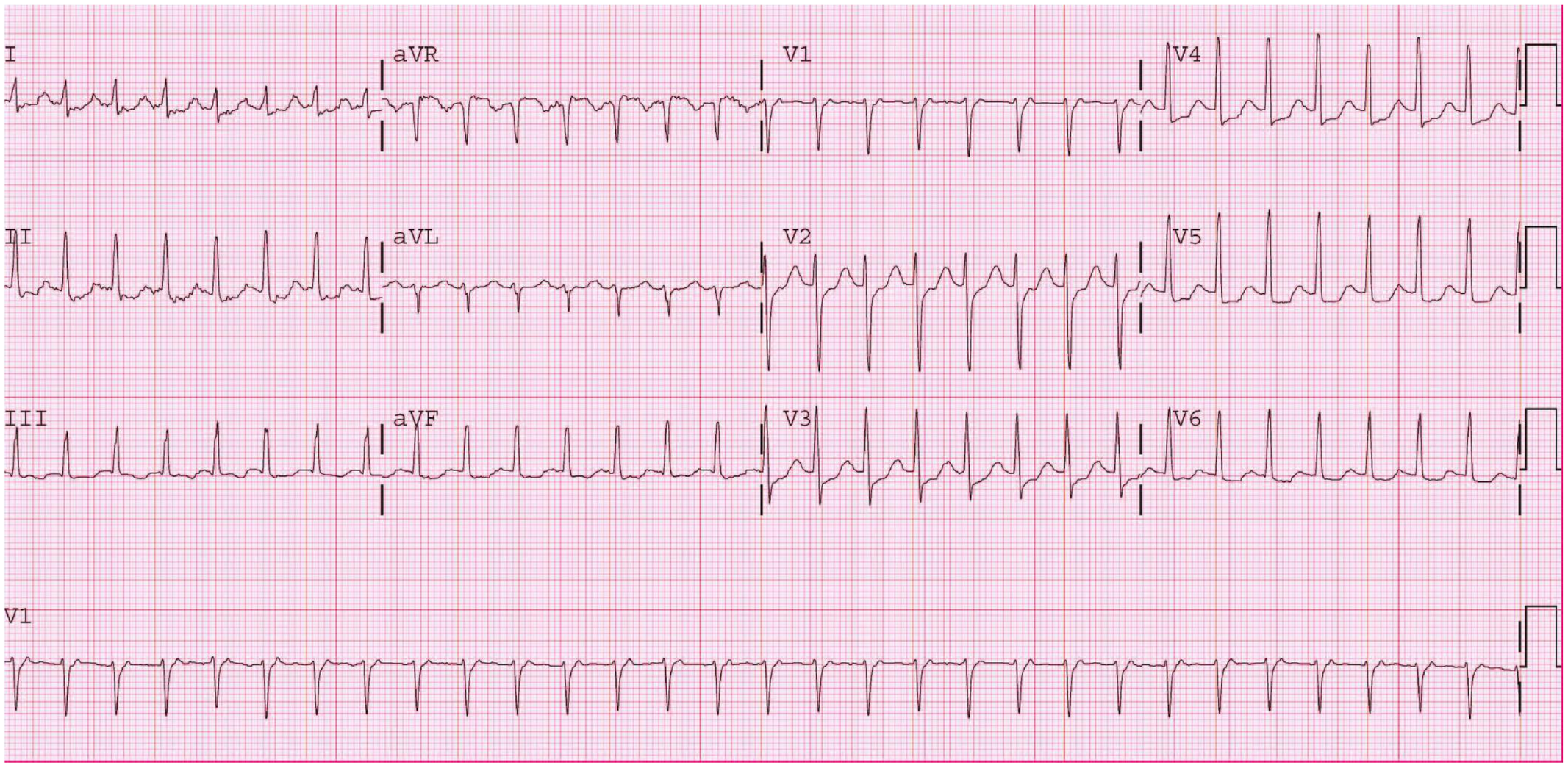


FIGURE 23.31A ■ **Supraventricular Tachycardia (AVNRT).** (ECG contributor: R. Jason Thurman, MD.)

ECG Findings

- Tachycardia, rate usually greater than 140 bpm
- Narrow QRS complex
- Absent, retrograde, or unusual P waves

Pearls

1. SVT occurs when the SA node rhythm is superseded by a faster rhythm, usually originating in the AV node.
2. Three common types are:
   A. Atrial tachycardia—originates from an ectopic focus in the atrium. P waves may have an unusual morphology or may be hidden by the preceding T wave.
   B. AV nodal reentrant tachycardia (AVNRT) occurs when an electrical impulse continues around the AV node in a circular pattern causing rapid depolarizations of the ventricles. Since the AV node is the origin of the atrial depolarization, the P-wave deflection should be inverted if seen (eg, downgoing in II, III, aVF).
   C. AV reentrant tachycardia (AVRT) as seen with bypass tracts outside of the AV node (WPW). Instead of intranodal reentrant activity as seen with AVNRT, an accessory tract provides the reentrant pathway to propagate the tachycardia.
3. Normal deflections seen in the ST segments are relatively wide based. When you see "tight little turns" in the ST segment, you should strongly consider the presence of retrograde P waves.

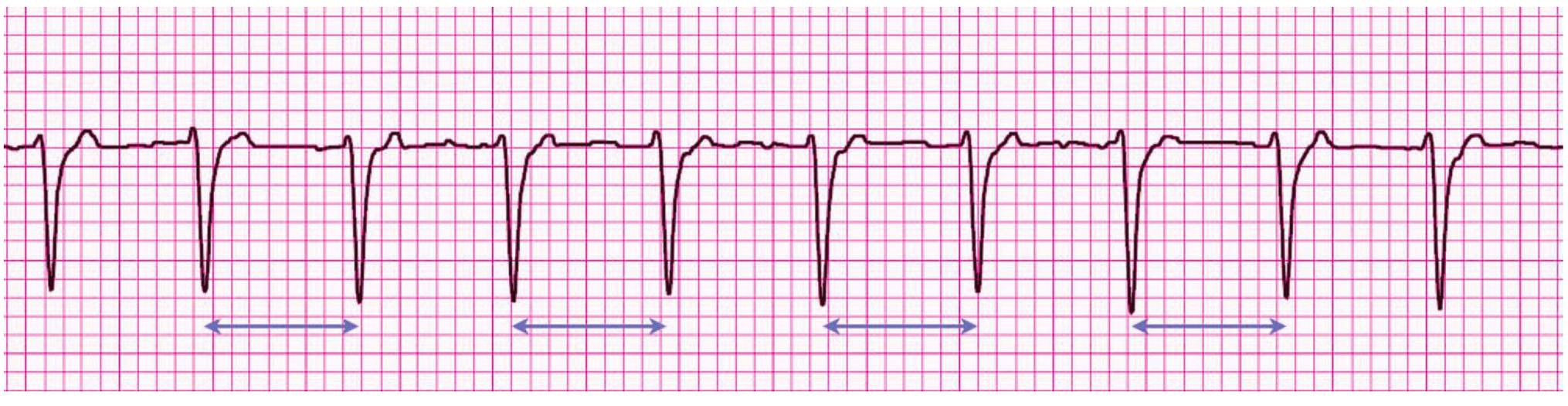


FIGURE 23.31B ■ A narrow-complex tachycardia, with no clear P waves preceding the QRS. R-R intervals are regular (double arrows), differentiating this from fine atrial fibrillation. This rhythm converted to a normal sinus rhythm after the administration of IV adenosine.

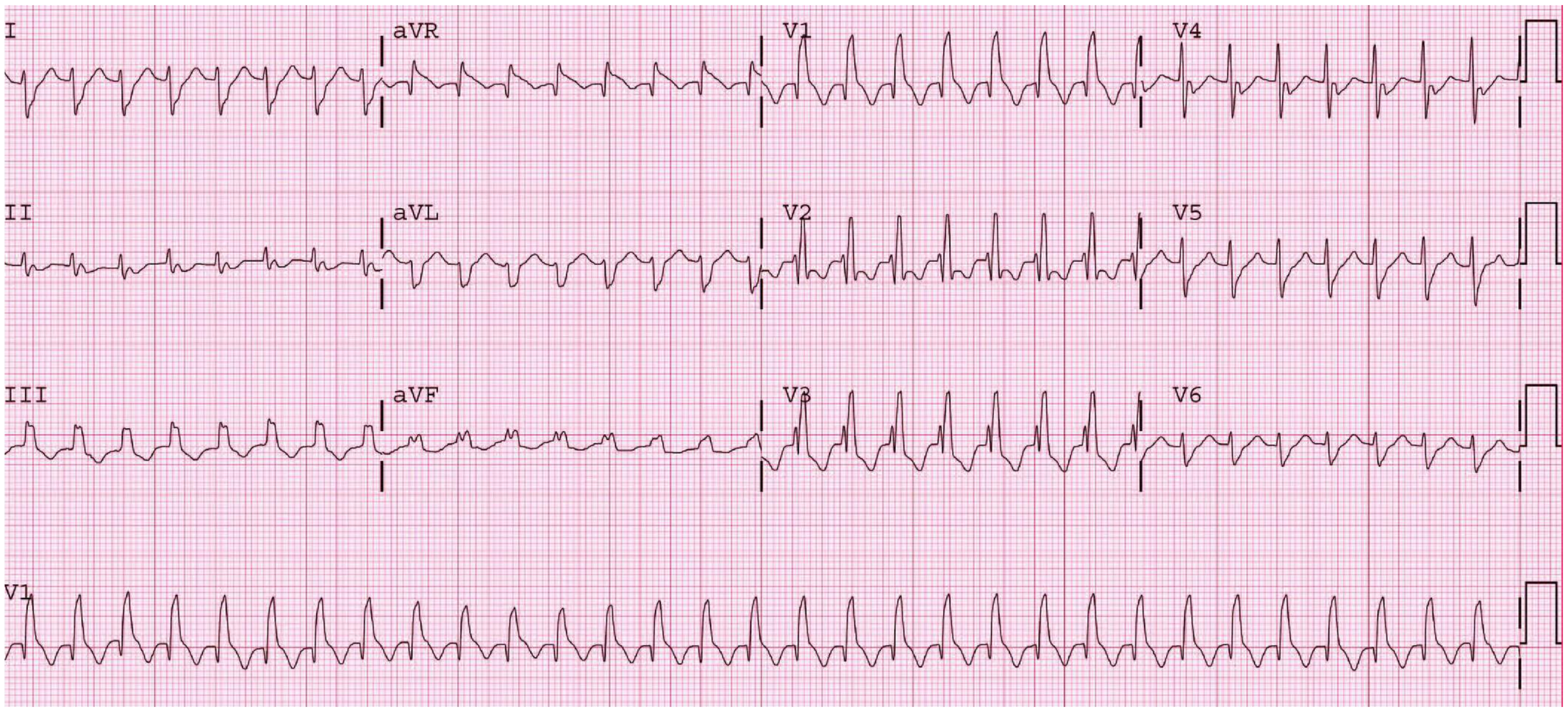


FIGURE 23.32A ■ **Supraventricular Tachycardia with Aberrant Conduction, Underlying RBBB.** (ECG contributor: Walter Clair, MD.)

ECG Findings

- Tachycardia (usually >140 bpm) with a wide QRS complex
- No "capture" or "fusion" beats or AV dissociation as seen with ventricular tachycardia
- QRS morphology consistent with one of the BBB patterns

Pearls

1. When a person with a chronic wide-complex (aberrant) BBB enters an SVT, the ECG will display a wide-complex tachycardia.
2. The rapid rate of an SVT may "outrun" the ventricular conducting system's ability to repolarize quickly, producing a rate-related BBB. The signal then must propagate cell-to-cell, producing a wide-complex tachycardia. A typical bundle branch pattern usually results.
3. An irregularly irregular or chaotic R-R interval, even if subtle, strongly suggests atrial fibrillation or flutter as the culprit SVT. In contrast, the R-R interval of ventricular tachycardia is almost never chaotic.
4. Ventricular rates of 140 to 160 bpm should prompt consideration of atrial flutter with a 2:1 block.

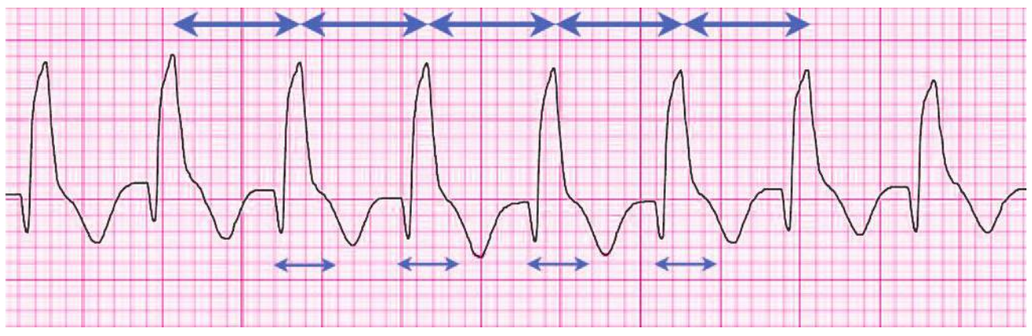


FIGURE 23.32B ■ Wide-complex tachycardia with a rate of 188 bpm. This patient has sudden onset of SVT with a known underlying RBBB. QRS complexes are wide (lower double arrows) and R-R intervals are regular (upper double arrows).

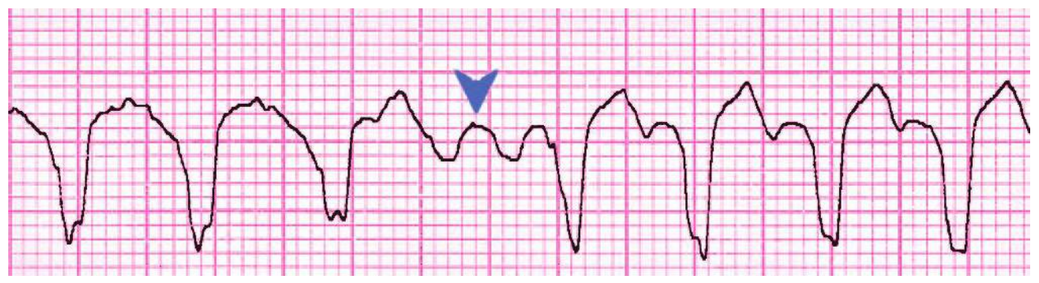


FIGURE 23.32C ■ Wide-complex tachycardia at approximately 150 bpm. The R-R interval is regular, except for one pause, when characteristic atrial flutter waves are apparent (arrowhead).

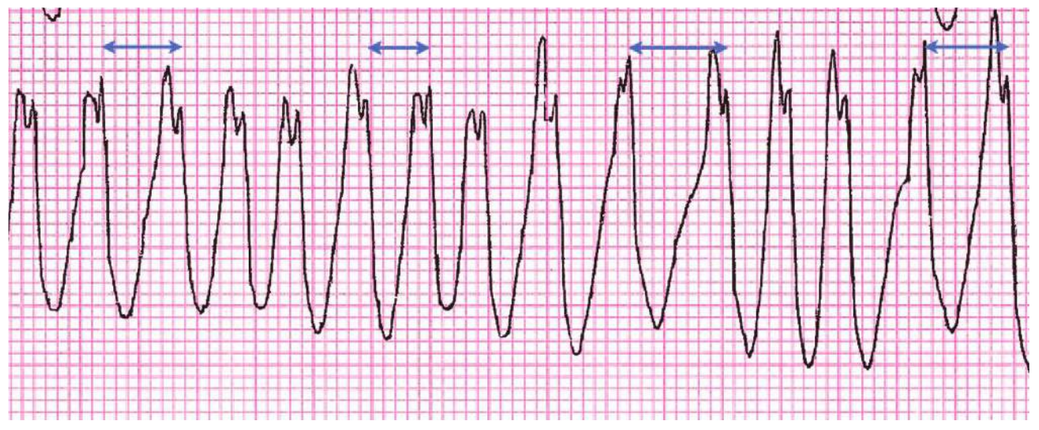


FIGURE 23.32D ■ Irregularity in the R-R interval, as seen most easily in the baseline (double arrows), strongly suggests the presence of rapidly conducted atrial fibrillation with aberrancy.

VENTRICULAR TACHYCARDIA

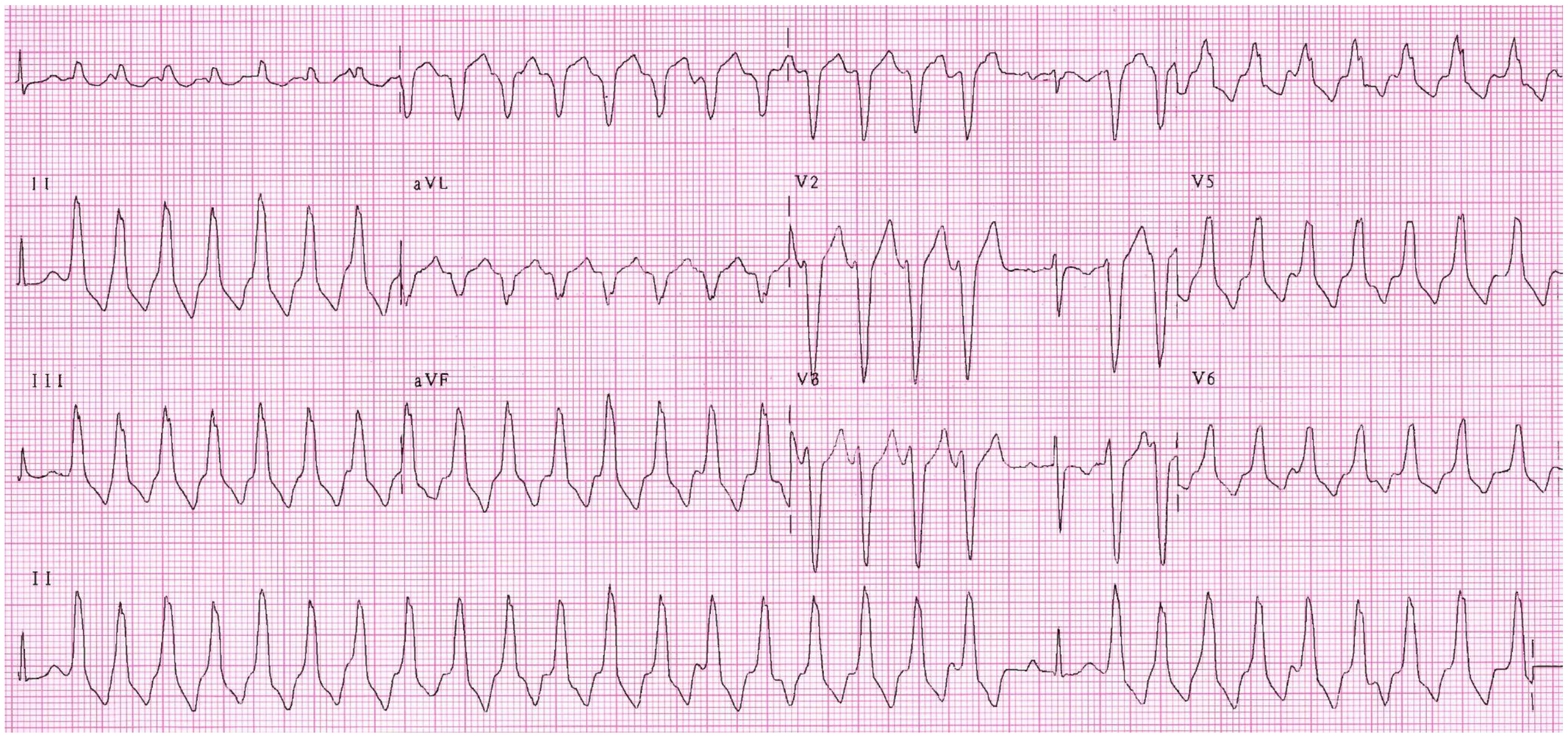


FIGURE 23.33A ■ **Ventricular Tachycardia with Capture Beat.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Tachycardia (usually >120 bpm) with a wide QRS complex.
- AV dissociation is present; P waves may appear periodically in the T wave or baseline.
- "Capture" beats may occur if atrial depolarization occurs prior to the intrinsic firing of the ventricle.
- "Fusion" beats may occur if atrial depolarization passes through the AV node at the same time as the intrinsic ventricle depolarization, producing a QRS that appears to be different or narrower than the other VT QRS complexes.

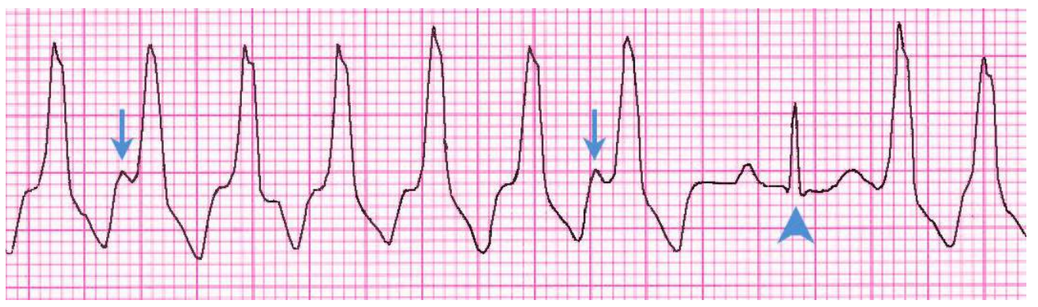


FIGURE 23.33B ■ A wide-complex tachycardia. AV dissociation is apparent, as P waves occasionally appear superimposed in the ST segment or just prior to the QRS (arrows). A capture beat occurs following a lapse in the VT (arrowhead).

Pearls

Findings more suggestive of VT versus aberrant SVT or antidromic WPW:

1. Apparent AV dissociation, capture, or fusion beats.
2. An unusual QRS axis, between 180 and 270 degrees.
3. Precordial concordance, in which QRS complexes in the precordium are all upgoing or all downgoing.
4. A completely upgoing QRS in V_1.
5. Predominately downgoing QRS in V_4, V_5, and V_6.

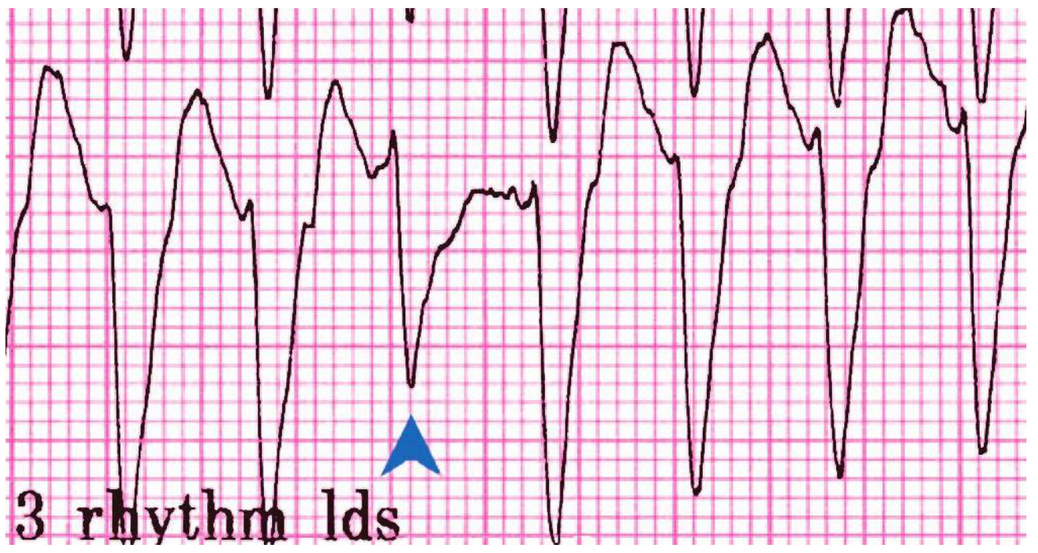


FIGURE 23.33C ■ Another example of ventricular tachycardia, featuring a fusion beat (arrowhead). (ECG contributor: Marc Mickiewicz, MD.)

VENTRICULAR FLUTTER

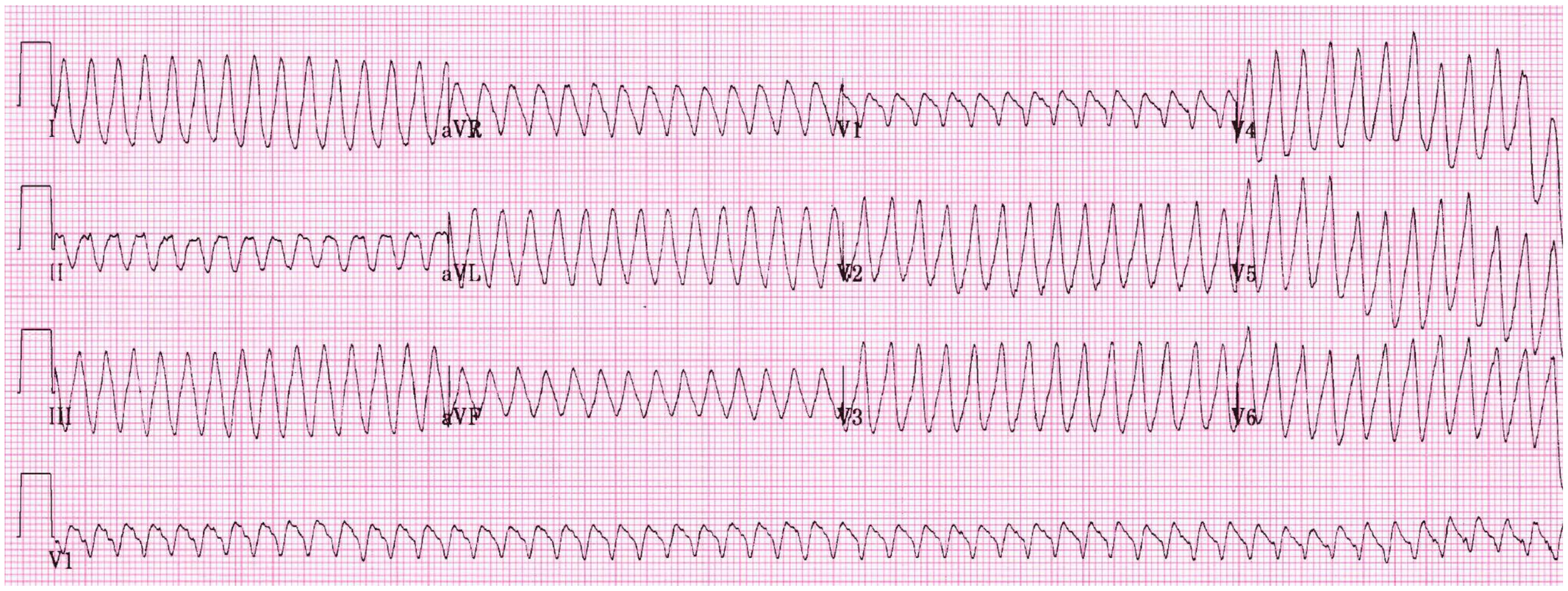


FIGURE 23.34A ■ **Ventricular Flutter.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Tachycardia with a wide monomorphic QRS complex.
- Ventricular rate may be very rapid (300 bpm).
- Sine wave appearance with regular large oscillations.

Pearls

1. Imagine an atrial flutter sawtooth with much larger amplitude.
2. When you see a very rapid wide-complex tachycardia (>240 bpm), consider ventricular flutter or WPW with atrial fibrillation or flutter.
3. WPW with atrial flutter may be indistinguishable from ventricular flutter.
4. Ventricular flutter is treated as ventricular tachycardia and usually leads to ventricular fibrillation if not promptly corrected with antiarrhythmic medications or electrical cardioversion.
5. Patients with such a rapid rate are almost always unstable. Emergent cardioversion is indicated. If the patient appears to be stable enough for chemical cardioversion, choose a medication which is safe to use with WPW, such as procainamide or amiodarone.

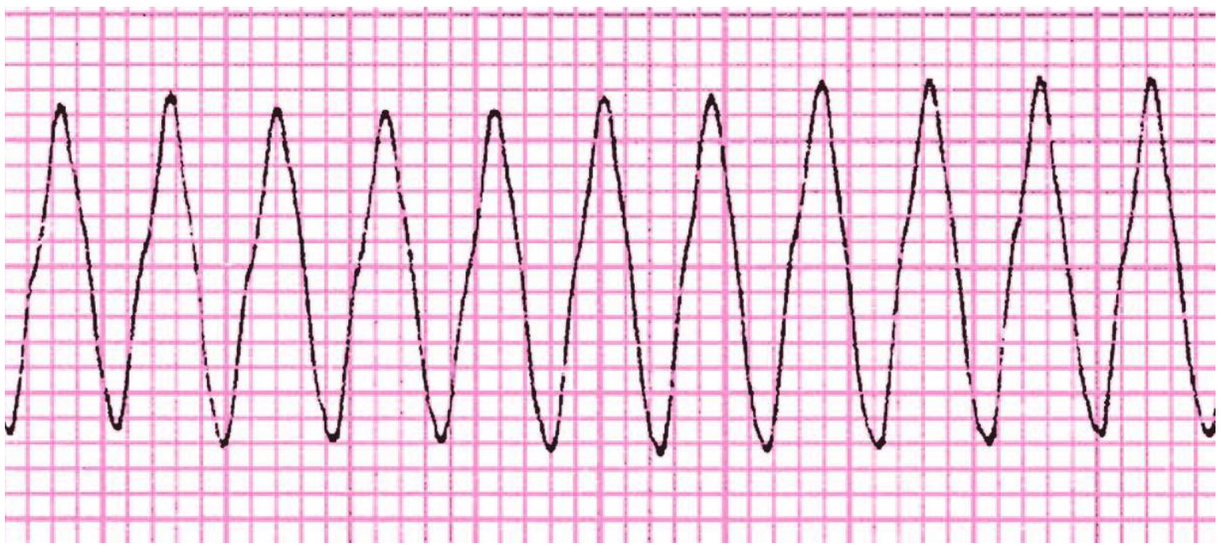


FIGURE 23.34B ■ Very rapid, regular, wide-complex tachycardia with sine-wave appearance. The rate in this example is 330 bpm. Differential diagnosis includes WPW with atrial flutter.

TORSADES DE POINTES

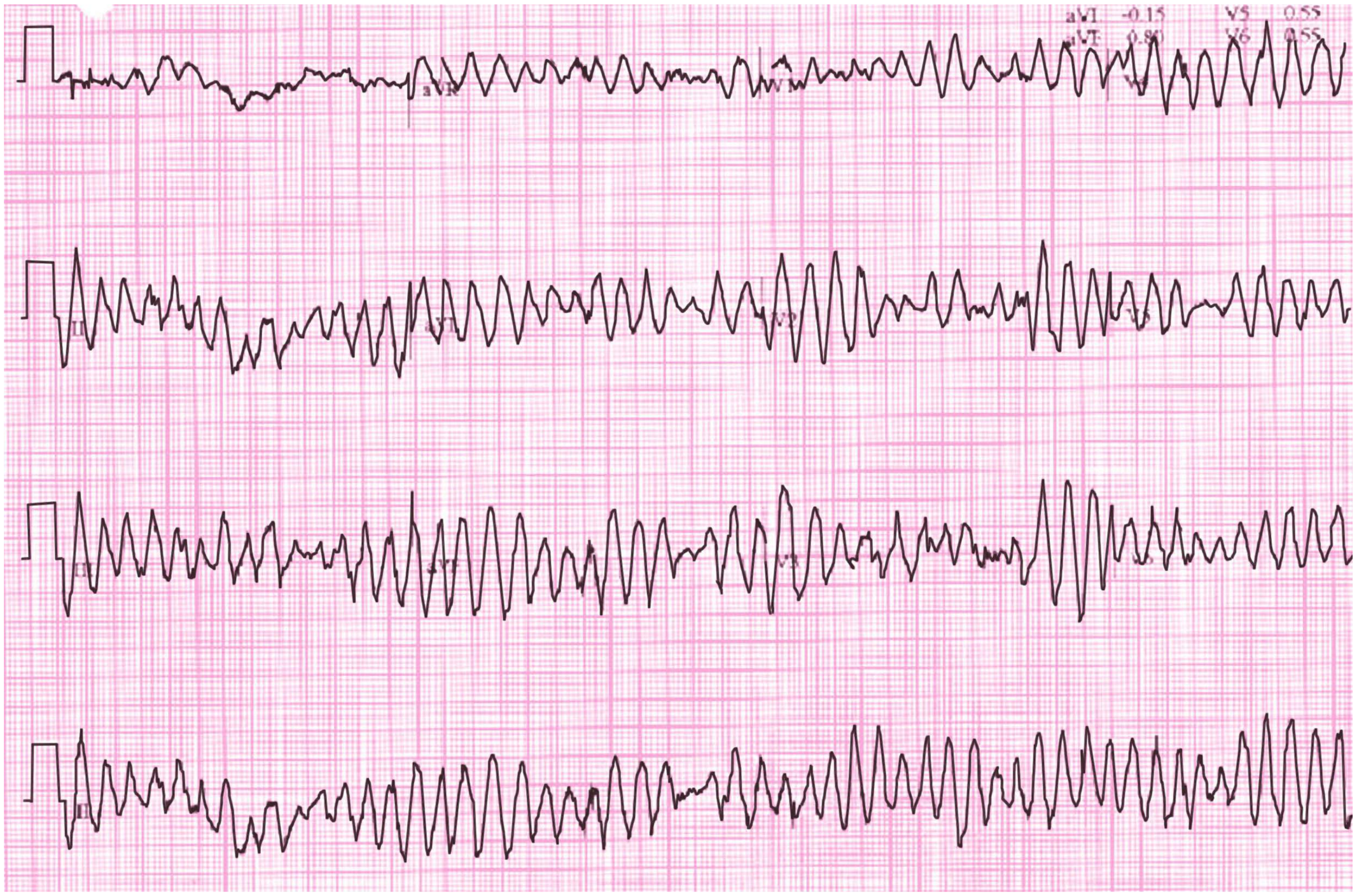


FIGURE 23.35A ■ **Torsades de Pointes.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Wide-complex tachycardia with QRS polymorphism.
- QRS morphology changes gradually throughout the tracing, appearing to rotate around the baseline to the opposite direction.

Pearls

1. The cyclic rotation of the QRS complex gives rise to the term torsades de pointes (French meaning "twisting of the points").
2. Common precipitating factors are etiologies which prolong the QT interval, such as medications, electrolyte abnormalities (hypocalcemia, hypomagnesemia, hypokalemia), and hereditary disorders.
3. Torsades can also occur in the setting of myocardial ischemia without prolongation of the QT interval.
4. Torsades is treated as a ventricular tachycardia, usually requiring defibrillation. Magnesium is used as an adjunct therapy to normal ACLS protocols.

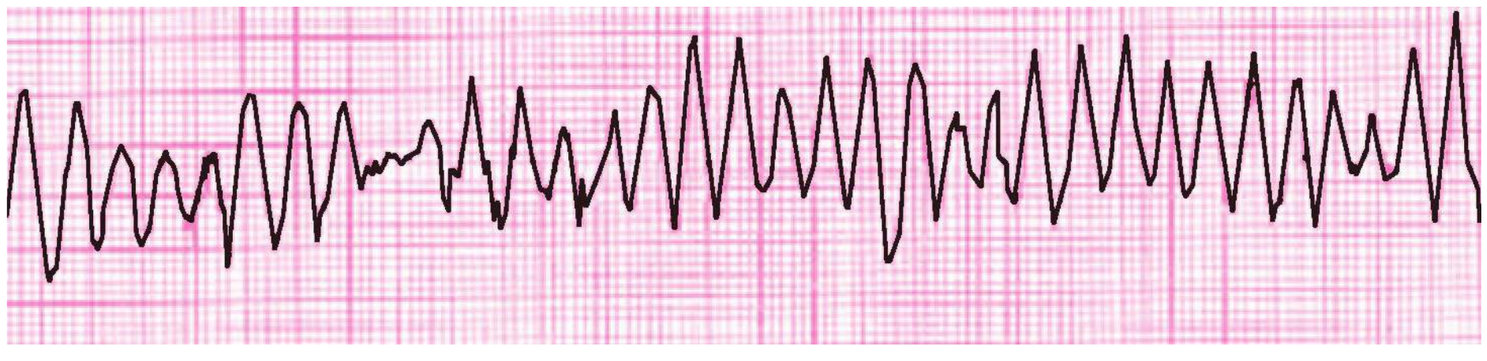


FIGURE 23.35B ■ Very rapid wide-complex tachycardia with sine-wave appearance and fluctuations in the amplitude of the QRS complexes consistent with torsades de pointes.

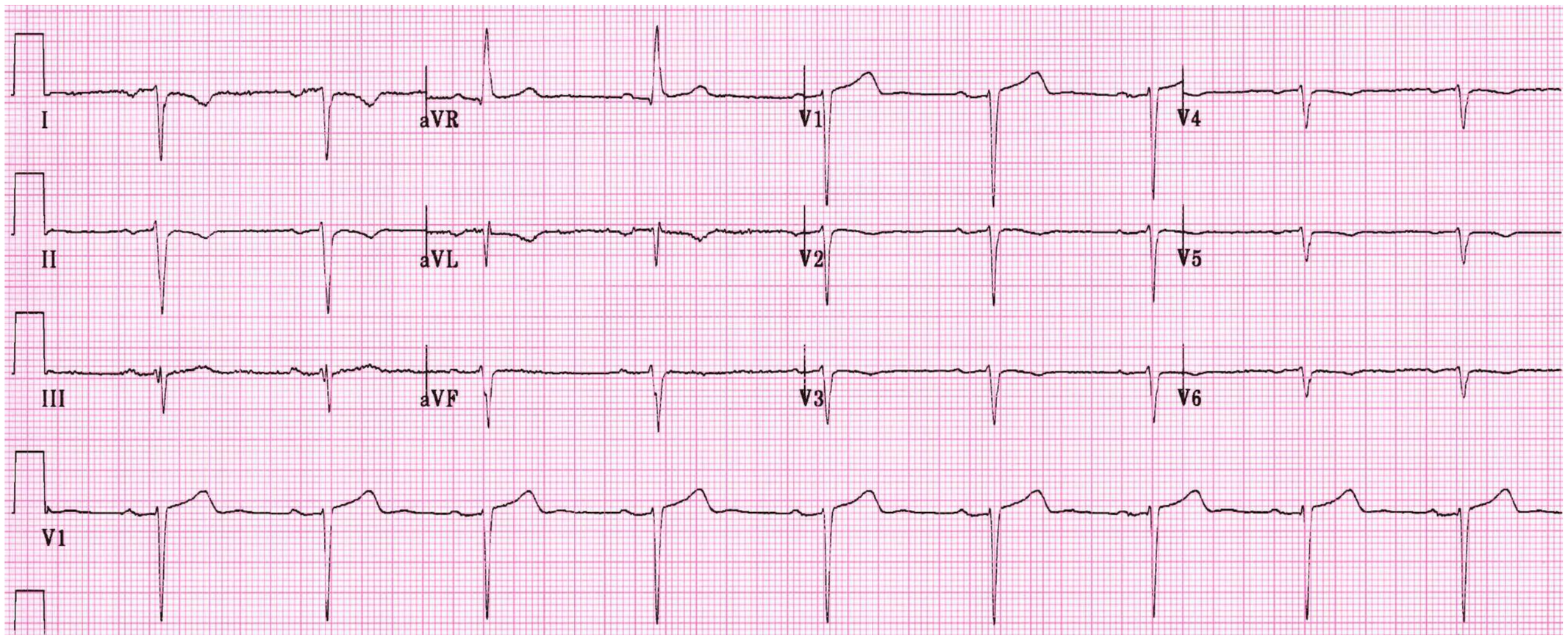


FIGURE 23.36A ■ **Dextrocardia.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- P, QRS, and T are downgoing in lead I, a mirror image of normal.
- QRS deflections in V_4 to V_6 are small and downgoing.

Pearls

1. The orientation of the heart in the chest cavity is reversed with the predominant electrical activity moving left to right (as opposed to right to left).
2. Normally placed precordial leads in a patient with dextrocardia are actually placed over the thinner right ventricle instead of the left ventricle.
3. Reversing all EKG leads should produce an essentially normal EKG.
4. A "reversed" lead I ('downward' QRS) and aVR ('upward' QRS) with normal-appearing V leads strongly suggests accidental limb lead reversal.

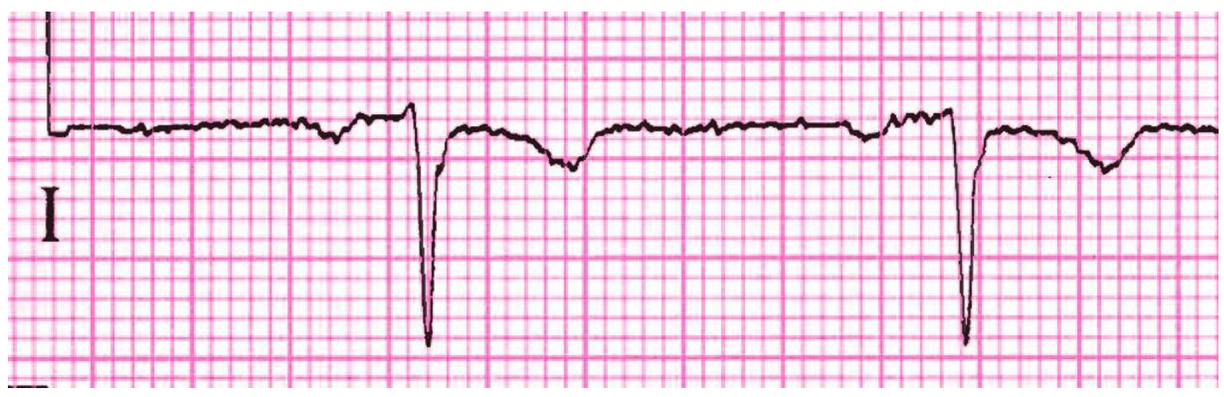


FIGURE 23.36B ■ The P wave, QRS, and T wave are downgoing in lead I. Differential diagnosis includes limb lead reversal and dextrocardia. The 12-lead ECG above represents dextrocardia as evidenced in the abnormal precordial leads.

LEFT VENTRICULAR HYPERTROPHY

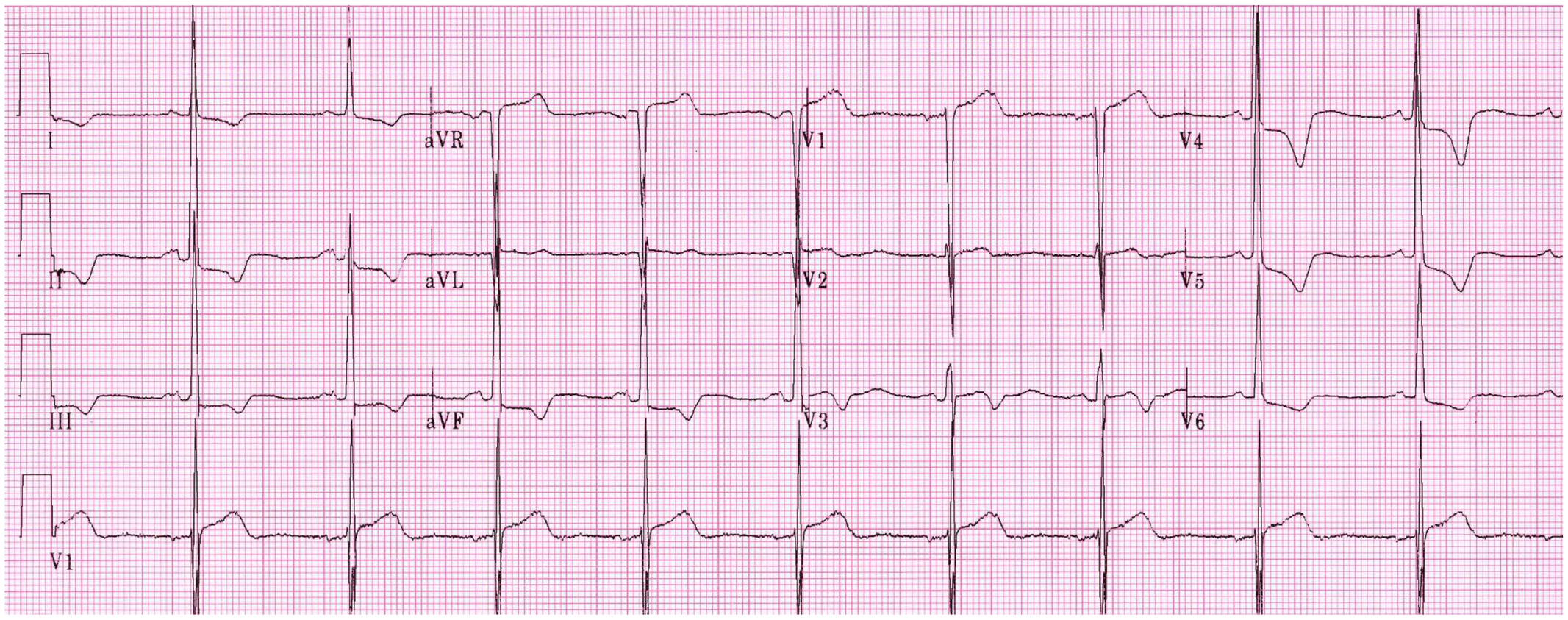


FIGURE 23.37A ■ **Left Ventricular Hypertrophy with Strain Pattern.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Large S waves in anterior precordial leads
- Large R waves in lateral precordial leads
- R wave in aVL + S wave in V_3 greater than 28 mm in males, greater than 20 mm in females
- S wave in V_1 + R wave in V_5 or V_6 greater than 35 mm if age over 40, greater than 40 mm if age 30 to 40, greater than 60 mm if age 16 to 30
- R wave in aVL greater than 11 mm
- T waves deflected opposite to QRS complex (strain pattern)

Pearls

1. The muscular left ventricle normally dominates the QRS morphology.
2. LVH can produce related changes such as left atrial abnormality. It may also produce ST-T wave changes, particularly in opposite deflections of the T wave with respect to the main deflection of the QRS complex. These ST deflections, often referred to as "LVH with strain," may be confused with ischemia.
3. LVH is often a sign of disease states such as systemic hypertension or aortic stenosis.
4. LVH may manifest on the ECG in many different ways. Several different systems for diagnosing LVH by ECG have been promoted. No one system is adequately sensitive and specific enough to warrant exclusion of all others.

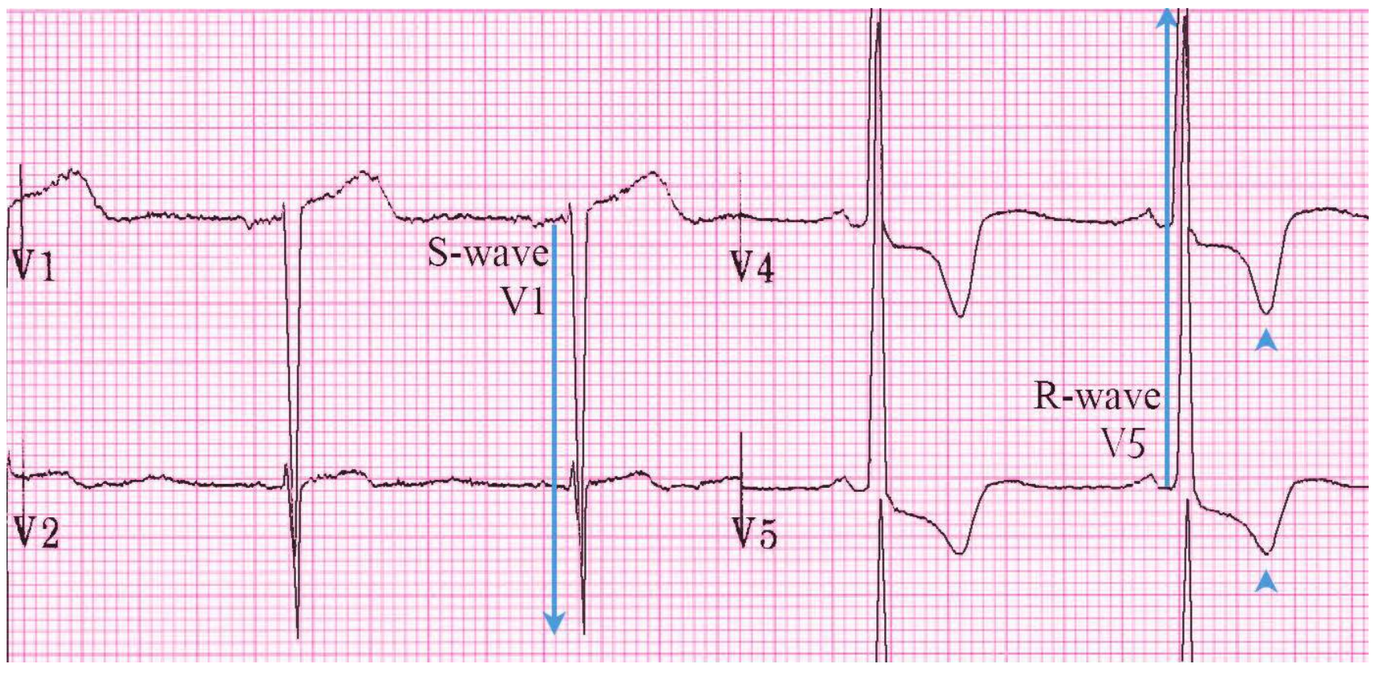


FIGURE 23.37B ■ The QRS deflections are very large. The R wave in V_5 plus the S wave in V_1 total approximately 75 mm (arrows). ST downsloping to inverted T waves in V_4 and V_5 (arrowheads) may also be seen, a finding often referred to as "LVH with strain."

RIGHT VENTRICULAR HYPERTROPHY

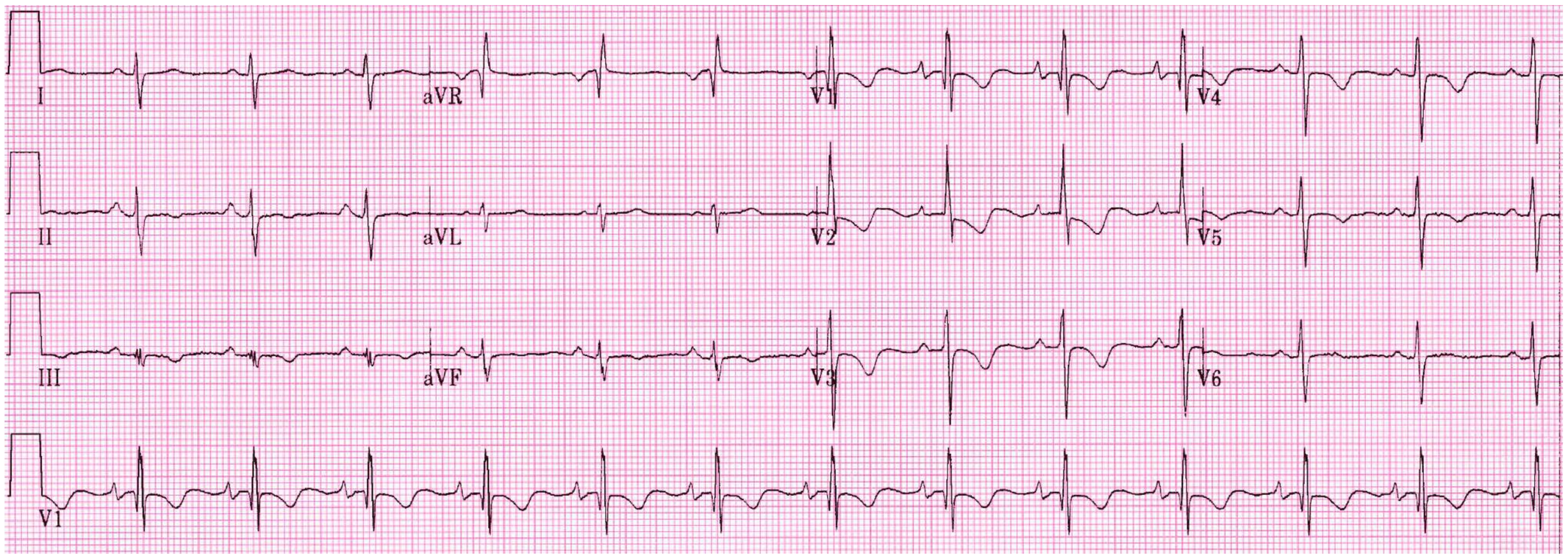


FIGURE 23.38A ■ **Right Ventricular Hypertrophy.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- S-wave amplitude increases in lateral leads (V_5, V_6, I, aVL).
- R-wave amplitude increases in aVR, V_1, V_2, and may exceed the S-wave amplitude (especially in lead V_1).
- Right axis deviation (> +90 degrees).
- T-wave inversions in relation to QRS complex.

Pearls

1. Hypertrophy of the right ventricle causes characteristic EKG changes as the predominant electrical signal of the left ventricle is overcome.
2. As right ventricular hypertrophy (RVH) persists, right atrial enlargement (RAE) may occur as seen in the example (P-wave amplitude in V_1 >1.5 mm).
3. Congenital heart disease, pulmonic or mitral stenosis, and pulmonary hypertension are common causes of RVH.

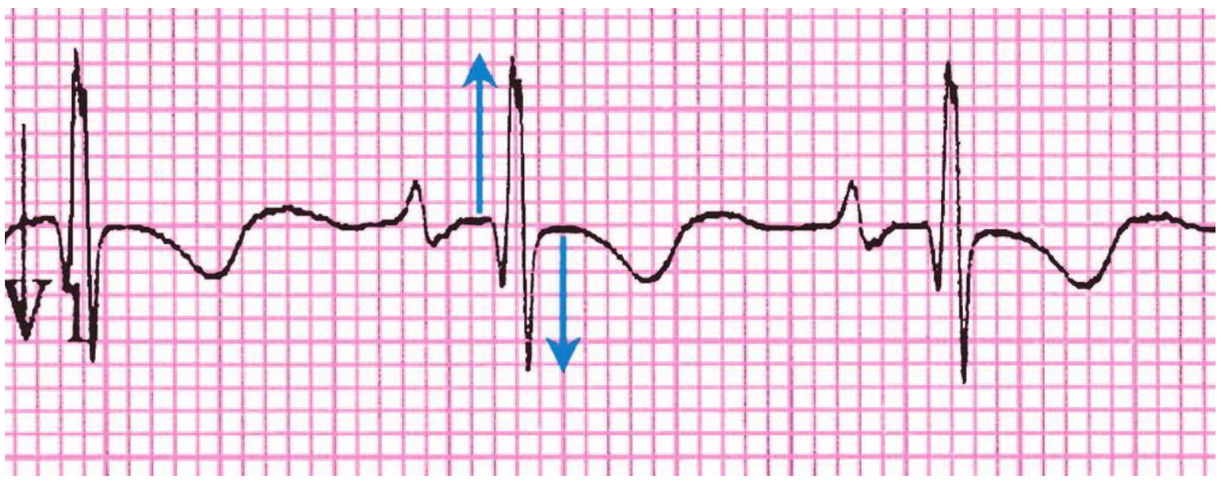


FIGURE 23.38B ■ The R-wave amplitude exceeds the S-wave amplitude (arrows) in lead V_1. In addition, the P-wave upward deflection exceeds 1.5 mm, indicating concomitant right atrial enlargement.

LEFT ATRIAL HYPERTROPHY

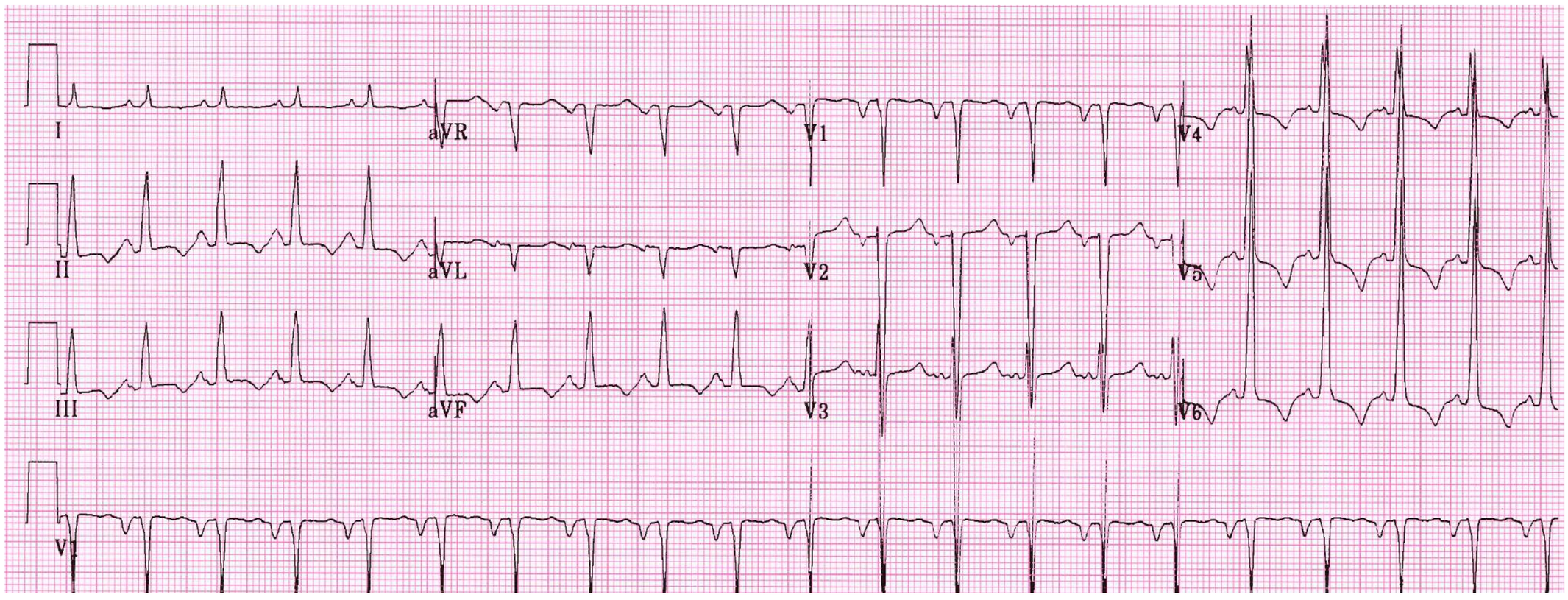


FIGURE 23.39A ■ **Left Atrial Hypertrophy with LVH Present.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Increased duration (width) of the P wave without affecting its upward amplitude (as commonly seen with right atrial abnormalities)
- Negative P-wave deflection in lead V_1, with width and depth greater than 0.04 seconds (one small box)
- Wide P wave in lead II
- Notched P wave in II, III, or aVF with duration greater than or equal to 0.12 seconds (also known as P-mitrale)

Pearls

1. Normal P-wave morphology has an amplitude of less than 2.5 mV (2.5 vertical boxes) and a duration (width) of less than 120 ms (three small boxes).
2. The left atrium depolarizes after the right atrium and therefore has the most effect on the second portion of the P wave.
3. Causes of left atrial abnormality or P-mitrale include valvular heart disease (mitral and aortic), CAD, cardiomyopathy, hypertension, and LVH.

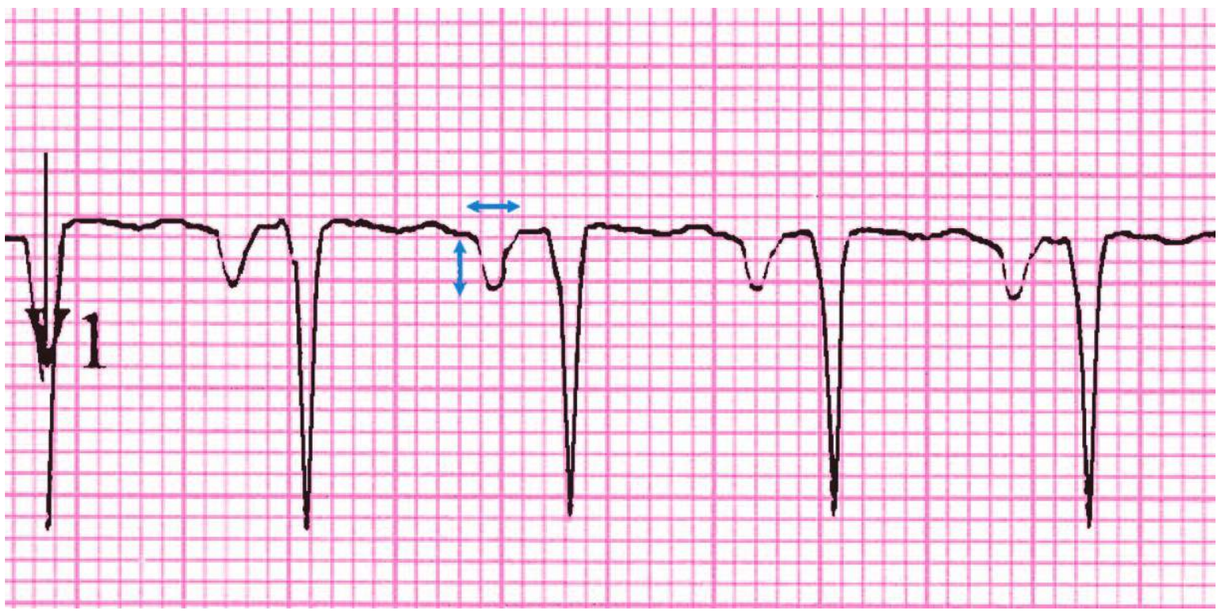


FIGURE 23.39B ■ The P wave in V_1 is downgoing. The downgoing segment is wider and deeper than one small block (double arrows).

RIGHT ATRIAL HYPERTROPHY

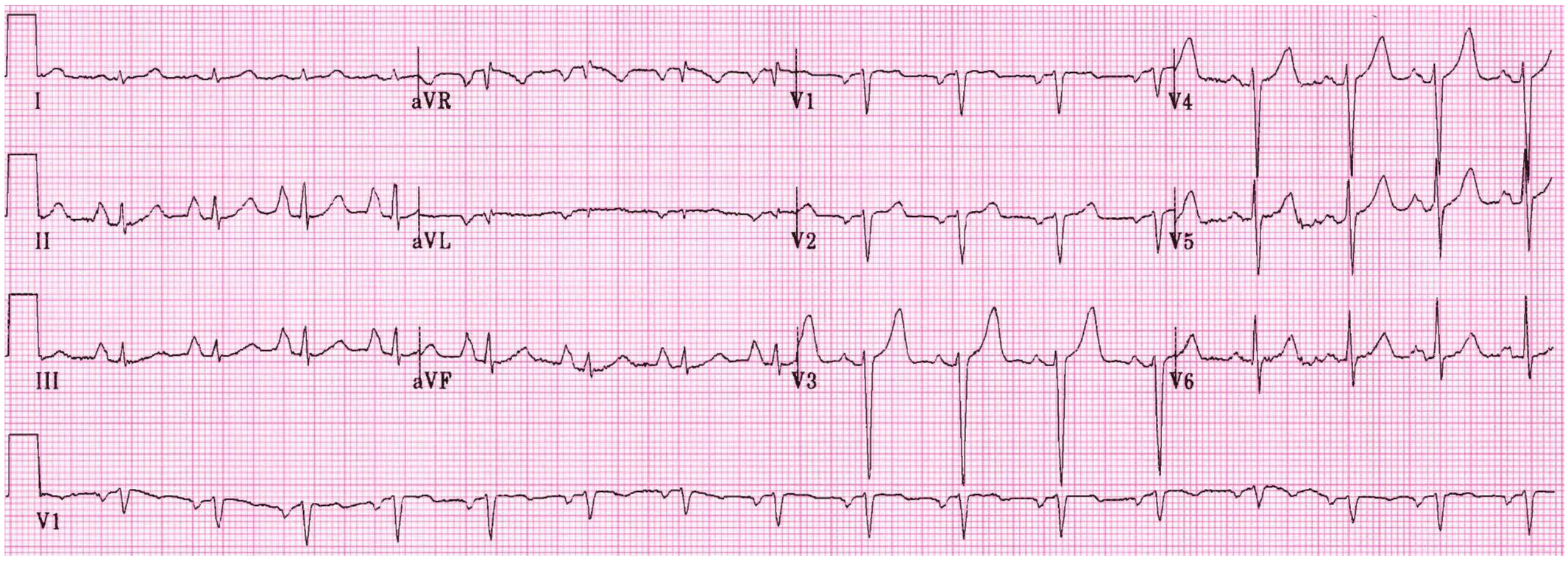


FIGURE 23.40A ■ **Right Atrial Hypertrophy.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Increased amplitude of P wave without affecting duration (as commonly seen with left atrial abnormalities)
- Peaked P waves (>2.5 mm) in leads II, III, aVF (also known as P pulmonale)
- P-waves upward deflection greater than 1.5 mm in lead V_1 or V_2

Pearls

1. Normal P-wave morphology has amplitude of less than 2.5 mV (2.5 small vertical boxes) and duration (width) of less than 120 ms (three small boxes).
2. The right atrium depolarizes before the left atrium and therefore has the most effect on the first portion of the P wave.
3. RAE is often associated with RVH, COPD, some congenital heart diseases, and pulmonary hypertension, and may be seen transiently in pulmonary embolus.

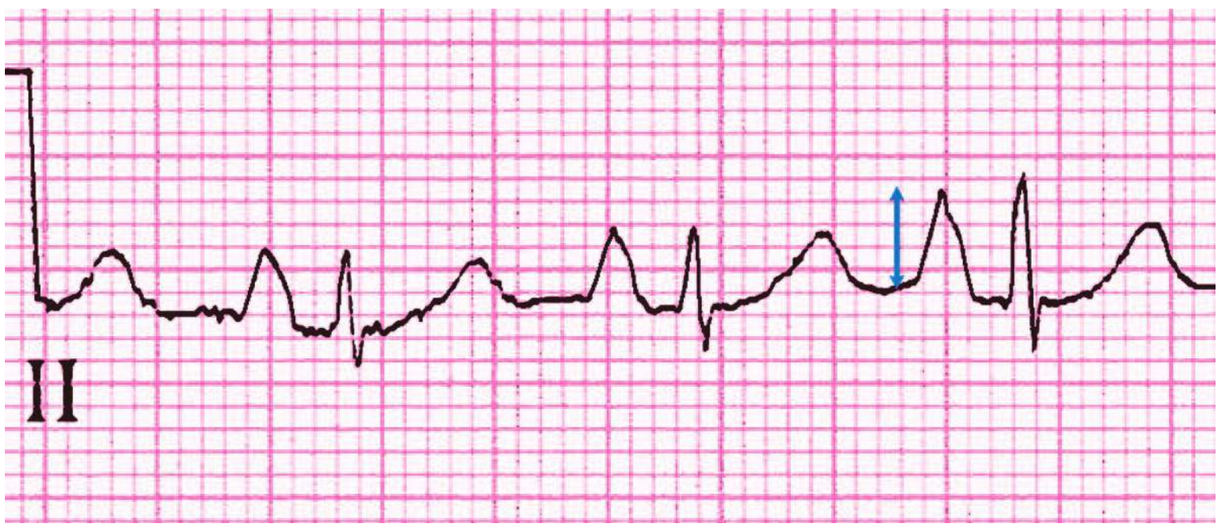


FIGURE 23.40B ■ The P wave in lead II (an inferior lead) is greater than 2.5 mm in amplitude (double arrow).

HYPERTROPHIC CARDIOMYOPATHY

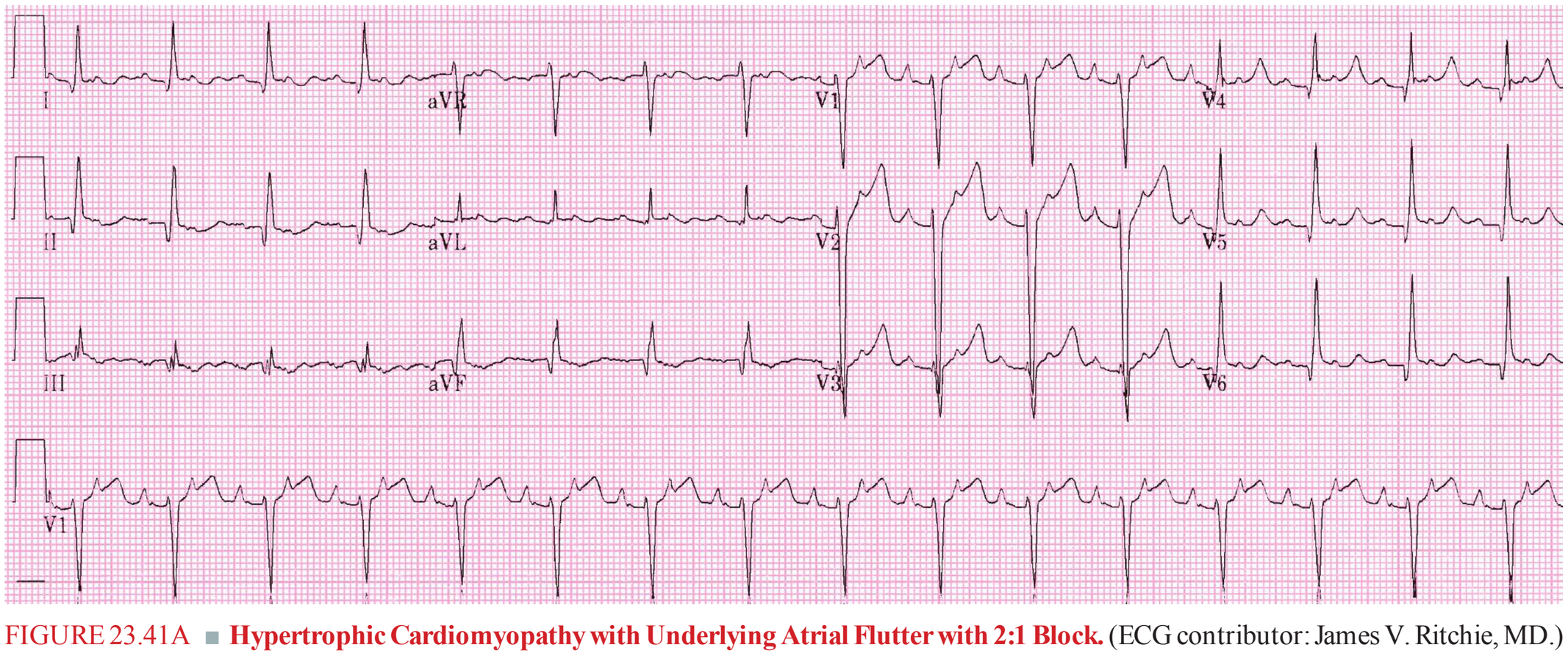


FIGURE 23.41A ■ **Hypertrophic Cardiomyopathy with Underlying Atrial Flutter with 2:1 Block.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- High-voltage QRS suggesting LVH
- Left atrial abnormality
- Prominent Q waves, especially in the lateral precordial leads
- Deep S waves in anterior precordial leads
- Poor R-wave progression across precordium
- Lateral T-wave inversions

Pearls

1. The ventricular myocardium hypertrophies abnormally, either concentrically or focally. Left ventricle outflow obstruction from the hypertrophy may lead to LVH without dilation.
2. Hypertrophic cardiomyopathy (HCM) is also known as idiopathic hypertrophic subaortic stenosis (IHSS), hypertrophic obstructive cardiomyopathy (HOCM), and muscular subaortic stenosis (MSS).
3. ECG changes are variable and usually do not include all listed above.
4. Always consider this condition in young athletes with syncope or unusual dyspnea.
5. HCM is associated with a systolic ejection murmur that diminishes with increases in preload (having the patient squat) and augments with decreases in preload (Valsalva maneuver).

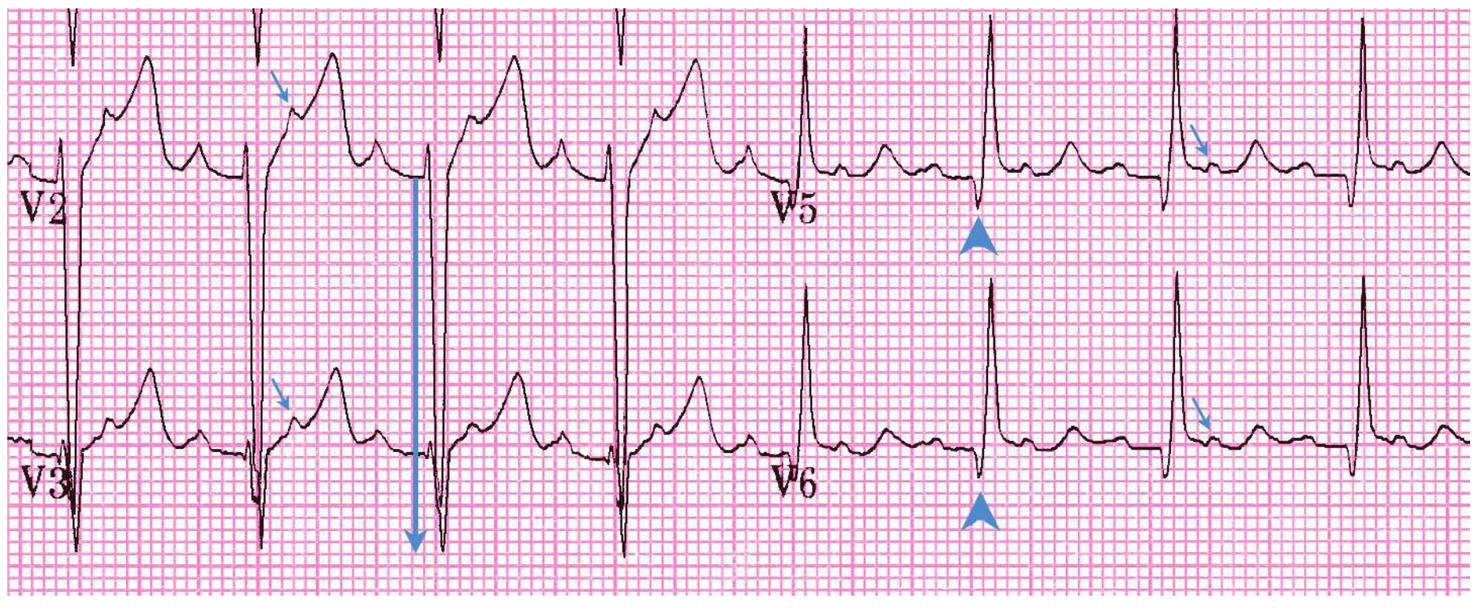


FIGURE 23.41B ■ Deep S-wave voltage (28 mm S in V_2, large arrow), and narrow Q waves in V_5 and V_6 (arrowheads). This patient also has atrial flutter with 2:1 block. The additional P waves appear in the ST segments (small arrows).

BRUGADA SYNDROME

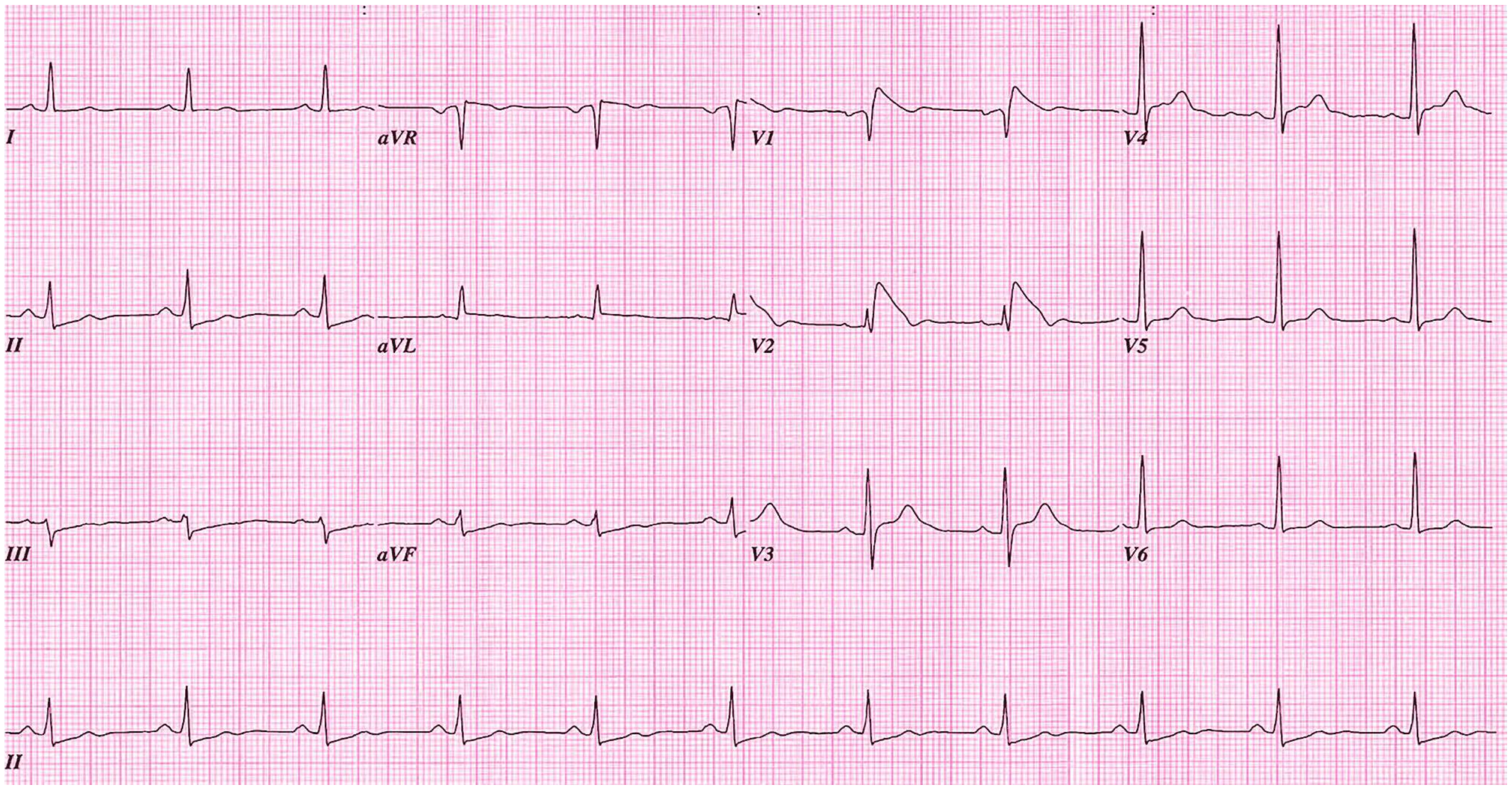


FIGURE 23.42A ■ **Brugada Syndrome.** (ECG contributor: Michael L. Juliano, MD.)

ECG Findings

- Incomplete or complete RBBB pattern in leads V_1, V_2, and sometimes V_3
- ST elevation of at least 1 mV in leads V_1, V_2, and sometimes V_3 followed by T inversion (convex pattern) or upright T (concave, or "saddle back" pattern)

Pearls

1. This syndrome was first described in individuals who experienced sudden cardiac death with structurally normal hearts, but congenitally abnormal ion channels in myocyte cell membranes have been associated with the disease.
2. Concern for spontaneous ventricular dysrhythmia is high.
3. Consultation with a cardiologist is recommended for electrophysiological testing and intracardiac defibrillator placement.

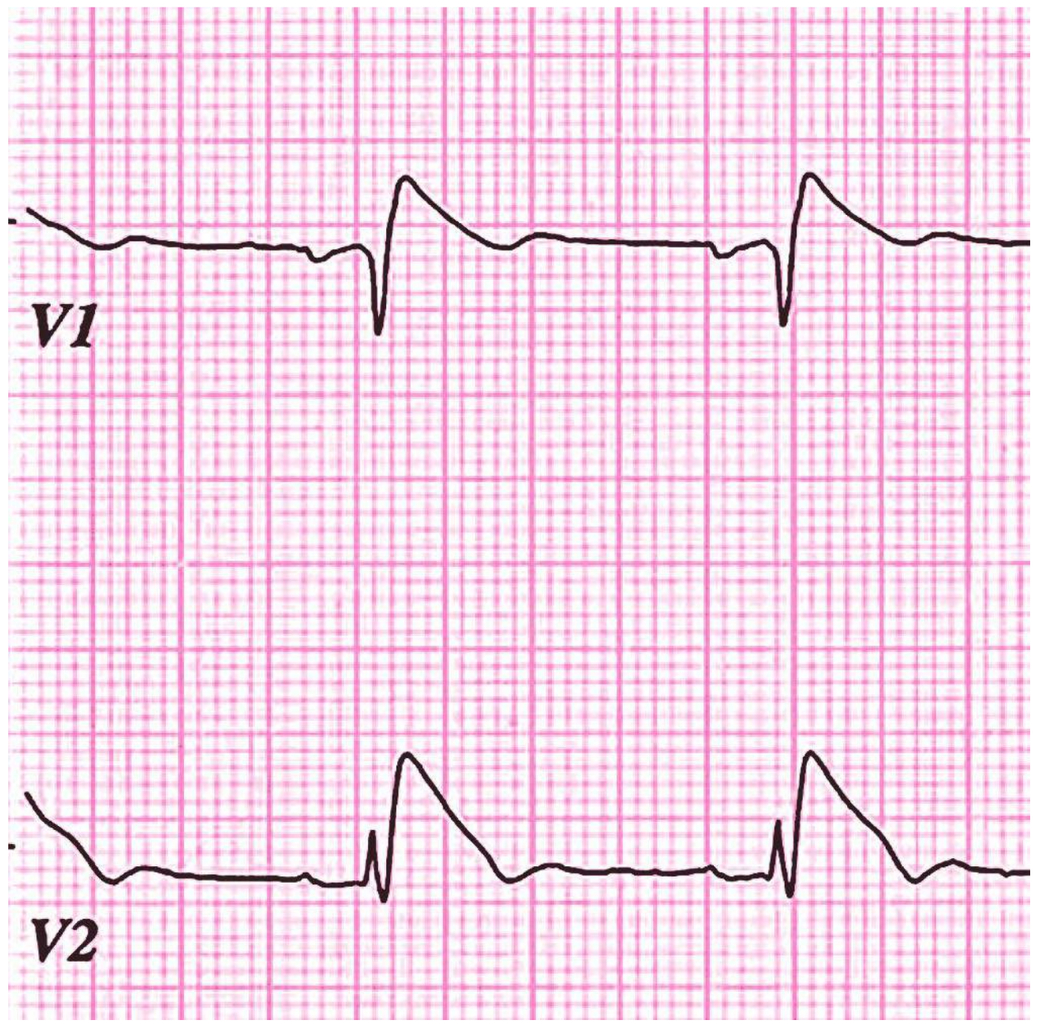


FIGURE 23.42B ■ RBBB pattern with ST elevation (type 1 Brugada syndrome).

WOLFF-PARKINSON-WHITE SYNDROME

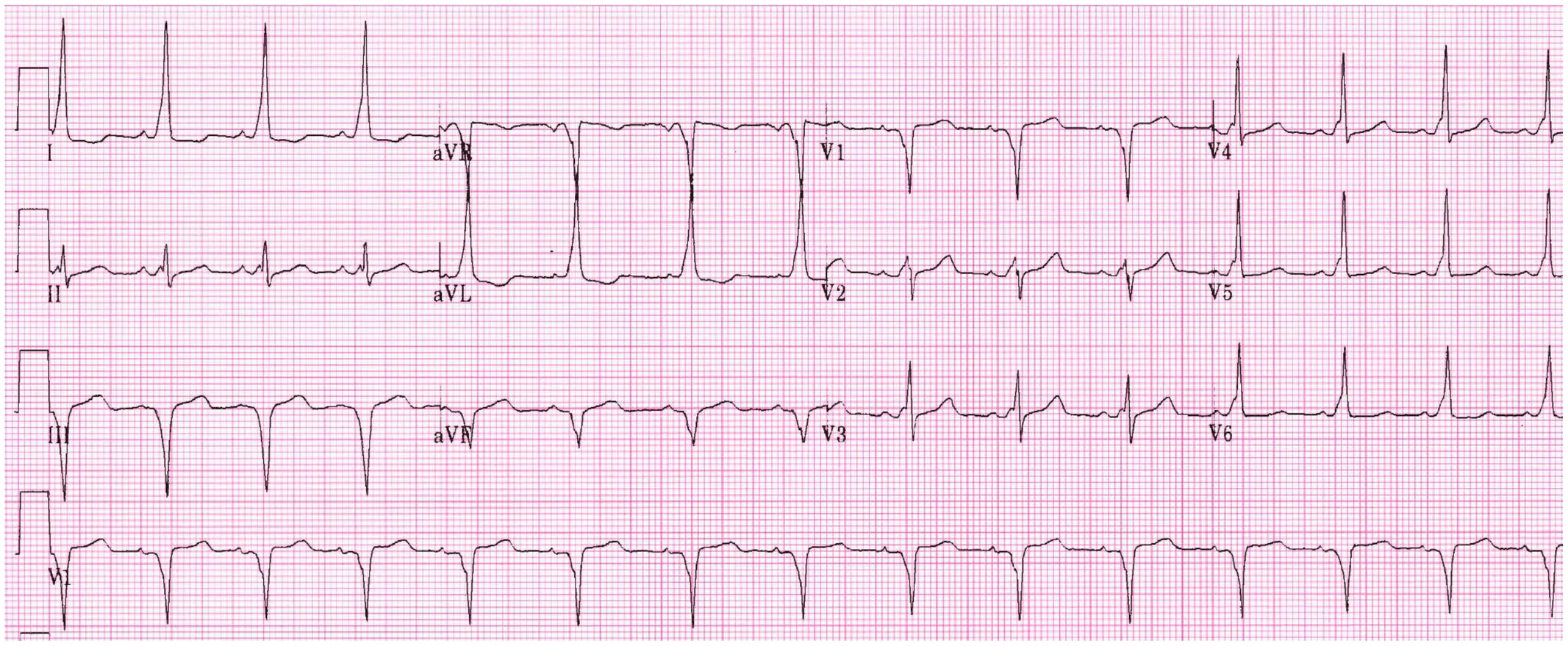


FIGURE 23.43A ■ **Wolff-Parkinson-White Syndrome.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Normal P waves
- Shortened PR interval
- Prolonged QRS interval
- Delta waves (slurring of the initial upstroke of R wave)

Pearls

1. Accessory tracts from the atria to the ventricles lead to depolarization of ventricles without using the AV node as the primary connecting route.
2. Tachycardia associated with WPW may be mistaken for ventricular tachycardia. Suspect WPW if the QRS complex is wide and tachycardia is extreme (ventricular rate > 240).
3. Do not treat this tachycardia with AV nodal blocking agents (calcium channel blockers, β-blockers, digoxin). This may lead to unopposed ventricular stimulation through the accessory tract and may worsen the tachycardia.
4. Procainamide, amiodarone, and cardioversion are accepted methods for conversion of a tachycardia associated with WPW.
5. Depolarization via the accessory pathway may produce "pseudo-Q waves" as shown in leads III and aVF in this example.

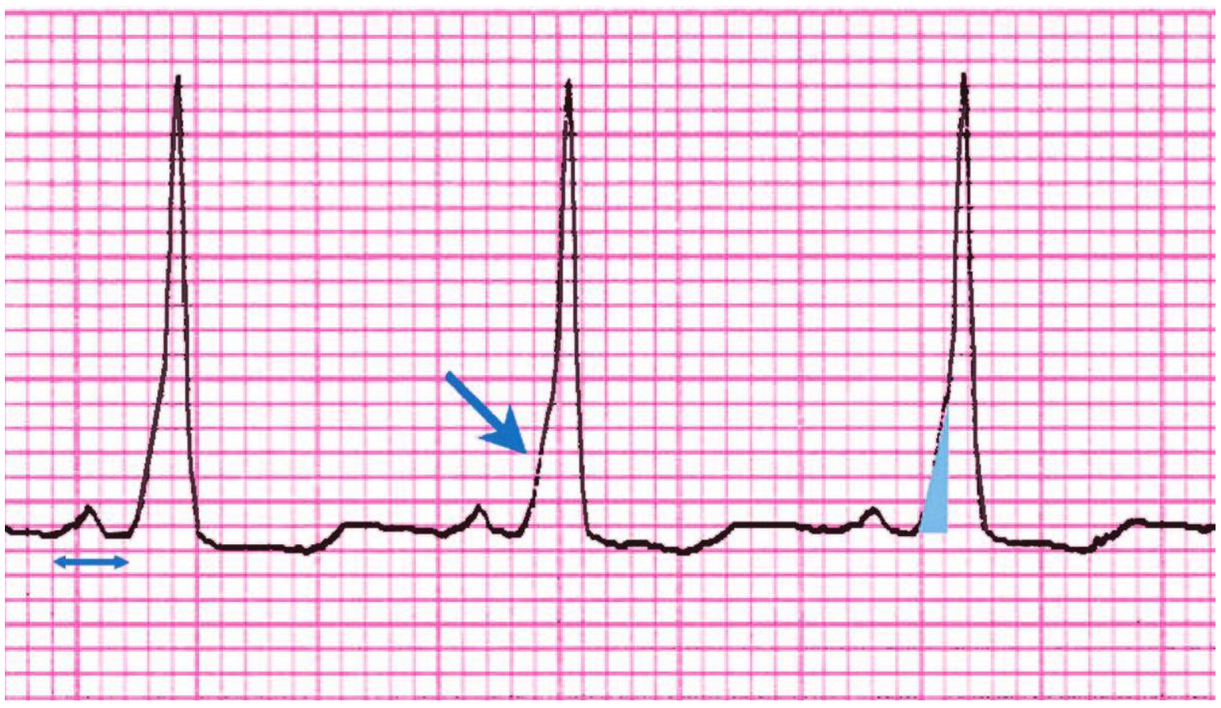


FIGURE 23.43B ■ The PR interval is shortened (double arrow) and a delta wave (upsloping initial QRS segment) is seen (arrow, shaded area).

HYPOTHERMIA

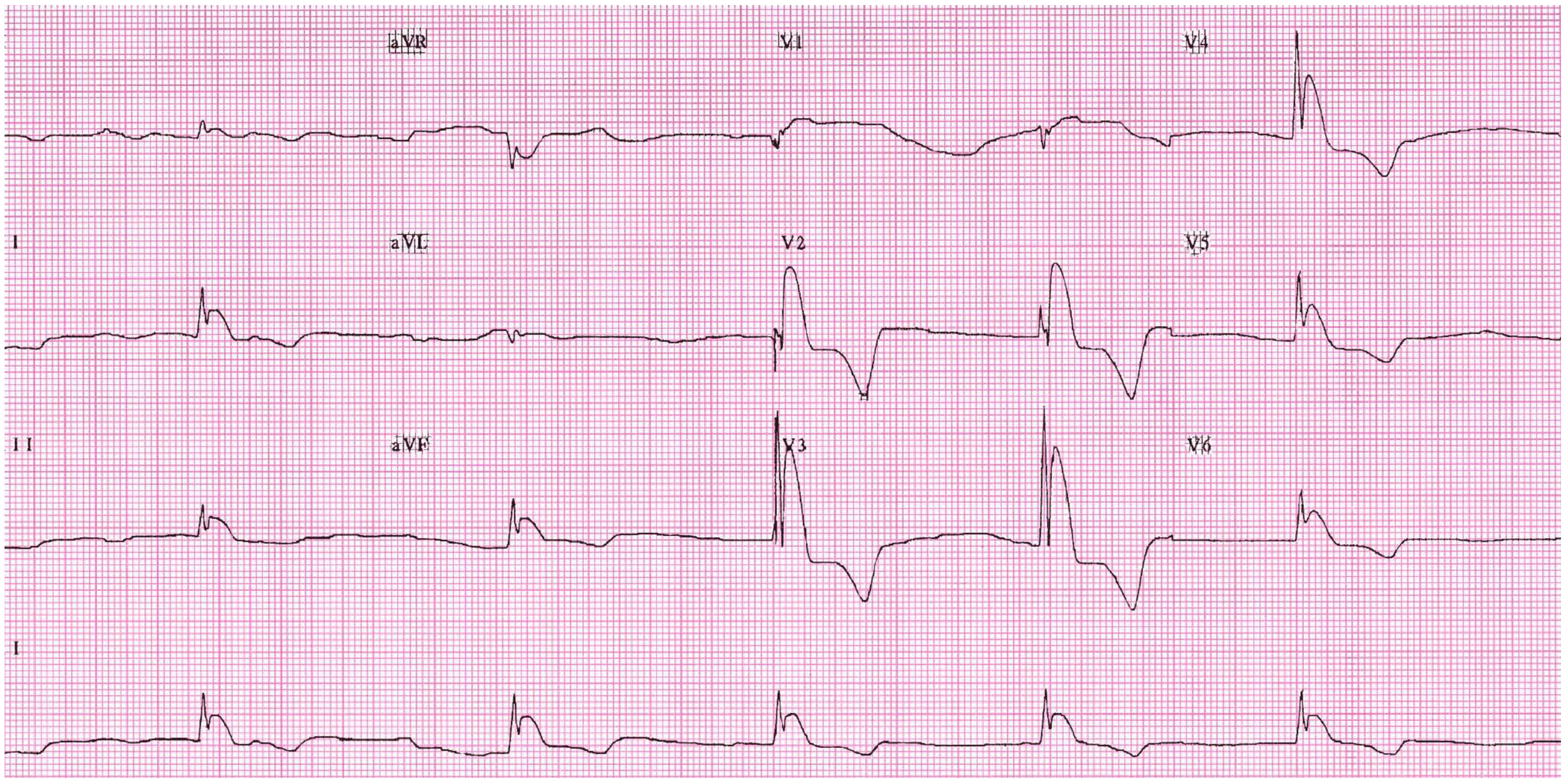


FIGURE 23.44A ■ **Hypothermia with Osborne Waves ("J" Waves) Present.** (ECG contributor: Michael L. Juliano, MD.)

ECG Findings

- Sinus bradycardia or atrial fibrillation with slow ventricular response.
- PR, QRS, and QT intervals are typically prolonged.
- Osborne or "J" wave (a positive deflection of the terminal portion of the QRS complex). The J wave may be subtle or large and "humped."

Pearls

1. The hypothermic patient's rhythm slows, proceeding from sinus bradycardia to atrial fibrillation with slow response and may proceed to other arrhythmias including ventricular fibrillation and asystole.
2. The amplitude of the "J" wave corresponds to the degree of hypothermia.
3. Myocardial damage and EKG changes associated with hypothermia are not necessarily due to low temperature. They may be indirectly caused by systemic circulatory issues such as hypoperfusion.
4. Defibrillation and many medications may be ineffective in the hypothermic patient. Rapid rewarming is indicated as an initial and critical resuscitative measure.

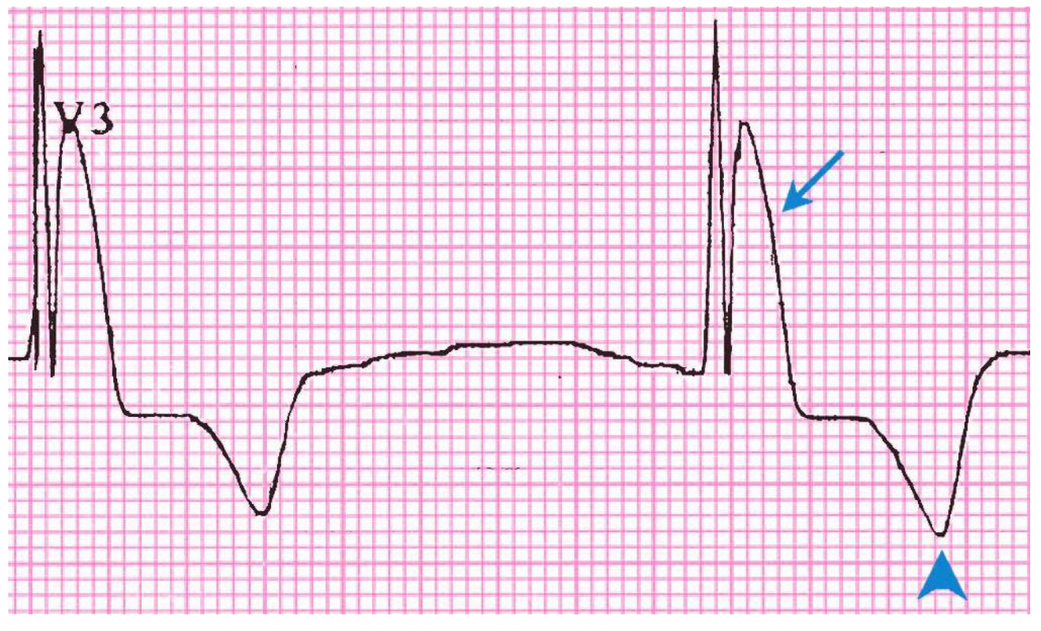


FIGURE 23.44B ■ A large Osborn wave (J wave) (arrow) follows the QRS, and is distinct from the T wave (arrowhead).



FIGURE 23.44C ■ This is a more typical appearance of a J wave (arrow).

HYPERKALEMIA

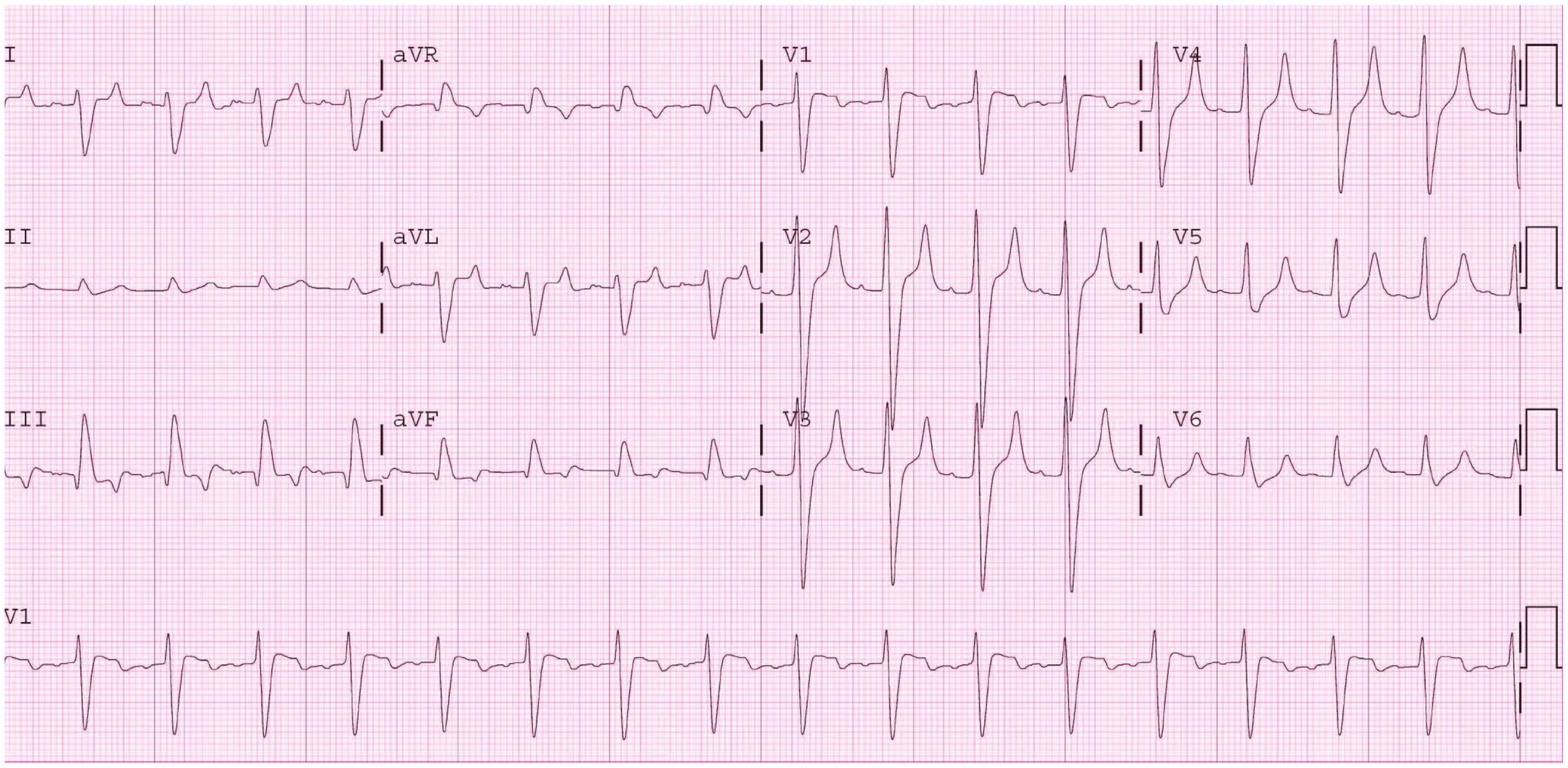


FIGURE 23.45A ■ **Hyperkalemia (K 7.1).** (ECG contributor: R. Jason Thurman, MD.)

ECG Findings

Findings are variable but tend to correlate with increasing serum potassium levels following the order below:

- Peaked T waves, tented with a narrow base (may be >10 mm high in precordial leads and/or >6 mm in limb leads)
- QT interval shortening
- QRS complex widening
- PR interval prolongation
- Decreased P-wave amplitude
- As potassium levels approach and exceed 8.0 mEQ/L:
  - Indiscernible P waves
  - Sine wave appearance of QRS-T complex
  - Left or right bundle branch pattern
  - Ventricular tachycardia, fibrillation, or asystole

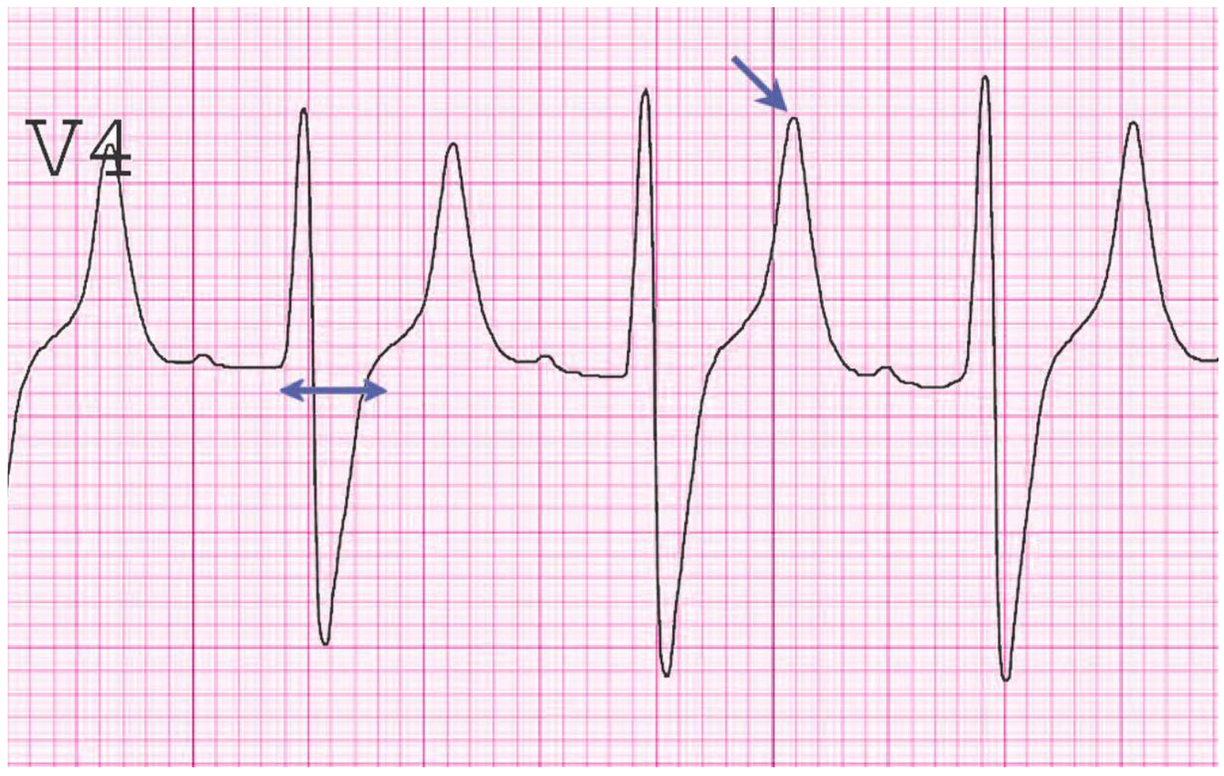


FIGURE 23.45B ■ Peaked T waves (arrow), widened QRS (double arrow), and subtle flattening of the P waves are seen in this patient with a serum K of 7.1.

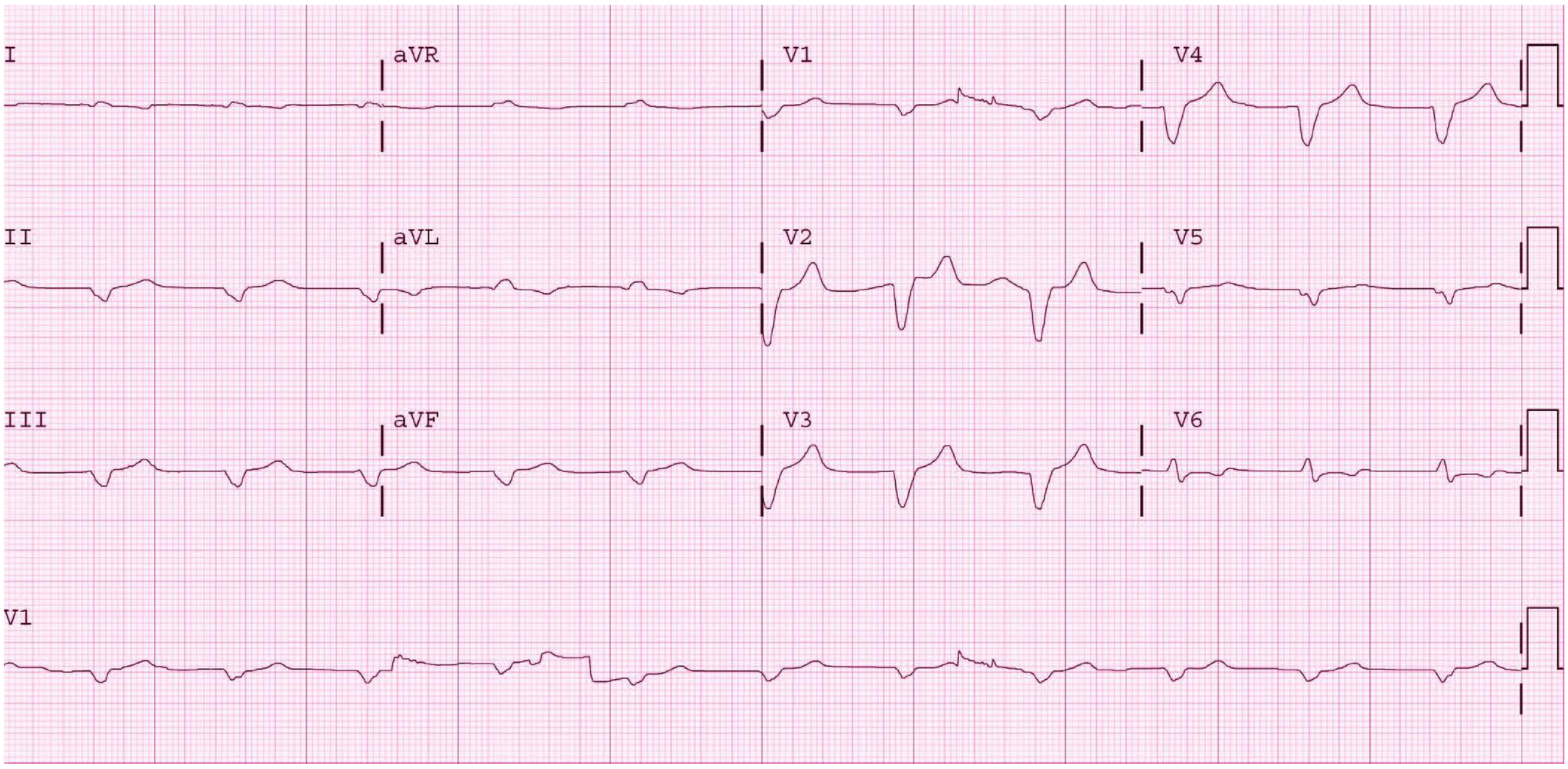


FIGURE 23.46A ■ **Severe Hyperkalemia (K 8.5).** (ECG contributor: Ben Heavrin, MD.)

Pearls

1. As the QRS complex widens, it appears more "blunted" than expected when compared with an LBBB.
2. Acute treatment for hyperkalemia includes insulin and glucose, sodium bicarbonate, and β-agonists in an attempt to drive potassium into the cell. Intravenous calcium may be used to stabilize the myocardium but has no effect on serum potassium levels. These are temporizing measures which must be followed by definitive treatment of the underlying problem, which may include the need for dialysis.

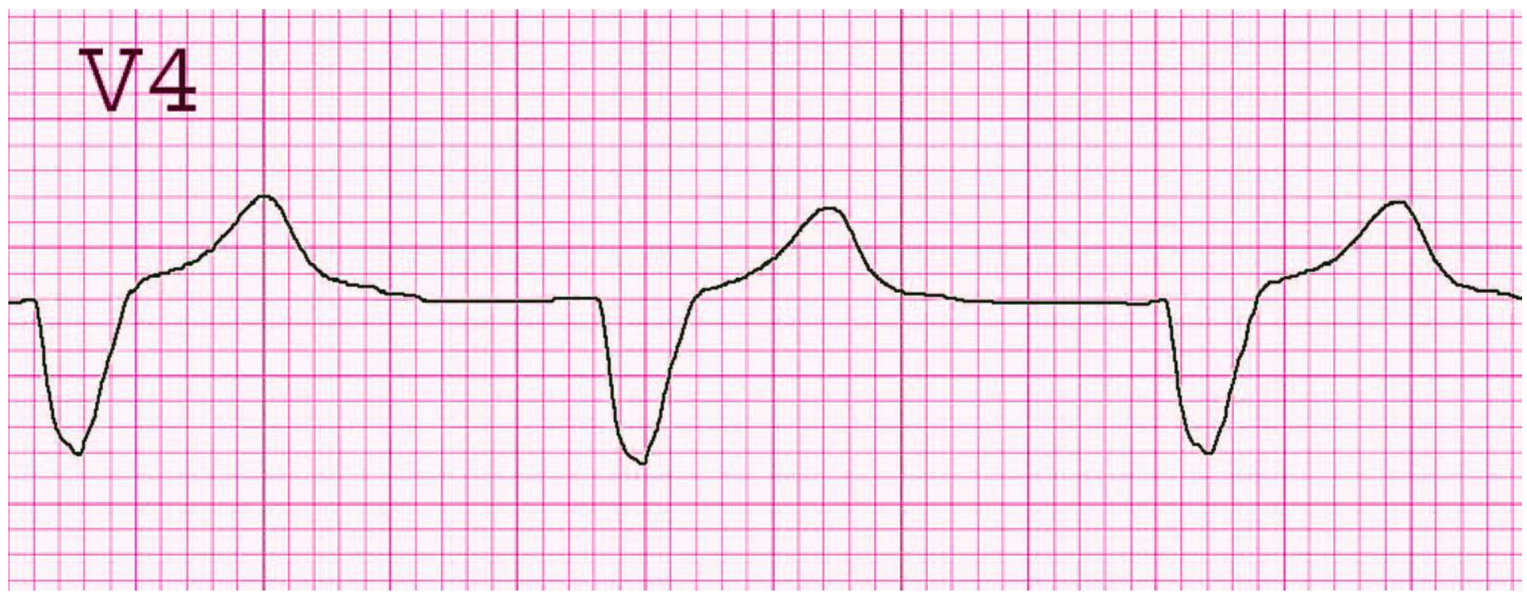


FIGURE 23.46B ■ Wide, blunted QRS with near sine-wave appearance. No P waves visible. Serum K was 8.5 in this patient. These abnormalities resolved with rapid treatment.

HYPOKALEMIA

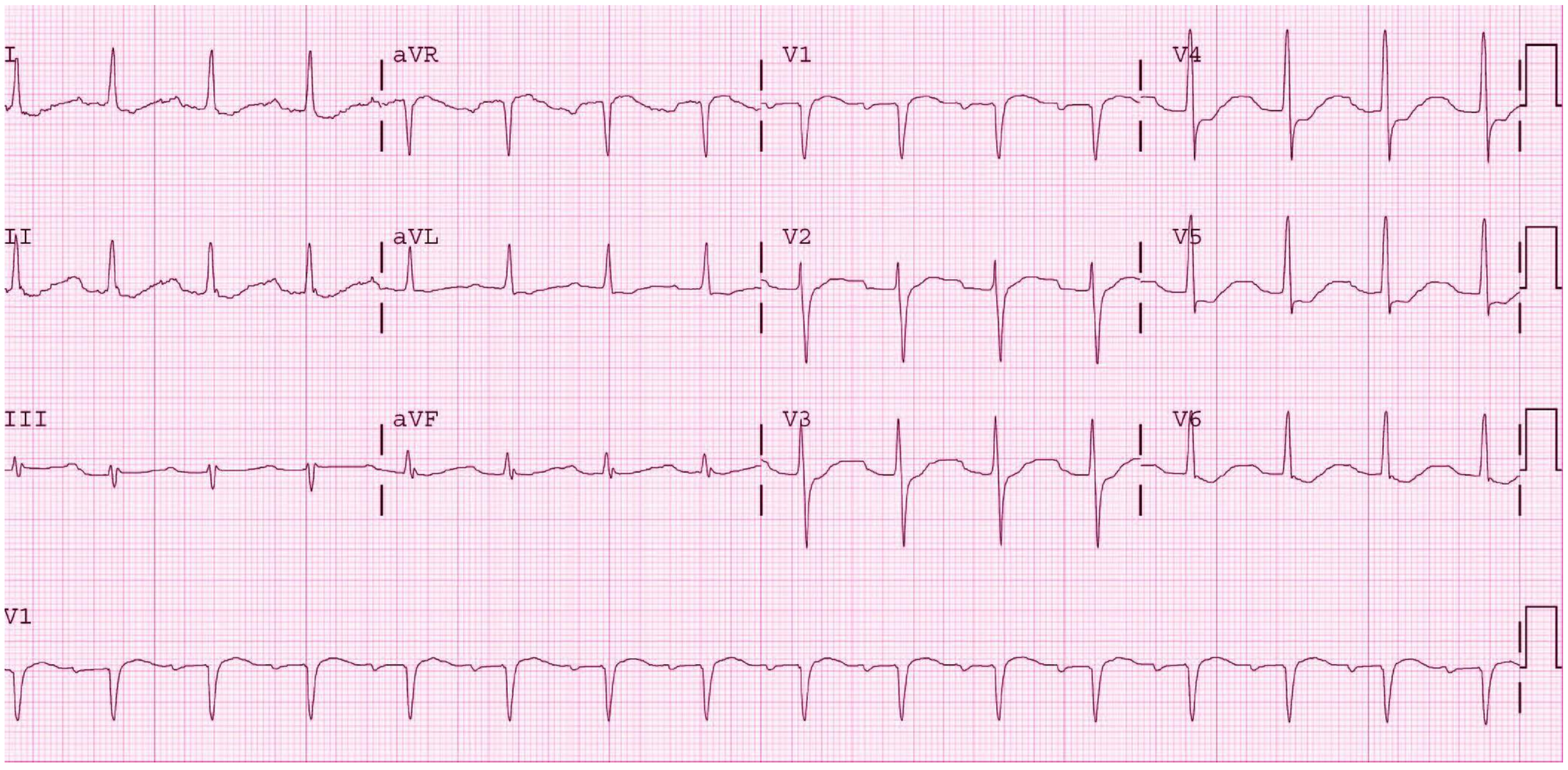


FIGURE 23.47A ■ **Hypokalemia.** (ECG contributor: R. Jason Thurman, MD.)

ECG Findings

- Flattened or inverted T waves
- Prominent U waves
- ST segment depression
- Conduction disturbances

Pearls

1. Hypokalemia can produce varied ECG changes associated with the repolarization phase of the cardiac cycle.
2. Unlike hyperkalemia, in hypokalemia there is no direct correlation with the potassium level and the severity of ECG changes. However, more ECG changes may become apparent as the potassium level falls.
3. Suspect hypomagnesemia if the ECG does not normalize after potassium replacement.

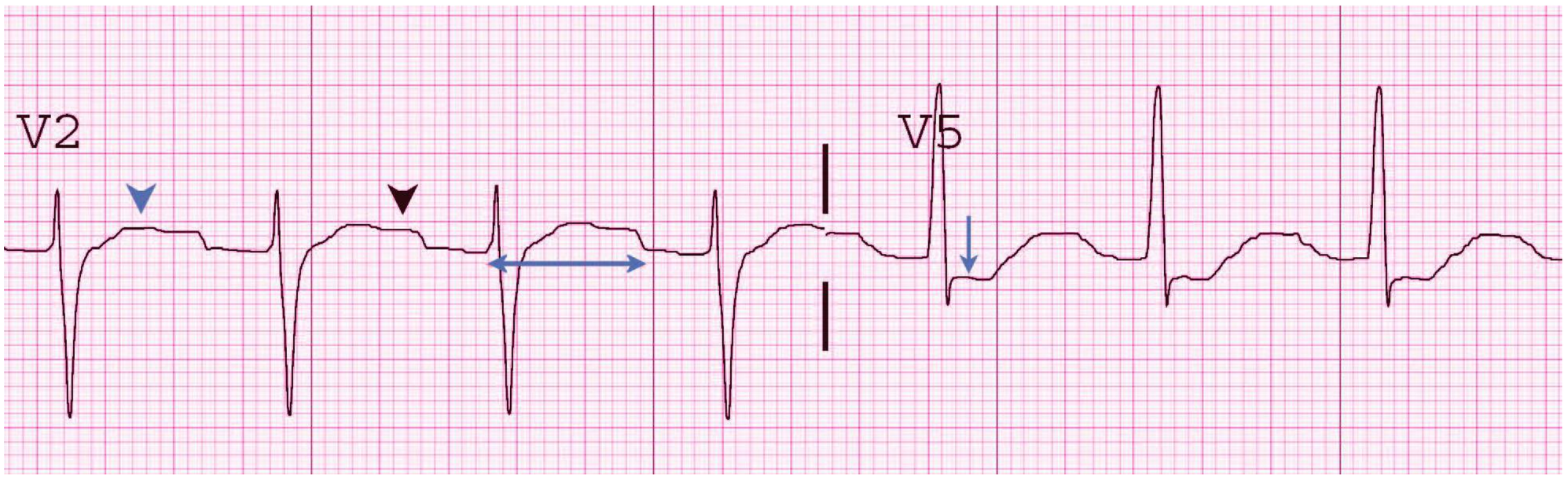


FIGURE 23.47B ■ This EKG demonstrates multiple findings consistent with hypokalemia: flattened T waves (blue arrowhead), U waves (black arrowhead), prolonged QT (QU) intervals (double arrow), and ST-segment depression (arrow). This patient's potassium level was 1.9.

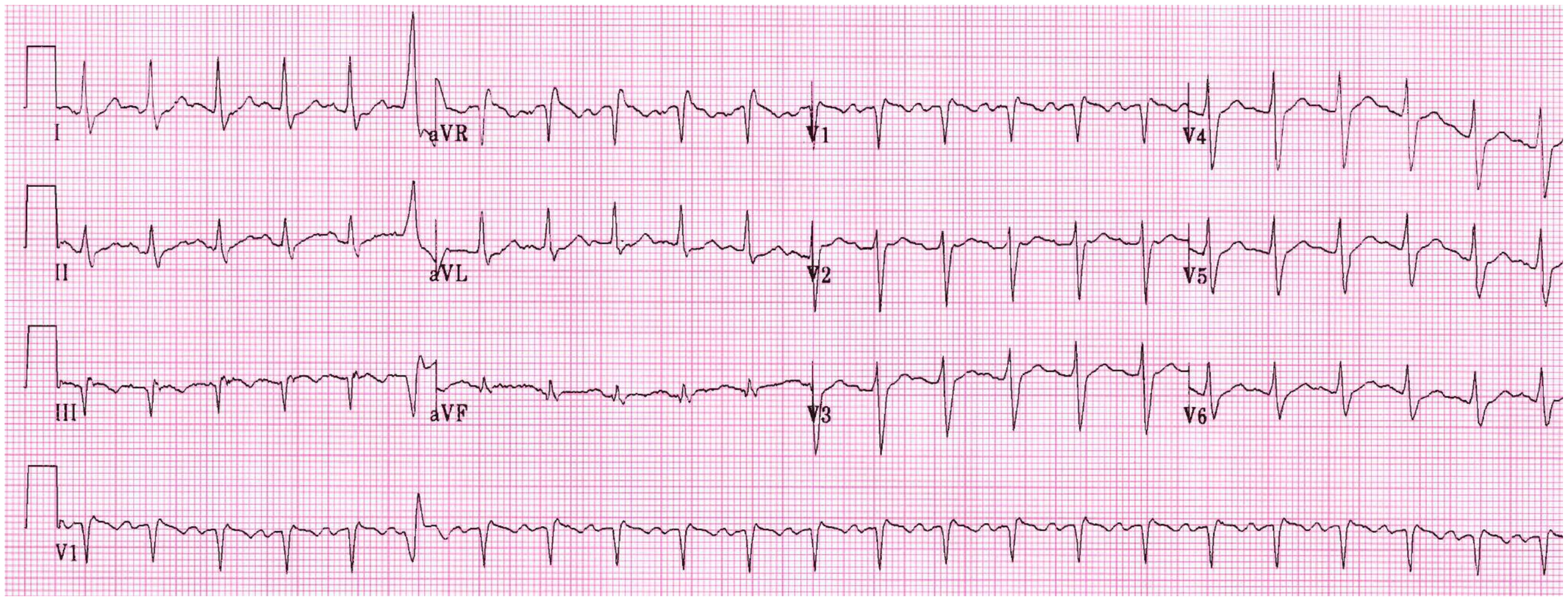


FIGURE 23.48A ■ **Sinus Tachycardia and S1Q3T3 Pattern in a Patient with Acute Pulmonary Embolism.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- Sinus tachycardia, nonspecific ST-T changes
- Precordial T-wave inversions
- Prominent S wave in lead I, Q wave in lead III, and inverted T wave in III (S1/Q3/T3)
- Incomplete or complete RBBB, P pulmonale (lead II)
- May see right axis deviation

Pearls

1. No EKG pattern is diagnostic for pulmonary embolism. Small-to-moderate emboli may not affect the EKG.
2. With large emboli, increased resistance to pulmonary arterial flow produces right ventricle overload and dilation.
3. Increased right atrial pressures may produce "P pulmonale" (tall P waves >2.5 mm in lead II) or atrial dysrhythmias.

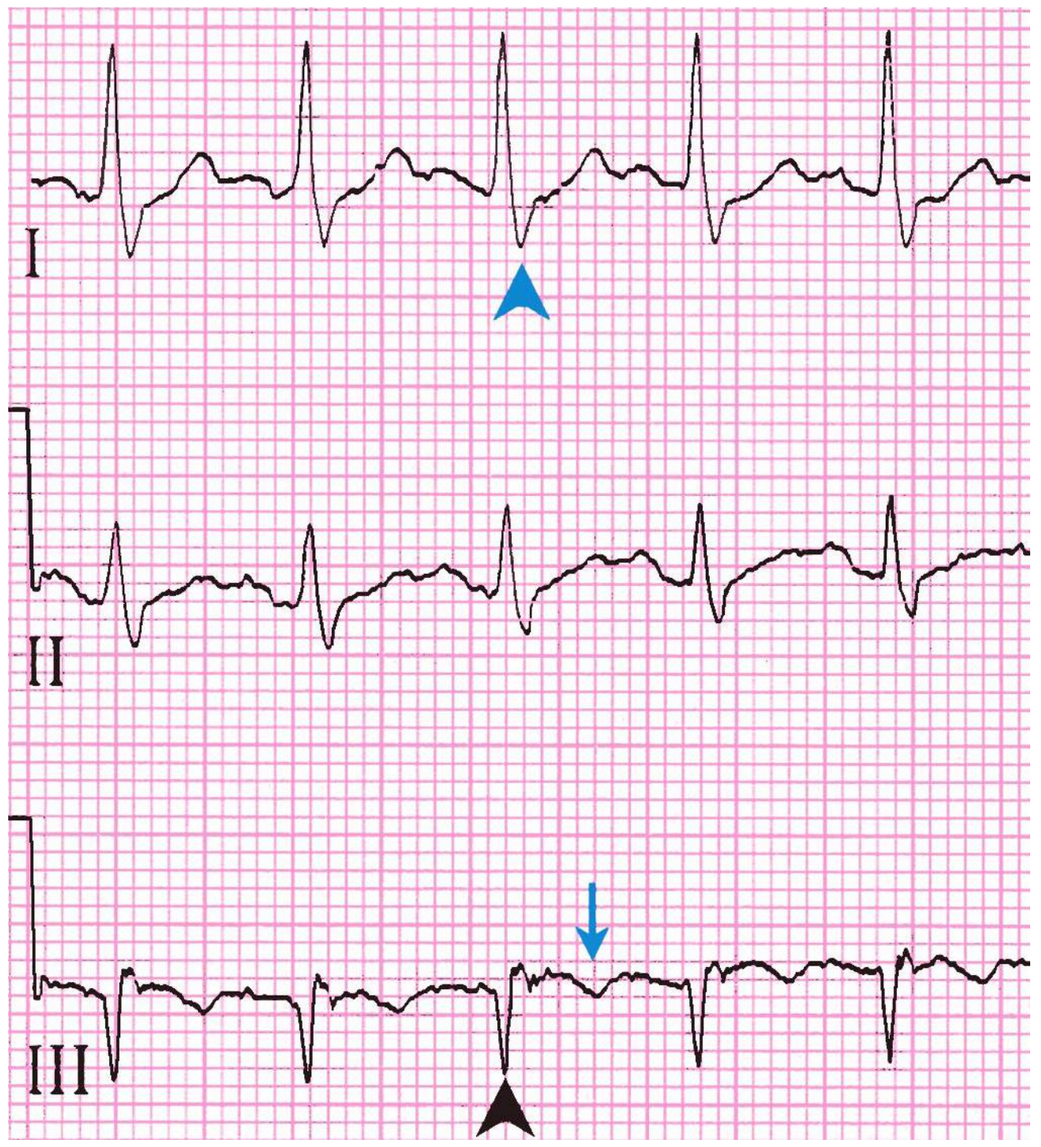


FIGURE 23.48B ■ S wave is apparent in lead I (blue arrowhead), Q wave in lead III (black arrowhead), and inverted T wave in lead III (blue arrow).

PERICARDIAL EFFUSION

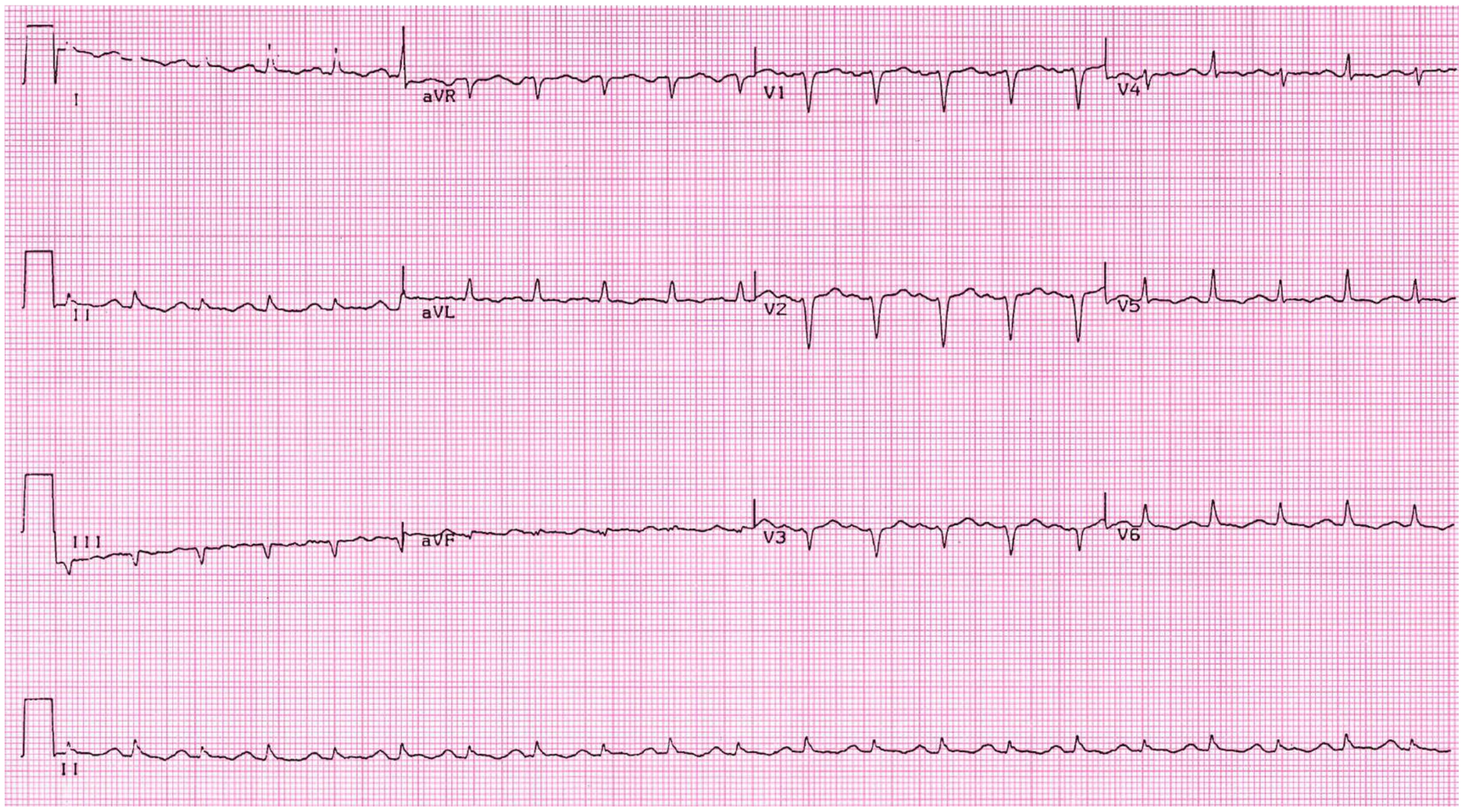


FIGURE 23.49A ■ **Pericardial Effusion with Electrical Alternans.** (ECG contributor: Kevin E. Zawacki, MD.)

ECG Findings

- Sinus tachycardia
- Low voltage of QRS complex (QRS averaging <5 mm height in limb leads, or <10 mm height in precordial leads)
- Electrical alternans (beat-to-beat change in electrical axis and/or amplitude of the QRS complex)

Pearls

1. A physiologically significant pericardial effusion compresses the heart, and affects the ability of the heart to fill properly. This typically results in a reflex sinus tachycardia to maintain cardiac output.
2. Pericardial effusion may be caused by pericarditis, malignancy, uremia, trauma, iatrogenic injury (CVL placement), aortic dissection with retrograde involvement of the pericardium, and free wall rupture after a myocardial infarction.
3. Initial treatment of physiologically significant pericardial effusion is with intravenous fluid bolus to increase preload. Pericardiocentesis should be reserved for hemodynamically threatening effusions due to a high associated morbidity. Surgical pericardial window may be necessary, especially in malignant effusions.
4. Electrical alternans is an uncommon finding. Pericardial effusion should be suspected in the setting of a sinus tachycardia and low voltage.

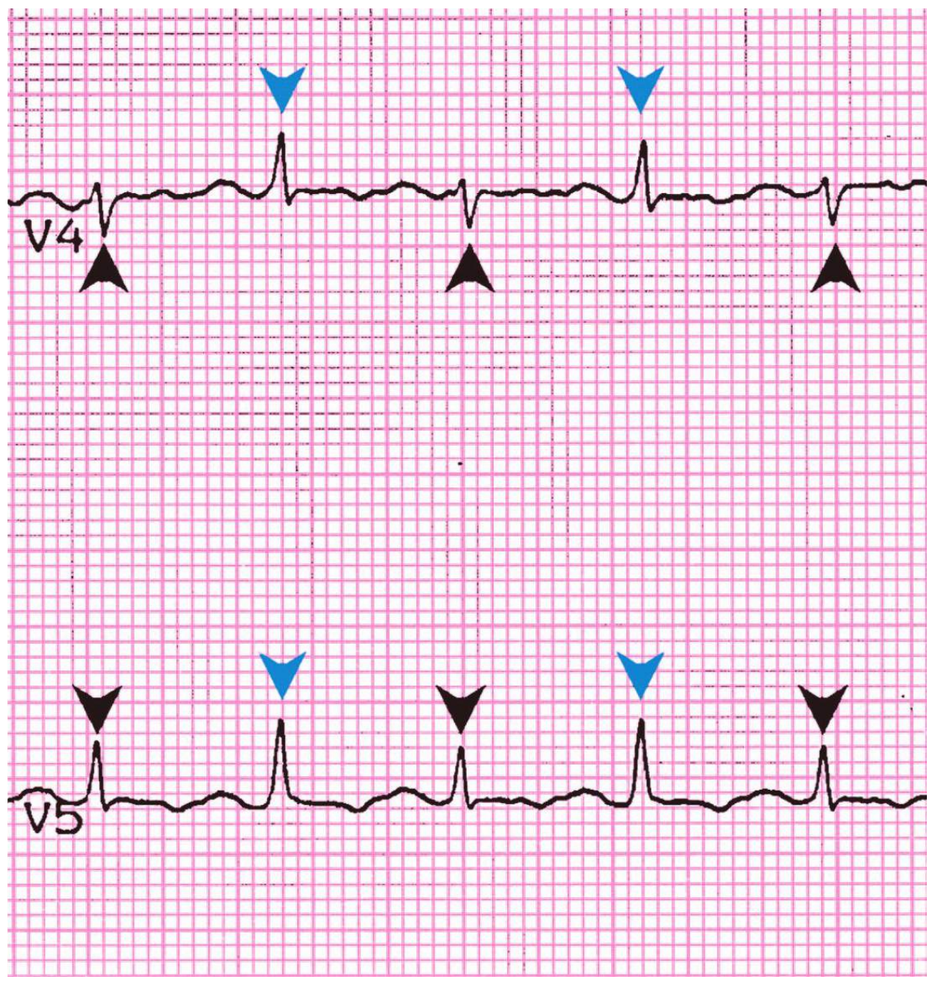


FIGURE 23.49B ■ Low voltage, sinus tachycardia, electrical alternans (arrowheads) demonstrate beat-to-beat alternating QRS electrical axis and/or amplitude. Electrical alternans is often best seen in the anterior precordial leads V_3 and V_4.

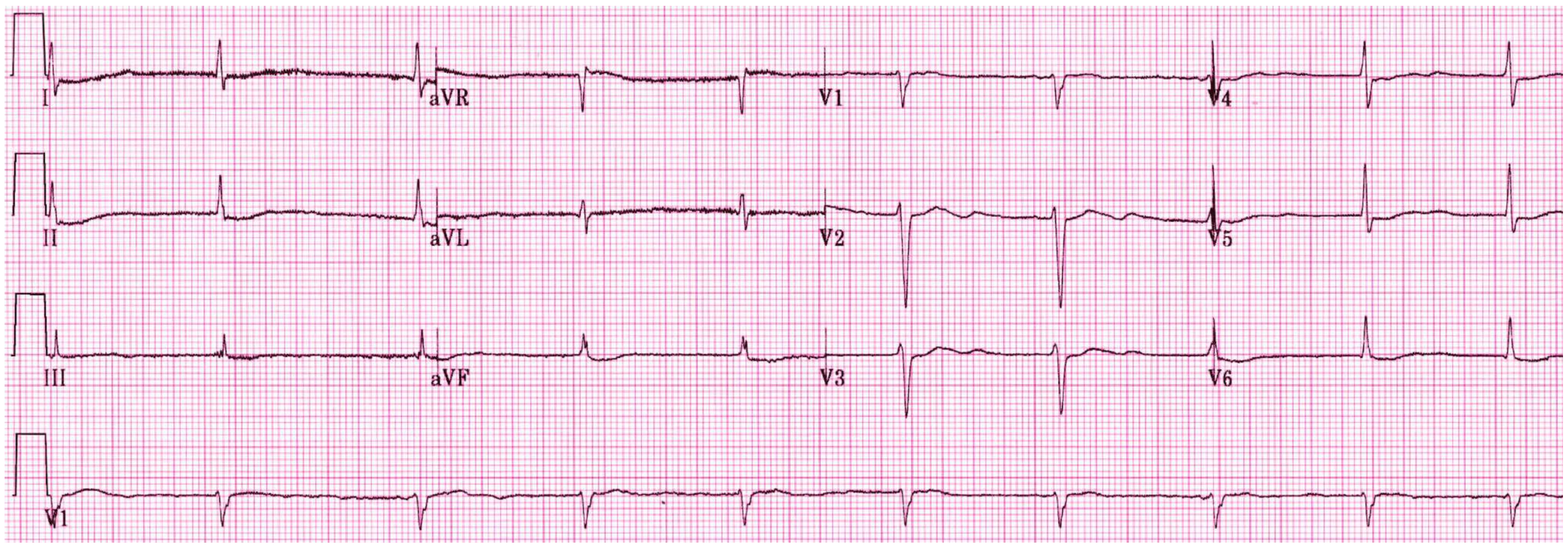


FIGURE 23.50A ■ **Digoxin Effect with Evidence of Toxicity.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- ST segment shortening and depression leading to a "scooped" appearance.
- QT interval shortening.
- PR interval prolongation.
- Decreased T-wave amplitude.
- Premature ventricular complexes are the most common dysrhythmia.
- Bradydysrhythmias, various heart blocks, especially with findings consistent with increased automaticity (atrial tachycardia with block, atrial fibrillation with slow ventricular response, accelerated junctional rhythms).
- Bidirectional ventricular tachycardia may rarely be seen (see Fig. 17.72).

Pearls

1. ECG changes associated with digoxin can be seen from therapeutic or toxic levels.
2. ST-segment changes may be exaggerated by myocardial disease or tachycardia.
3. An acute overdose of a digoxin is usually associated with hyperkalemia which may increase the height of the T wave.
4. Avoid calcium for treatment of hyperkalemia in the setting of digoxin toxicity as this may potentiate some adverse effects of digoxin.
5. A digoxin overdose can lead to almost any dysrhythmia, but it commonly blocks the transmission of impulses through the AV node leading to bradycardic rhythms and accelerated escape rhythms.

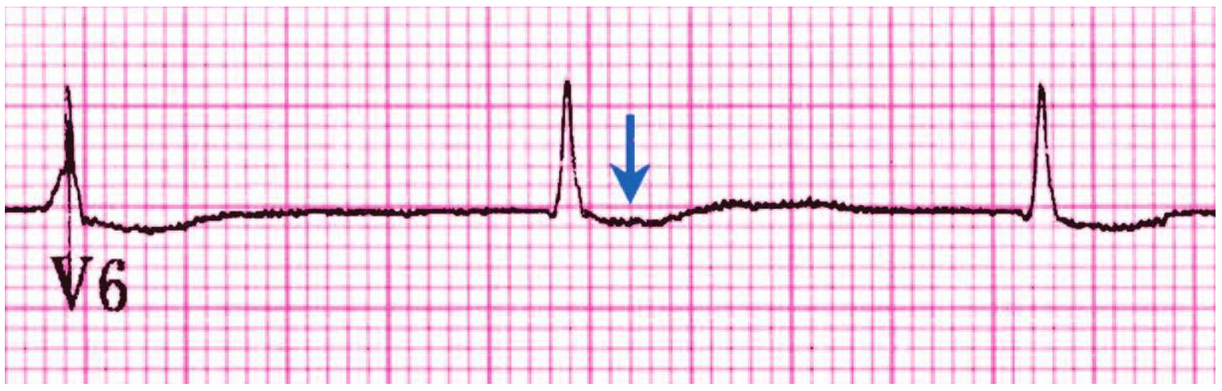


FIGURE 23.50B ■ The "sagging" appearance of the ST segment (arrow) is characteristic of digoxin therapy, and is not a sign of toxicity. However, this patient also has a sign of chronic digoxin toxicity. Atrial fibrillation is present, but the R-to-R interval has become regular. Digoxin toxicity has produced a total AV block but has also excited the AV node, producing a relatively accelerated junctional escape rate.

TRICYCLIC ANTIDEPRESSANT EFFECT

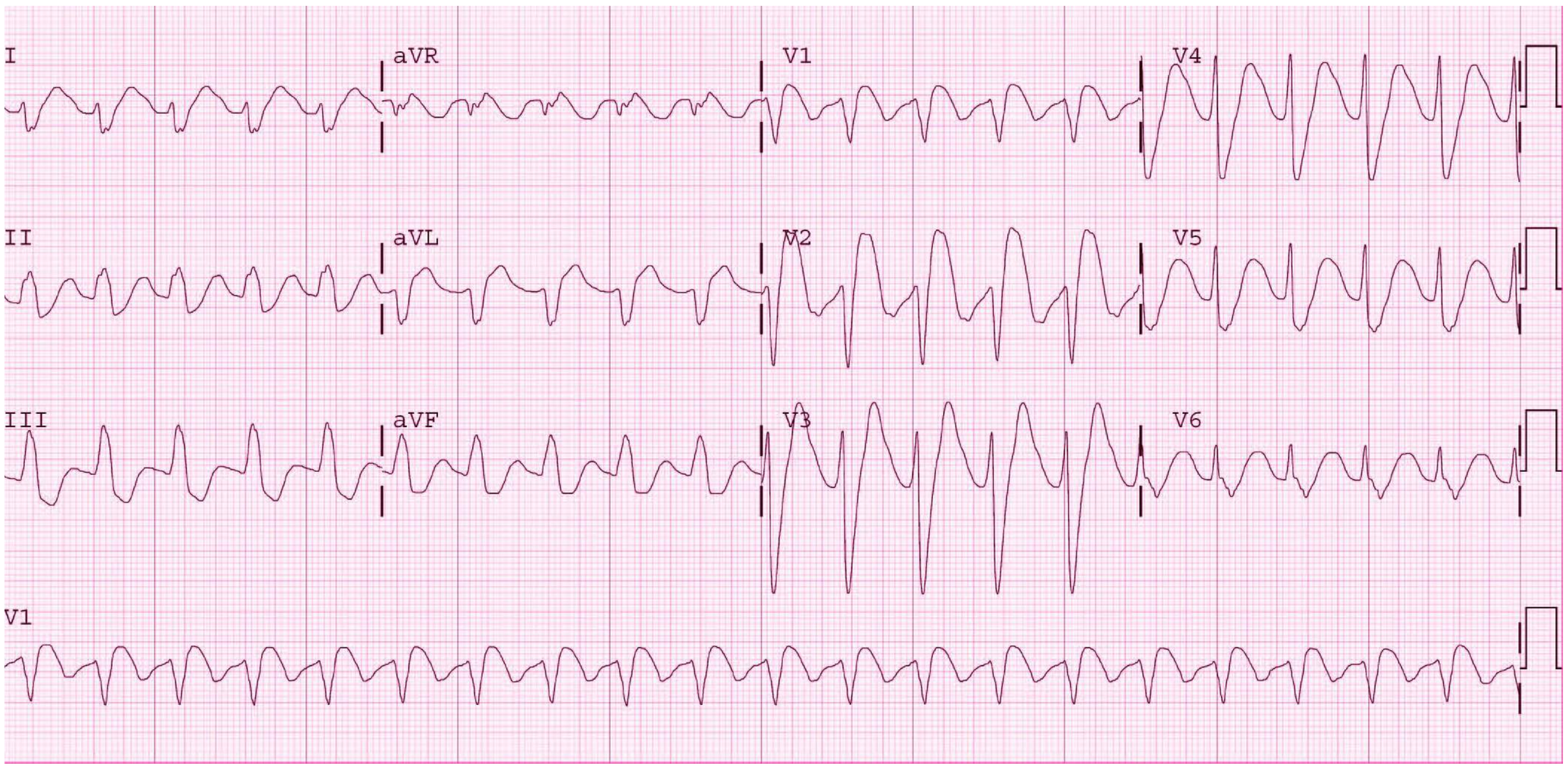


FIGURE 23.51A ■ **Tricyclic Antidepressant Toxicity.** (ECG contributor: Saralyn R. Williams, MD.)

ECG Findings

- Tachycardia
- QRS complex widening
- QT prolongation
- Prominent terminal R wave in aVR or V_1
- Prominent S in Lead I

Pearls

1. Tricyclic antidepressants (TCAs) produce their effects by several mechanisms. Anticholinergic effects may induce tachycardia and sodium channel blockage may lead to QRS widening.
2. The QRS widening seen in a TCA overdose has a nonspecific pattern and is typically unlike any BBB morphology.
3. ECG effects are rate-dependent and become more pronounced with tachycardia and acidosis.

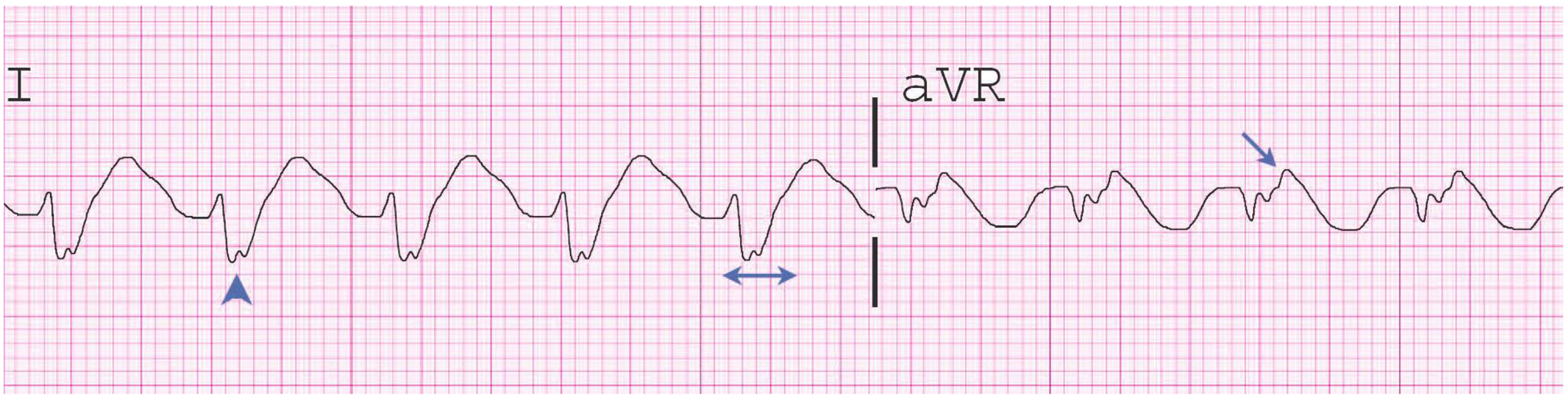


FIGURE 23.51B ■ Prominent S wave in lead I (arrowhead) with prominent terminal R wave in aVR (arrow). The QRS complex is wide (double arrow), the QT interval is prolonged, and the patient is tachycardic.

LIMB LEAD REVERSAL

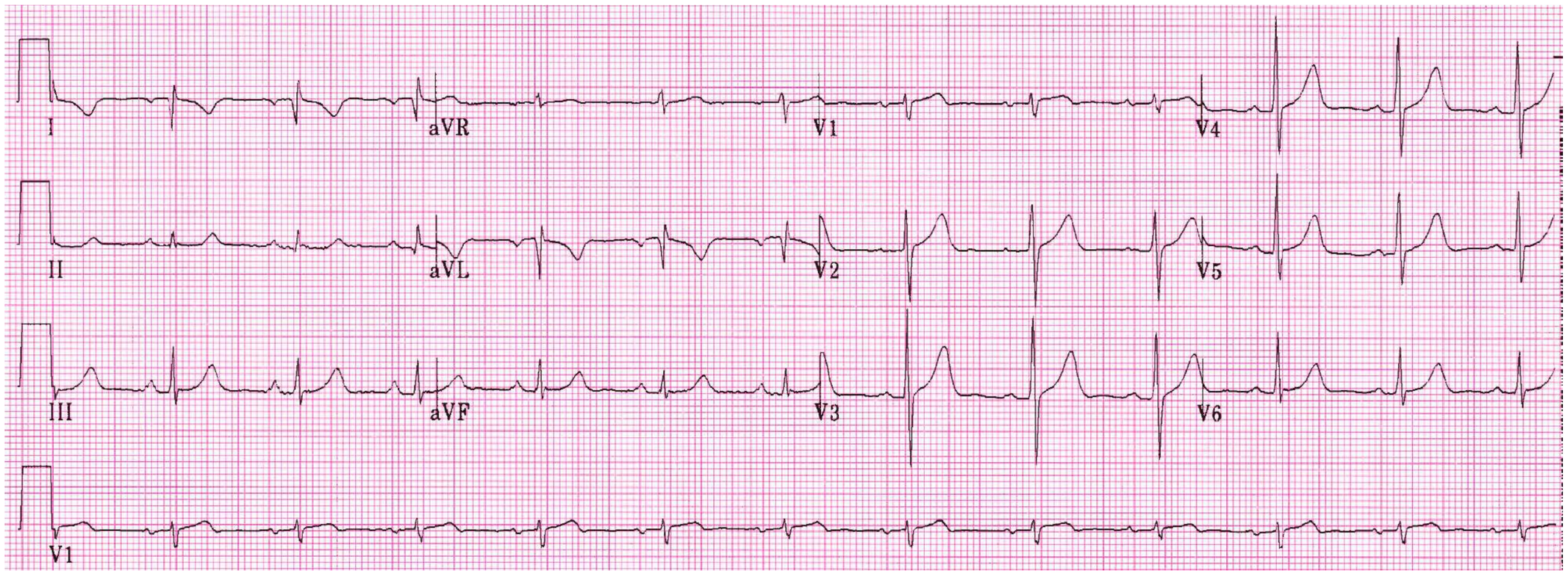


FIGURE 23.52A ■ **Limb Lead Reversal.** (ECG contributor: Michael L. Juliano, MD.)

ECG Findings (dependent on which leads are reversed)

- Reversal of the left arm (LA) and right arm (RA) most common
- P, QRS, and T predominantly downgoing in lead I
- P, QRS, T upgoing in lead aVR
- Precordial leads unaffected
- Reversal of the leg leads (left leg [LL] and right leg [RL])
- Does not commonly produce EKG changes because RL is used as a grounding electrode
- Reversal of LA-LL
- Transposition of leads I and II and leads aVF and aVL with reversal of lead III
- Reversal of RA-RL
- Transposition of aVR and aVL and inversion of lead II
- Incorrect precordial lead placement
- Isolated reversal of the usual R-wave progression from V_1 to V_6

Pearls

1. If the ECG seems to have an unusual axis or appearance, especially when compared with a prior ECG on the same patient, consider a lead misplacement and repeat the tracing, confirming correct lead positions.
2. A "reversed" lead I with normal-appearing V leads strongly suggests accidental limb lead reversal as opposed to dextrocardia. Dextrocardia features a "reversed" lead I, while QRS deflections in V_4 to V_6 appear small and downgoing.



FIGURE 23.52B ■ The P wave, QRS, and T wave are inverted in lead I in this EKG. Normal-appearing V leads in the 12-lead ECG above suggest limb lead reversal rather than dextrocardia. The arm leads were indeed reversed, and correction produced a normal-appearing tracing.

LOW VOLTAGE

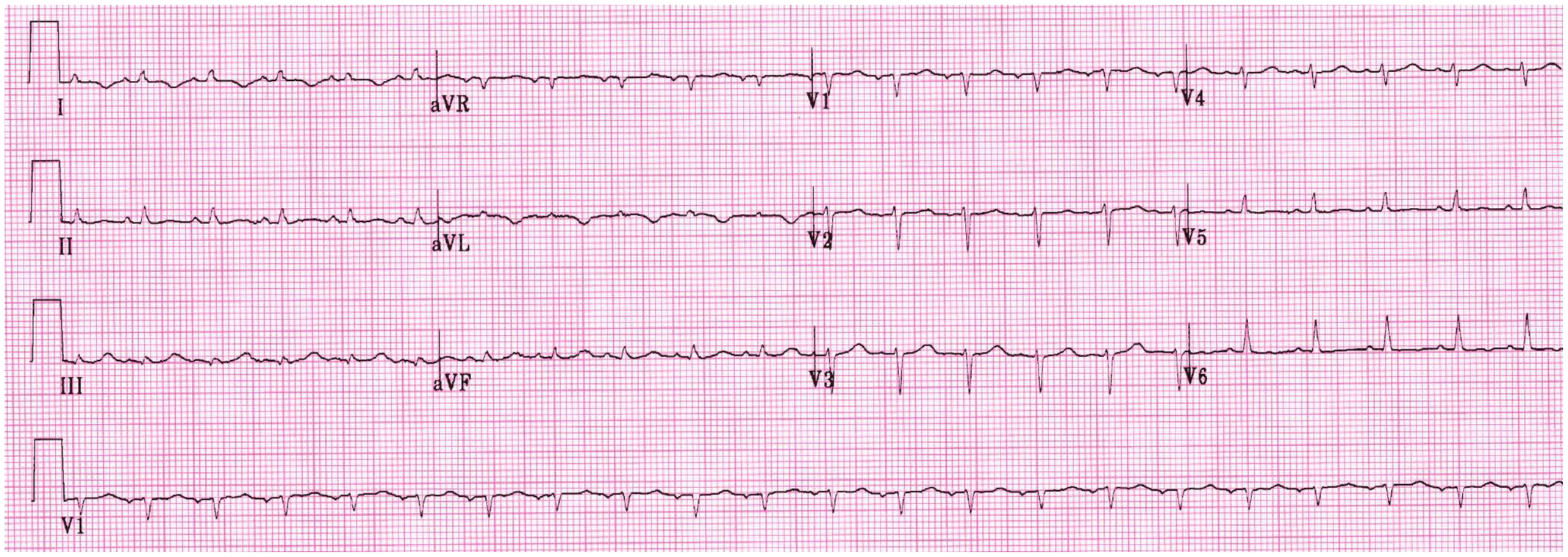


FIGURE 23.53A ■ **Low-Voltage EKG.** (ECG contributor: James V. Ritchie, MD.)

ECG Findings

- QRS amplitude of less than or equal to 5 mV in all limb leads or a sum of all limb lead QRS amplitude less than or equal to 30 mV and/or QRS amplitude less than or equal to 10 mV in all precordial leads

Pearls

1. Differential diagnosis includes normal variant, low standardization of the ECG machine, pericardial or pleural effusion, obesity/anasarca, COPD/emphysema, cardiac infiltrate (tumor, amyloid), myocardial infarction, myocarditis, cardiomyopathy, adrenal insufficiency, or hypothyroidism.
2. Always check the calibration markings at the left of the ECG to check for low standardization of the ECG machine as an etiology for the observed tracing.

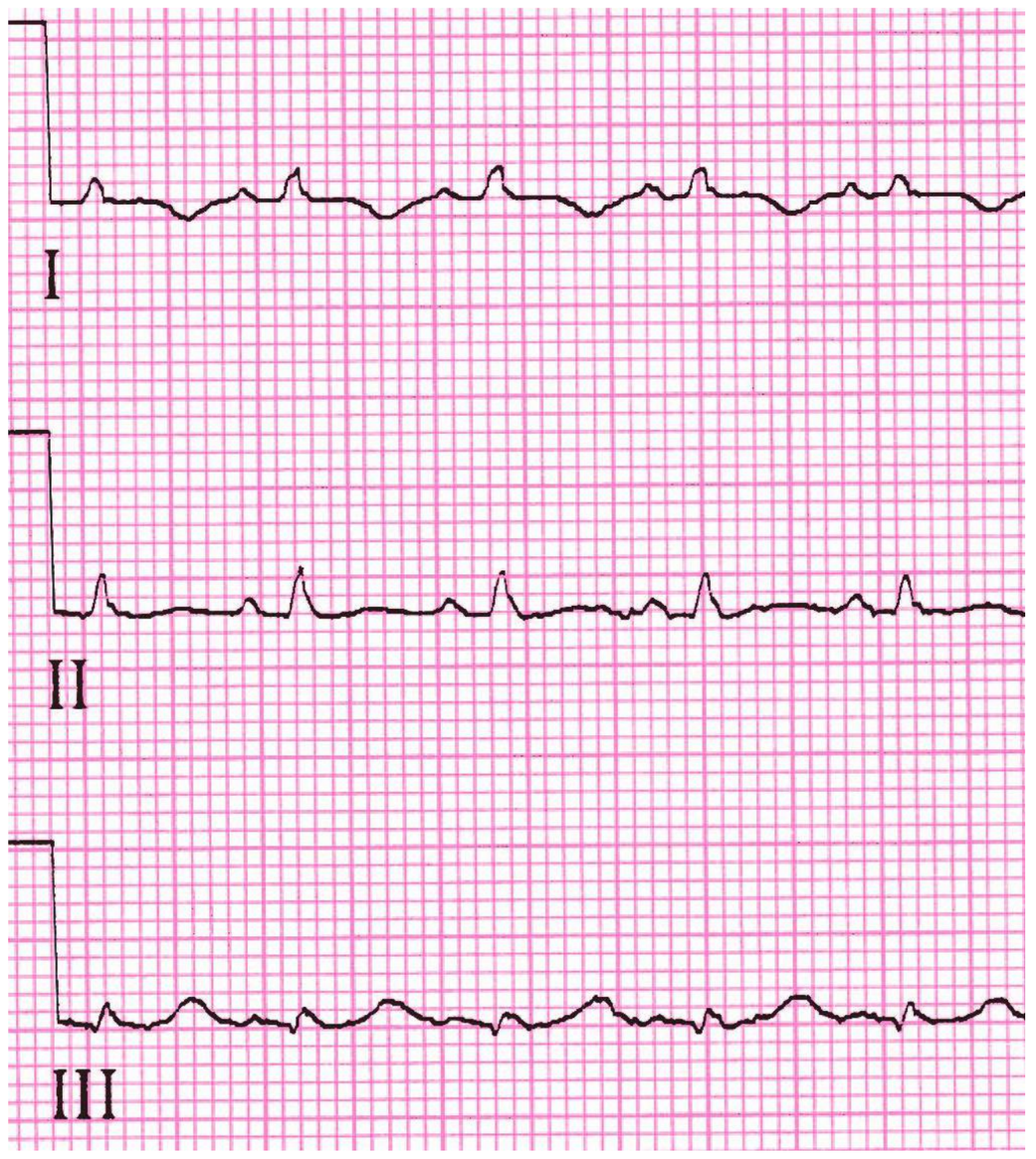


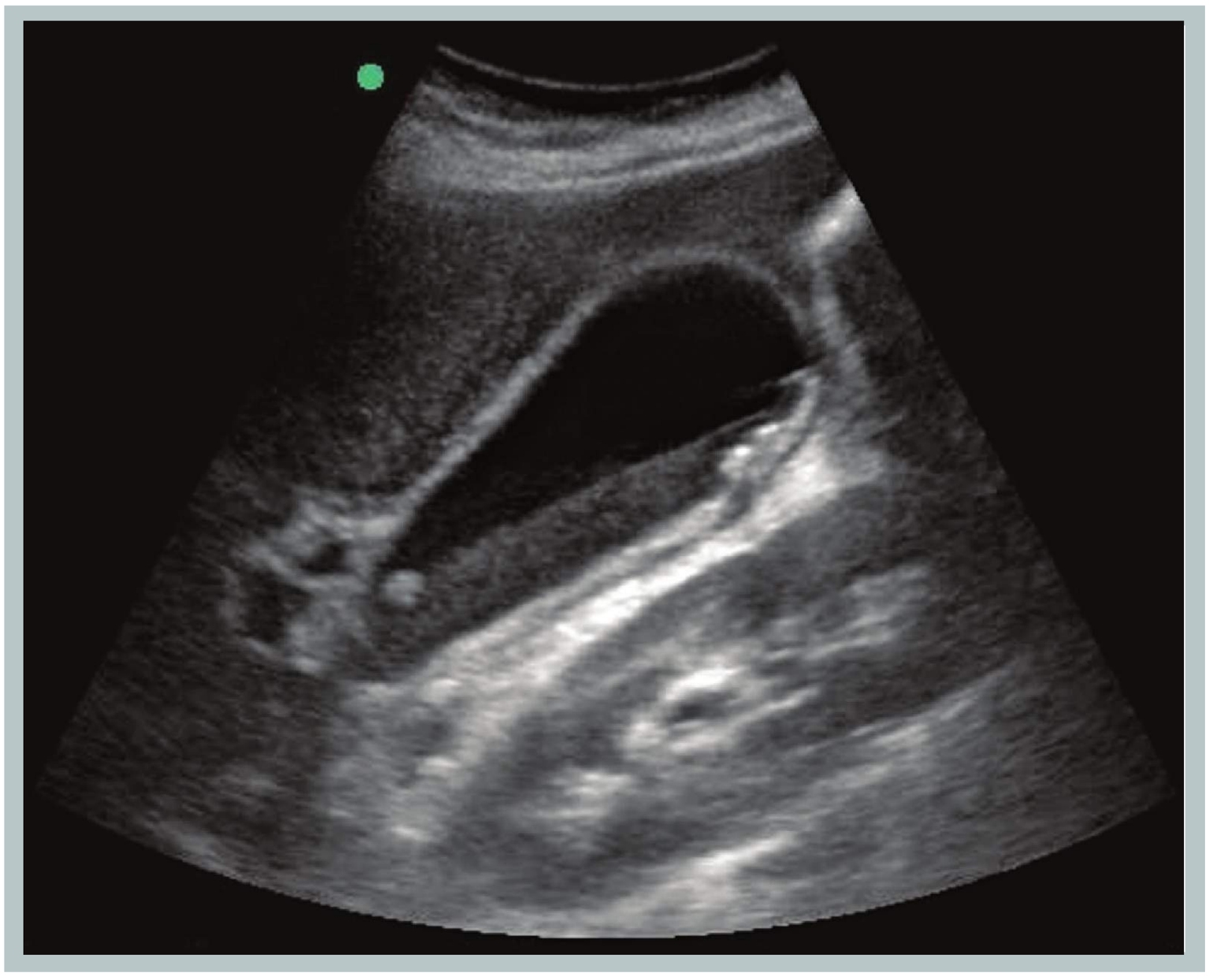
FIGURE 23.53B ■ QRS height is less than 5 mm in limb leads in this normally calibrated tracing.

Chapter 24

EMERGENCY ULTRASOUND

Jeremy S. Boyd
Jordan D. Rupp
Robinson M. Ferre

Scan QR code or go to www.ematlas.com for video related to chapter



Acute Cholecystitis. Longitudinal image of a gallbladder demonstrating gallstone, biliary sludging, and pericholecystic fluid. (Ultrasound contributor: Jeremy S. Boyd, MD.)

EMERGENCY ULTRASOUND: INTRODUCTION

Emergency ultrasound (EUS) is the use of point-of-care ultrasound by clinicians in the emergency department (ED) to answer focused clinical questions and/or assist in performing invasive procedures. It is not an extension of the physical exam by means of advanced technology, but rather uses ultrasound technology to sonographically assess patients for the presence or absence of pathologic conditions that commonly present to the ED. The use and scope of EUS has grown and evolved since the FAST exam was first introduced as part of the evaluation of the trauma patient. For more than a decade, EUS has been a required component of Emergency Medicine Residency training with additional training offered as part of a dedicated EUS fellowship programs. These dedicated EUS fellowship training programs have surged in recent years to meet the demand, with over 90 such programs currently operating.

EUS can be used by clinicians across the spectrum of diseases that present to the ED. Properly trained emergency clinicians use ultrasound to answer focused questions regarding the patient's presenting complaint or condition. Rather than a comprehensive approach, the EUS examination is goal-directed and focused, often seeking to answer dichotomous, "yes/no," questions such as "Is there fluid within the peritoneum?"

This text is not intended to provide an in-depth description of the limitations, efficacy, sensitivity, or appropriateness of EUS. Every application described herein is intended as a rapid visual review for those trained in basic EUS applications. Applicable protocols are based on the imaging guidelines of the American College of Emergency Physicians and American Institute of Ultrasound in Medicine (AIUM), as well as the authors' collective experience. Basic information—transducer recommendations, scanning protocols, anatomic schematics—is presented within each application to represent both image acquisition and normal and pathological findings.

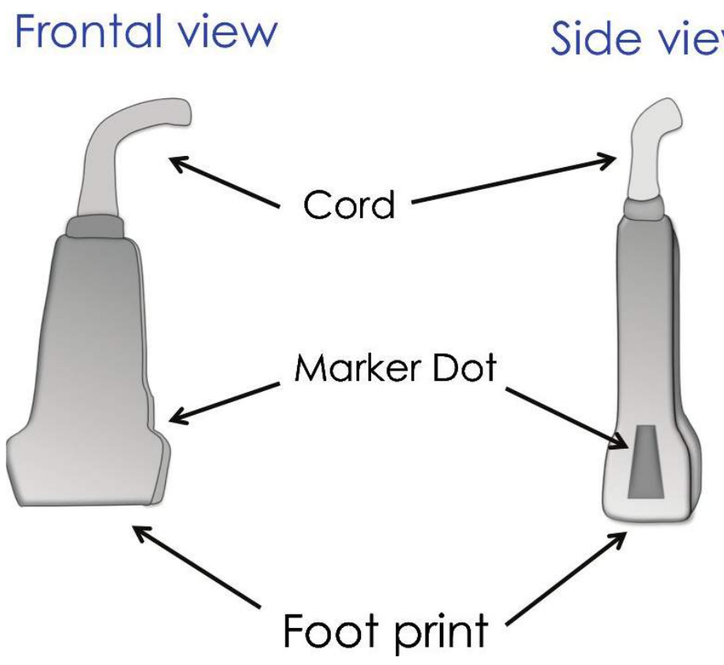


FIGURE 24.1 ■ Probe anatomy demonstrating location of transducer indicator/probe marker/marker dot and foot print. (Illustration courtesy of Robinson M. Ferre, MD.)

Key Terms

Sonographic windows—Anatomical locations on the body where an ultrasound probe is placed in order to view internal organs.

Transducer indicator/probe marker/marker dot—Usually a bump or ridge on the ultrasound transducer that corresponds to a symbol on the ultrasound screen. Used to indicate which side of the image corresponds with the transducer's edge (Fig. 24.1).

Foot print—The part of the ultrasound probe that contacts the patient's skin (Fig. 24.1).

Hyperechoic/echogenic—Used to describe objects on the ultrasound screen that are bright and therefore reflect sound waves.

Hypoechoic/anechoic—Used to describe objects on the ultrasound screen that are dark and therefore transmit sound waves.

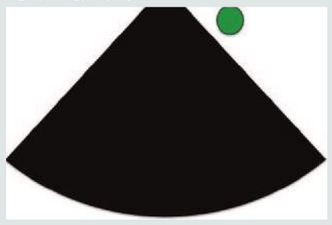
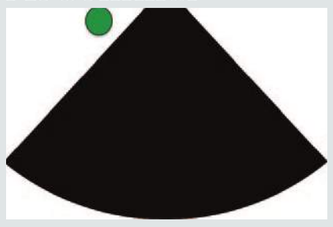
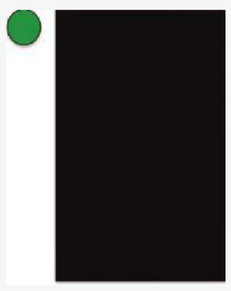
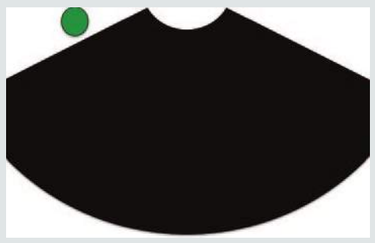
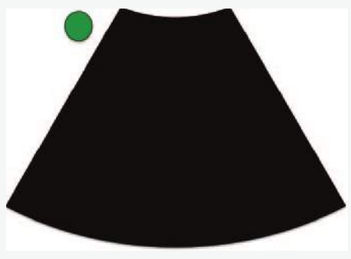
Echotexture—The characteristic appearance of specific organs when viewed using ultrasound. The liver has a characteristic echotexture.

Transducer movements—When performing sonography, specific terms are used to describe characteristic movements of the transducer used to obtain the image desired. Some examples of this include dragging, rocking, fanning, and rotating (Fig. 24.2).

Transducers

Emergency sonography is performed using a wide variety of transducers. Lower frequency transducers are used to visualize structures deep within the body, while higher frequency transducers allow better image resolution of superficial anatomic structures. The size and shape of transducers are also configured for imaging specific anatomic locations. Table 24.1 lists the basic transducer types frequently used in EUS.

TABLE 24.1 ■ TRANSDUCER, APPEARANCE, AND APPLICATIONS

Transducer	Image Appearance	Applications*
Phased array  FIGURE 24.3A • Small footprint for imaging between ribs • Lower frequency (1-5 MHz) = better penetration, less resolution • Wide far-field view (wedge-shaped window)	Cardiac  FIGURE 24.3B Abdominal  FIGURE 24.3C	(+) Cardiac, abdominal, thoracic (−) Soft tissue, pregnancy, vascular
Linear array  FIGURE 24.4A • Flat footprints in various sizes • High frequency (5-13 MHz) = better resolution of superficial structures • Rectangular window (at right)	 FIGURE 24.4B	(+) Procedural, vascular, soft tissue/MSK, superficial, thoracic (pleural), ocular (−) Abdominal, cardiac, pregnancy
Endocavitary probe  FIGURE 24.5A • Curved footprint probe for examining internal structures • High frequency (5-8 MHz) = better resolution of nearby structures • Designed for intracavitary use, primarily transvaginal ultrasound • Wide wedge-shaped window (at right)	 FIGURE 24.5B	(+) Transvaginal pregnancy, intraoral (−) Any external exam
Curvilinear/convex array transducer  FIGURE 24.6A • Wide, curved footprint with large surface area decreases discomfort with applied pressure to the abdomen • Lower frequency 2-5 MHz (but with better detail than phased array probe) • Wide wedge-shaped window (at right)	 FIGURE 24.6B	(+) Aorta, abdominal, transabdominal pregnancy (−) Cardiac

*(+) = best use; (-) = not ideal

(Photo contributor: Lawrence B. Stack MD, Illustrations courtesy of Robinson M. Ferre, MD.)

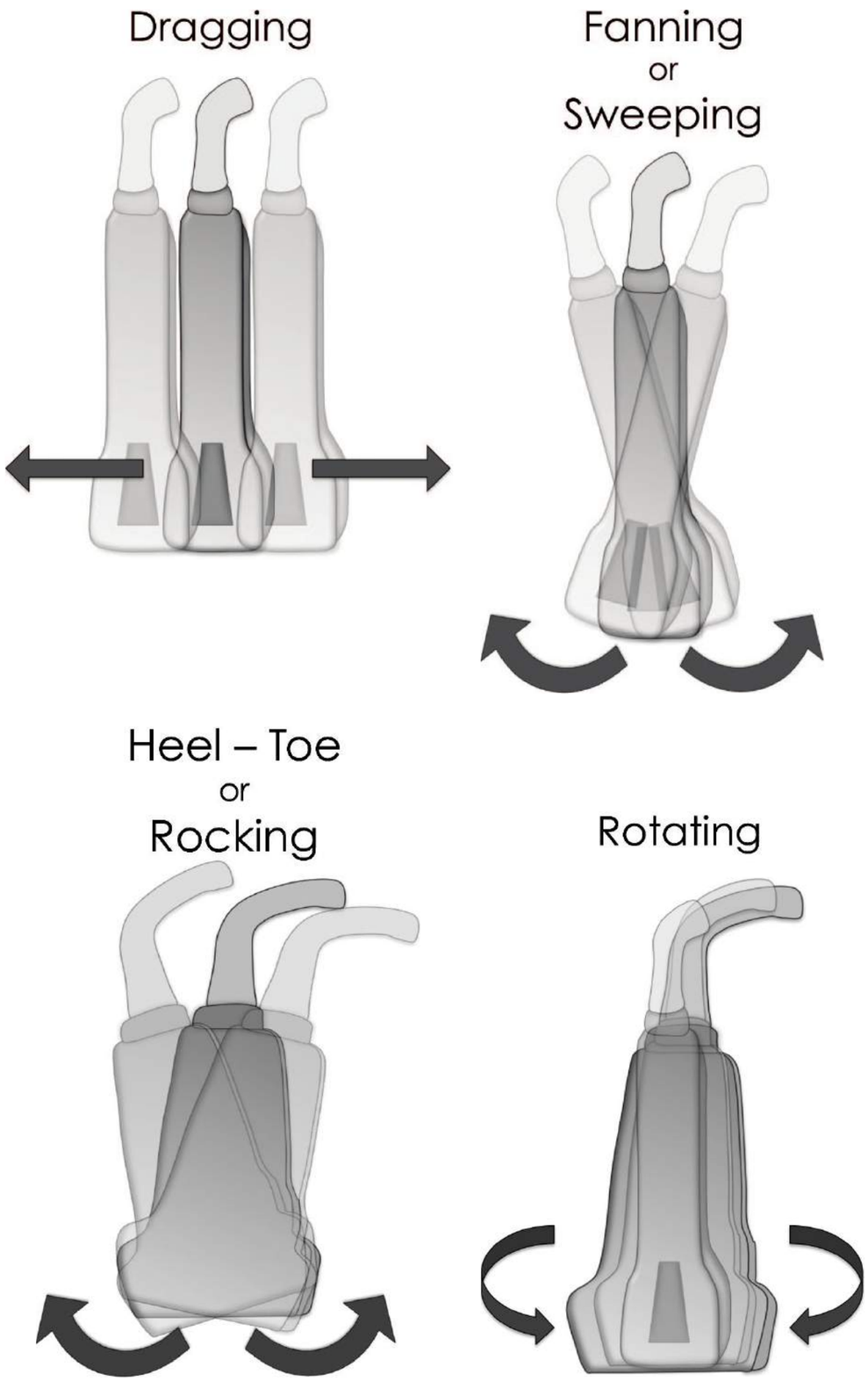


FIGURE 24.2 ■ **Transducer Movements**. (Illustration courtesy of Robinson M. Ferre, MD.)

Clinical Summary

The Extended Focused Assessment with Sonography in Trauma (E-FAST) is a protocolized series of sonographic views that attempt to identify the presence or absence of fluid or air in anatomic potential spaces such as the pericardium, the peritoneum, and the thorax. The goal of this thoracoabdominal survey is to identify or exclude immediate life threats in the trauma or critically ill patient. Though initially intended for the evaluation of the trauma patient, the E-FAST examination and its component views are also extremely valuable in the evaluation of several emergent complaints and clinical conditions including the patient with undifferentiated hypotension.

Indications

- Trauma (blunt and penetrating)
- Unexplained hypotension (traumatic and nontraumatic)
- Ectopic pregnancy (to evaluate for rupture)

The FAST examination uses four primary sonographic views to evaluate the patient. It is recommended that all four views are evaluated for a complete exam, but isolated views may be obtained when indicated. The extended component of the FAST exam (termed E-FAST) incorporates imaging of both the anterior and lateral hemithoraces to identify the presence or absence of a pneumothorax or hemothorax (Fig. 24.7). It is important to note that these are not static "single" views, but a series of images obtained in each plane as the transducer is moved or "fanned" through the area of interest.

Views for the E-FAST Examination

1. Pericardial (usually a subcostal/subxiphoid view—alternatively, one may obtain a parasternal long view of the heart if a subxiphoid view is unobtainable)
2. Right upper quadrant (RUQ; pouch of Morison)
3. Left upper quadrant (LUQ) (perisplenic view)
4. Suprapubic (pelvic view)
5. Right thorax
6. Left thorax

Patient Positioning

The E-FAST exam should be done with the patient in the supine position.

Recommended Transducers for the E-FAST Examination

- Convex array (abdominal, cardiac, and lung imaging)
- Phased array (abdominal, cardiac, and lung imaging)
- Linear array (lung only)

The E-FAST examination is a thoracoabdominal examination. Ideally, this is done using a single transducer that can image all three of these areas, but may result in some compromise of image quality and require the use of different probes for different components of the examination.

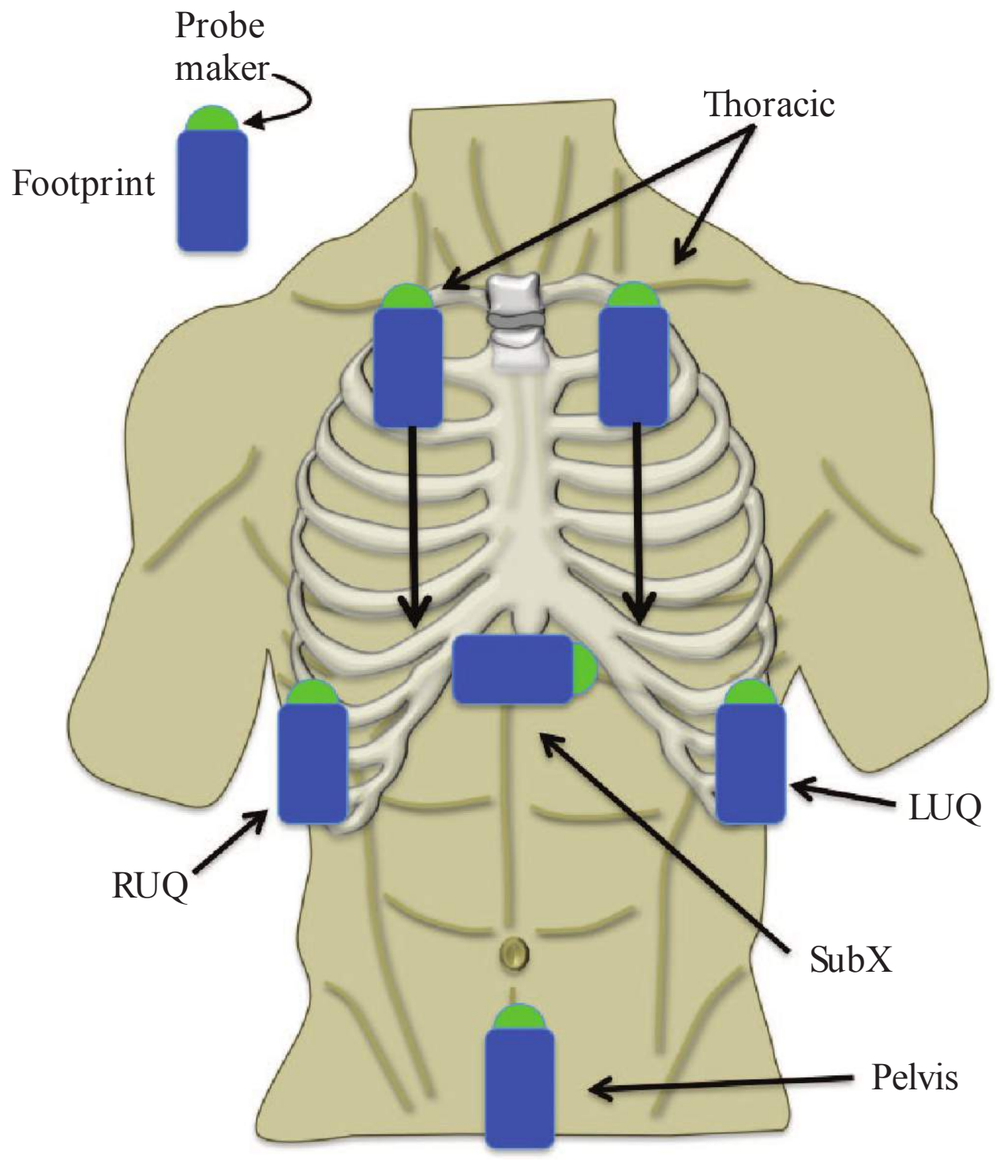


FIGURE 24.7 ■ **E-FAST Trauma Series.** Ultrasound transducer and probe marker positions for evaluation of pericardial fluid, hemoperitoneum, or pneumothorax. (Illustration courtesy of Robinson M. Ferre, MD.)

PERICARDIAL (SUBXIPHOID VIEW)

Technique

- Direct the transducer indicator to the patient's right (in abdominal or general preset). Note that the E-FAST exam is traditionally performed with abdominal preset settings, but the subxiphoid view is often performed with cardiac preset settings as part of a full focused cardiac ultrasound exam. When this is the case, the marker dot is typically on the opposite side of the screen and the indicator is oriented to the patient's left.
- With the transducer indicator pointing to the patient's right, the transducer is directed under the xiphoid process toward the left shoulder in a horizontal plane (Fig. 24.8).
- Pivot, sweep, and tilt the transducer as necessary to view all four cardiac chambers and pericardium.
- Identify the liver (if in view), heart, four cardiac chambers, and surrounding pericardium (Fig. 24.9).



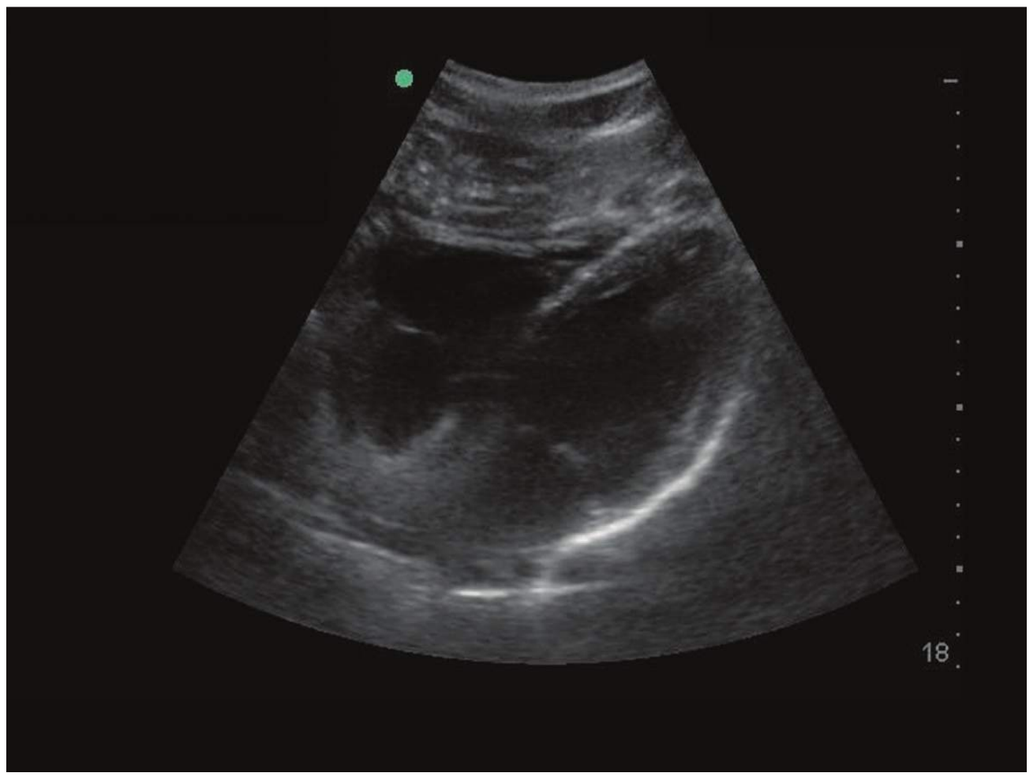
FIGURE 24.8 ■ **Subxiphoid View.** The transducer is directed under the xiphoid process toward the left shoulder in a horizontal plane. (Photo contributor: Lawrence B. Stack, MD.)

Abnormal Findings

- Hemopericardium (pericardial effusion): Pericardial fluid will appear as an anechoic (black) region noted between the pericardium and the right ventricle. As more fluid collects, the fluid will be seen completely surrounding all four chambers of the heart. Occasionally, internal echoes representing fibrin, clot, or cardiac tissue may be present within the pericardial space (Fig. 24.10).
- Asystole: No cardiac activity present.
- Hyperdynamic cardiac activity: Extensive cardiac contraction with near-total or complete collapse of the cardiac chambers, often associated with tachycardia and hypovolemia.

Pearls

1. When the view is obscured by gas, slide the transducer slightly to the patient's right subcostal region, and use the right lobe of the liver as the sonographic window.



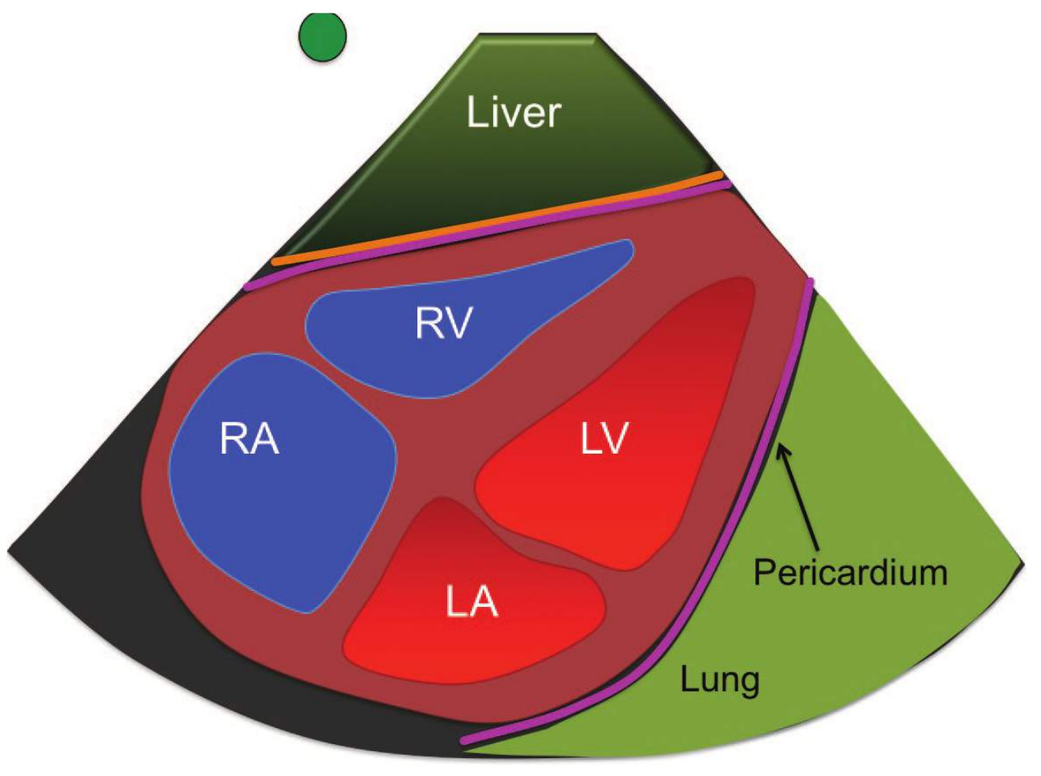


FIGURE 24.9 ■ **Subxiphoid View.** The heart, four cardiac chambers, and surrounding pericardium are seen in this view. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

2. If unable to view the heart in the true subxiphoid or subcostal window, move to a parasternal long-axis view (see "Focused Cardiac Ultrasound" below).
3. A frequent mistake in imaging is to direct the transducer posterior toward the spine rather than cephalad toward the shoulder. You will often require less than a 30-degree angle between the transducer and the skin to view the heart.
4. Start imaging with the depth/scale setting near its maximum (eg, 24-35 cm). This should allow you to image the anterior and posterior pericardium in your initial view. Gradually decrease the depth/scale (eg, 14-18 cm) to fill the entire sector image with the heart as you continue to optimize your image.
5. Hypotensive trauma patients with a pericardial effusion and findings such as right ventricular (RV) collapse and/or a distended inferior vena cava (IVC) warrant emergent pericardiocentesis (see Fig. 24.34).

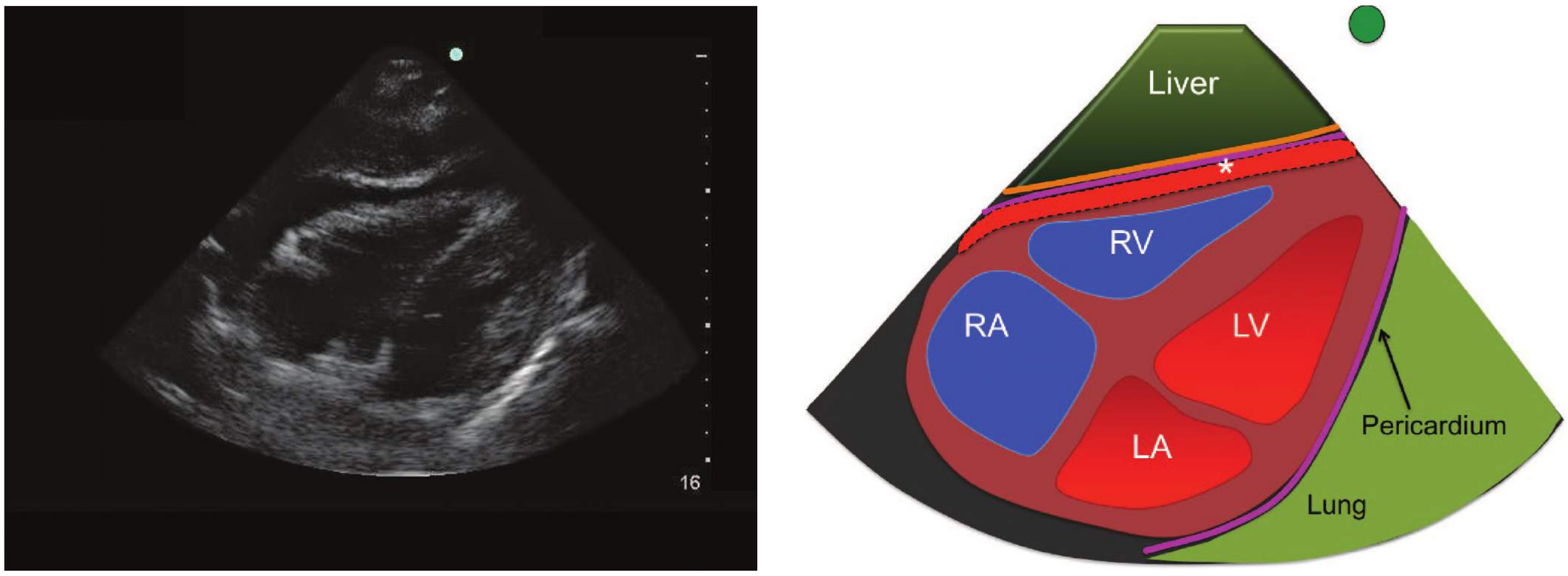


FIGURE 24.10 ■ **Hemopericardium**. Pericardial fluid appears as an anechoice (black) region noted between the pericardium and the right ventricle. Hemopericardium is denoted by the asterisks in the diagram. (Ultrasound contributor: Jim Fiechtl, MD; illustration courtesy of Robinson M. Ferre, MD.)

RIGHT UPPER QUADRANT (POUCH OF MORISON)

Technique

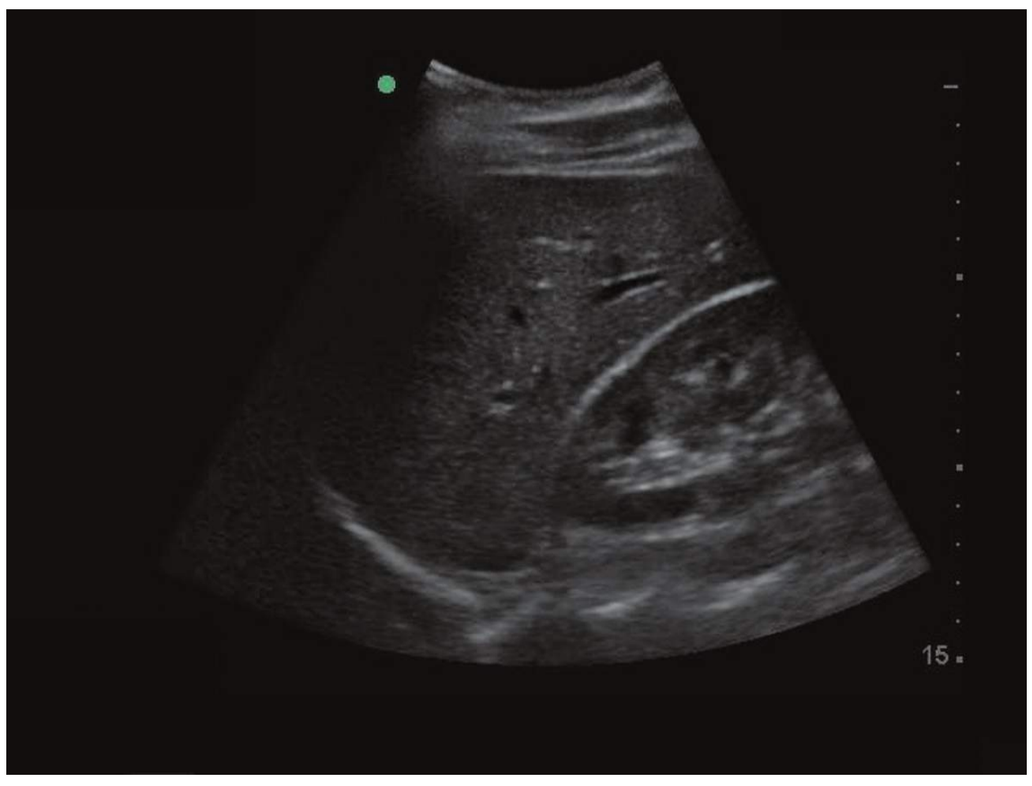
- With the indicator directed toward the patient's head, the transducer is oriented in a coronal section through the body in the midaxillary line, extending from the 9th through 12th ribs. Start between the 11th and 12th ribs initially, then move cephalad or caudal, anterior or posterior, to complete the evaluation (Fig. 24.11).
- Identify and evaluate the interface of the liver and right kidney. This region is the potential space known as the pouch of Morison. Normally, the liver and kidney are in direct contact with one another or separated by adipose tissue of heterogeneous echoes (Fig. 24.12).
- Evaluate the right paracolic gutter by identifying and evaluating the caudalmost portion of the hepatic parenchyma (inferior tip of the liver).

Abnormal Findings

- Hemoperitoneum: Anechoic (black) region between the liver and right kidney or in the paracolic gutter. Rarely is blood only seen collecting between the diaphragm and the liver in the subdiaphragmatic space (Fig. 24.13).
- Solid organ injury: Solid organ injury such as hepatic and renal lacerations as well as organ rupture have been described but are not the goal of this examination as ultrasound is an insensitive exam for solid organ injury.



FIGURE 24.11 ■ **Right Upper Quadrant View.** The transducer is oriented in a coronal section through the body in the midaxillary line extending from the 9th through 12th ribs. (Photo contributor: Lawrence B. Stack, MD.)



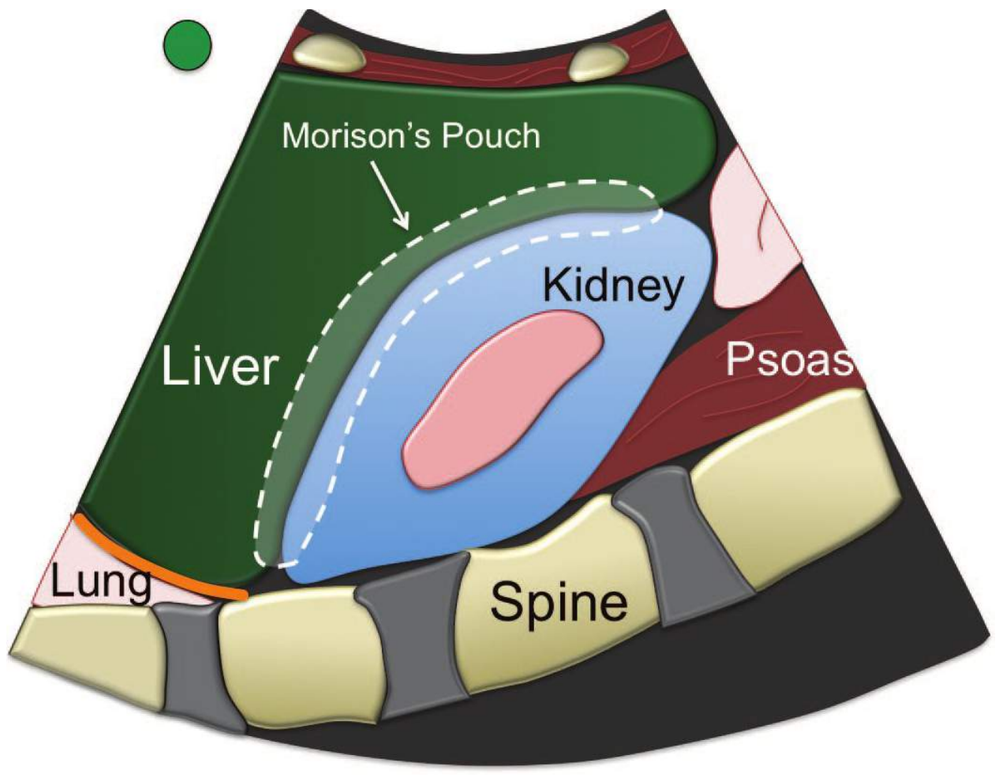
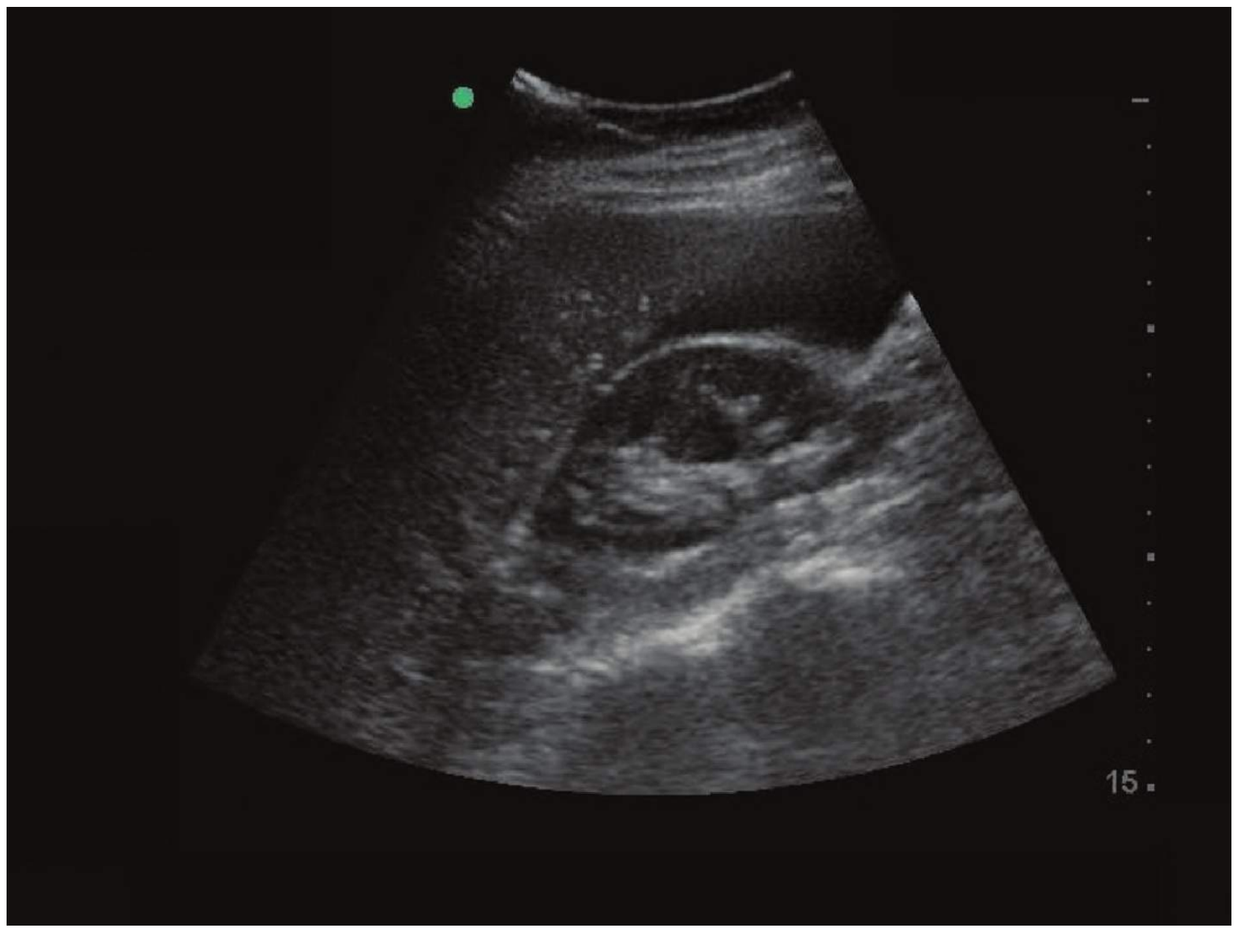


FIGURE 24.12 ■ **Normal Right Upper Quadrant View (Morison Pouch).** Identify and evaluate the interface of the liver and right kidney. This region is the potential space known as the pouch of Morison. Normally, the liver and kidney are in direct contact with one another or separated by adipose tissue of heterogeneous echoes. The reflection of the anterior border of the lumbar spine is visualized deep to the kidney and terminates at the costophrenic angle in the absence of fluid within the thoracic cavity. (Ultrasound contributor: Jeremy S. Boyd, MD; illustration courtesy of Robinson M. Ferre, MD.)



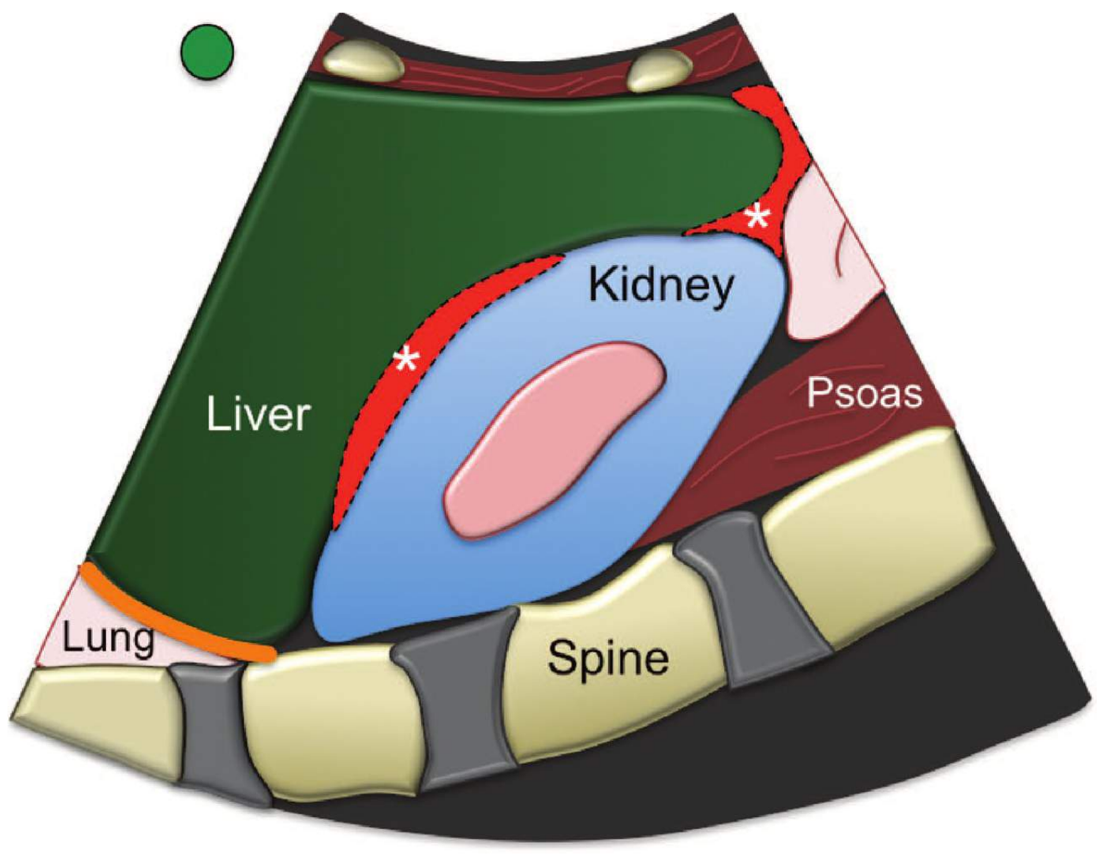


FIGURE 24.13 ■ **Hemoperitoneum (Right Upper Quadrant view).** An anechoic (black) region between the liver and right kidney is fluid (blood in this case) accumulating within the potential space. The diagram illustrates the typical locations of these peritoneal potential spaces, marked by asterisks. (Ultrasound contributor: Jeremy S. Boyd, MD; illustration courtesy of Robinson M. Ferre, MD.)

LEFT UPPER QUADRANT (PERISPLENIC VIEW)

Technique

- The transducer indicator is directed toward the axilla.
- With the indicator pointed toward the patients head, the transducer is oriented in a coronal section through the body in the mid to posterior axillary line extending from the 9th through 12th ribs. Start between the 11th and 12th ribs initially, then move cephalad or caudal, anterior or posterior, to complete the evaluation (Fig. 24.14). As a general rule of thumb, the perisplenic view is more posterior and cephalad than that of the RUQ view.
- Identify and evaluate the area surrounding the spleen, including its upper and lower poles, the interface with the diaphragm, and the interface with the left kidney. Normally, the surrounding tissues of the spleen and kidney are in direct contact with one another (Fig. 24.15).

Abnormal Findings

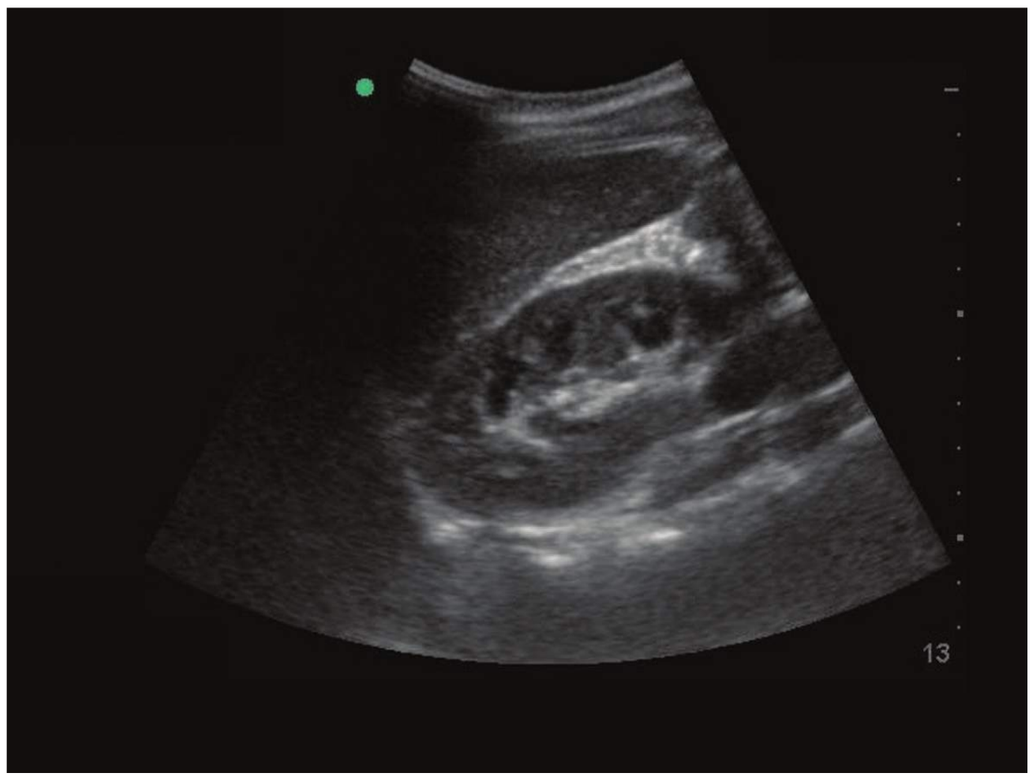
- Hemoperitoneum: Anechoic (black) region around the spleen. This may be visible at the superior or inferior poles of the spleen, between the spleen and the diaphragm or between the spleen and left kidney. Unlike the pouch of Morison, blood cannot flow beyond the inferior pole of the spleen down the paracolic gutter due to the phrenicocolic ligament. Blood that collects in the perisplenic space must first pass out of the lesser peritoneal sac and into the greater peritoneal sac before it will be seen collecting along the left paracolic gutter (Fig. 24.16).
- Solid organ injury: Solid organ injury such as splenic and renal lacerations as well as organ rupture have been described but are not the goal of this examination as ultrasound is an insensitive exam for solid organ injury.

Pearls for RUQ and LUQ Views

1. The diaphragmatic recess includes a superior region, which is the inferior border of the right thorax (often referred to as the costophrenic angle), and an inferior region (subdiaphragmatic recess), which is the superior border of the abdomen. Fluid in the diaphragmatic recess



FIGURE 24.14 ■ **Left Upper Quadrant View.** The transducer is oriented in a coronal section through the body in the mid to posterior axillary line extending from the 9th through 12th ribs. (Photo contributor: Lawrence B. Stack, MD.)



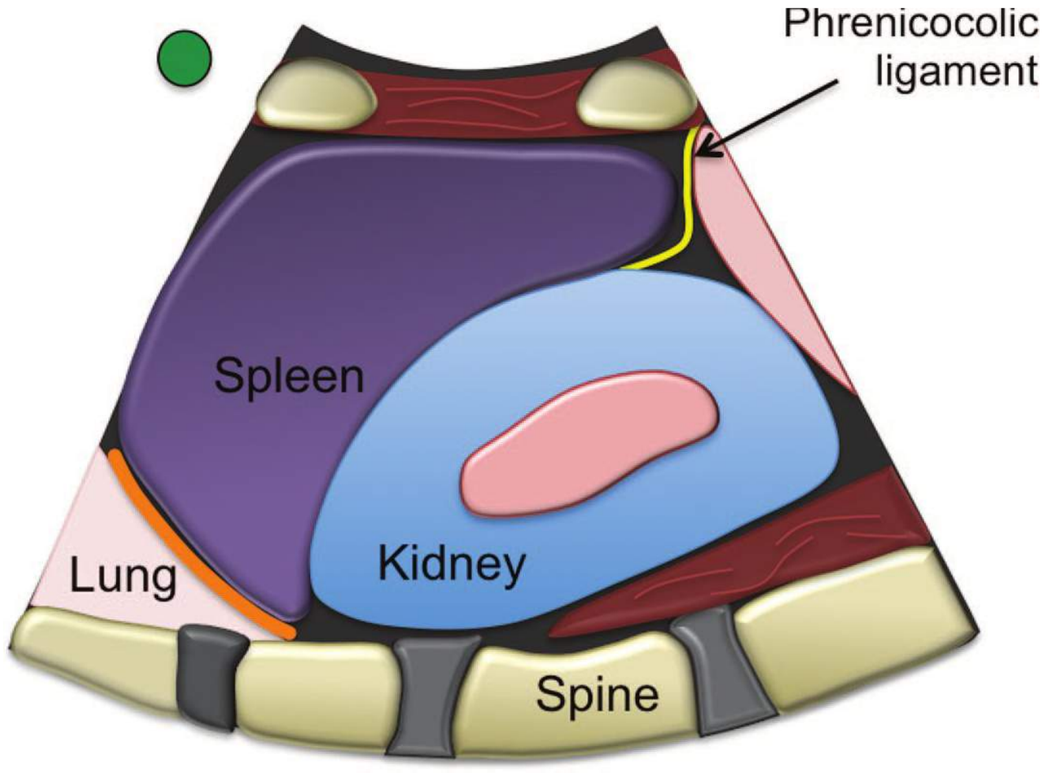
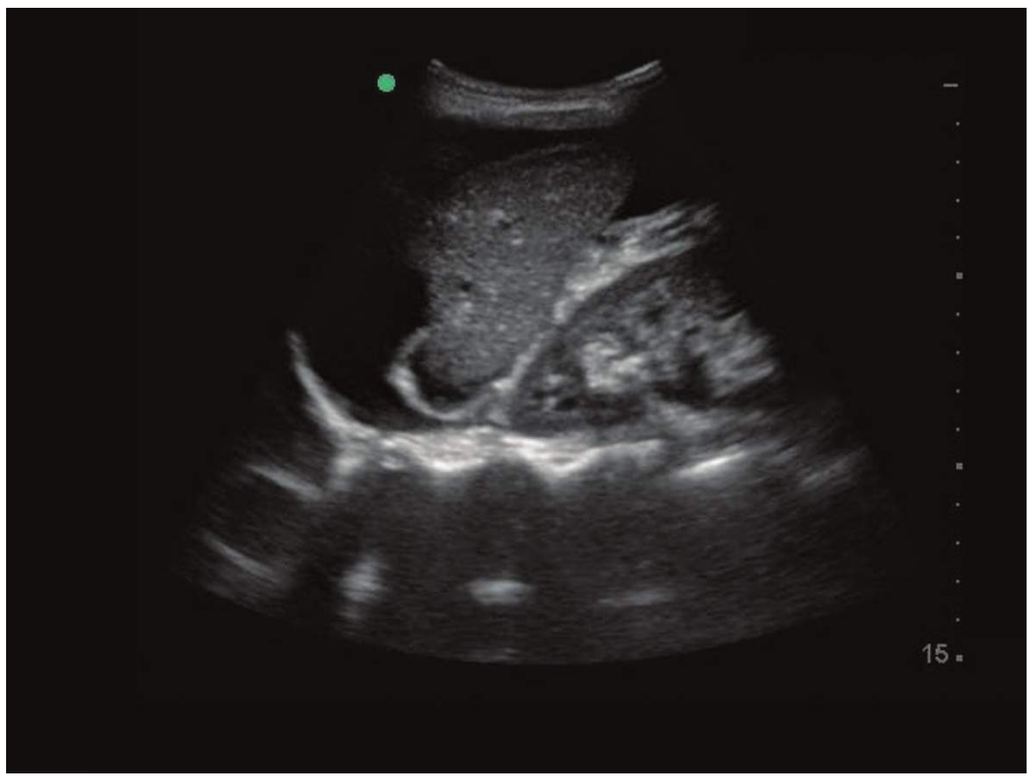


FIGURE 24.15 ■ **Normal Left Upper Quadrant View.** The region between the spleen and left kidney is a physiologic potential space (splenorenal recess); however, due to the phrenicocolic ligament (rarely seen on ultrasound but shown on illustration), free fluid is often shunted toward the subdiaphragmatic space between the diaphragm (orange line) and spleen. (Ultrasound contributor: Jeremy S. Boyd, MD; illustration courtesy of Robinson M. Ferre, MD.)

can represent a hemothorax when located superior/cephalad to the diaphragm or a hemoperitoneum (inferior to the diaphragm) in the setting of trauma.

2. If you are uncertain whether a finding is real or artifact, evaluate it in a second plane. Turn the transducer 90 degrees from your initial transducer position to see if the finding is still noted on the image. If the entire image is unchanged, it is less likely to be an artifact.
3. The liver is affixed to the diaphragm via the coronary ligament but the spleen lacks a similar attachment. Dependent fluid collects in the left subdiaphragmatic area more often than the right and this space should be evaluated sonographically during the examination.



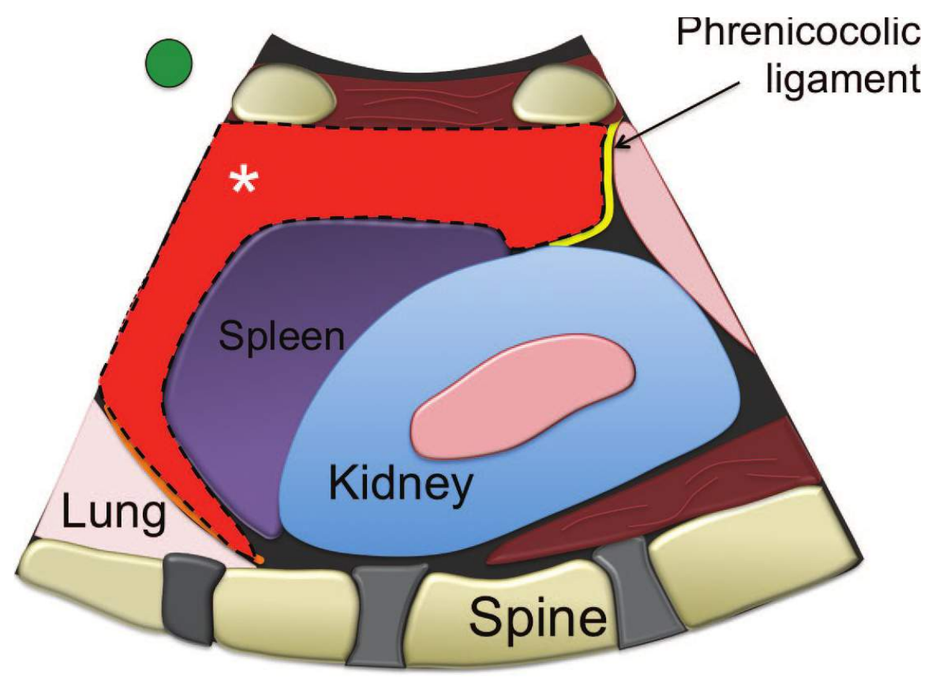


FIGURE 24.16 ■ **Hemoperitoneum, Left Upper Quadrant View.** In this view, fluid is seen in the subdiaphragmatic space between the diaphragm and the spleen. The diagram illustrates other common potential sites for free fluid, marked by asterisks. (Ultrasound contributor: Jeremy S. Boyd, MD; illustration courtesy of Robinson M. Ferre, MD.)

Technique

Sagittal View (Longitudinal)

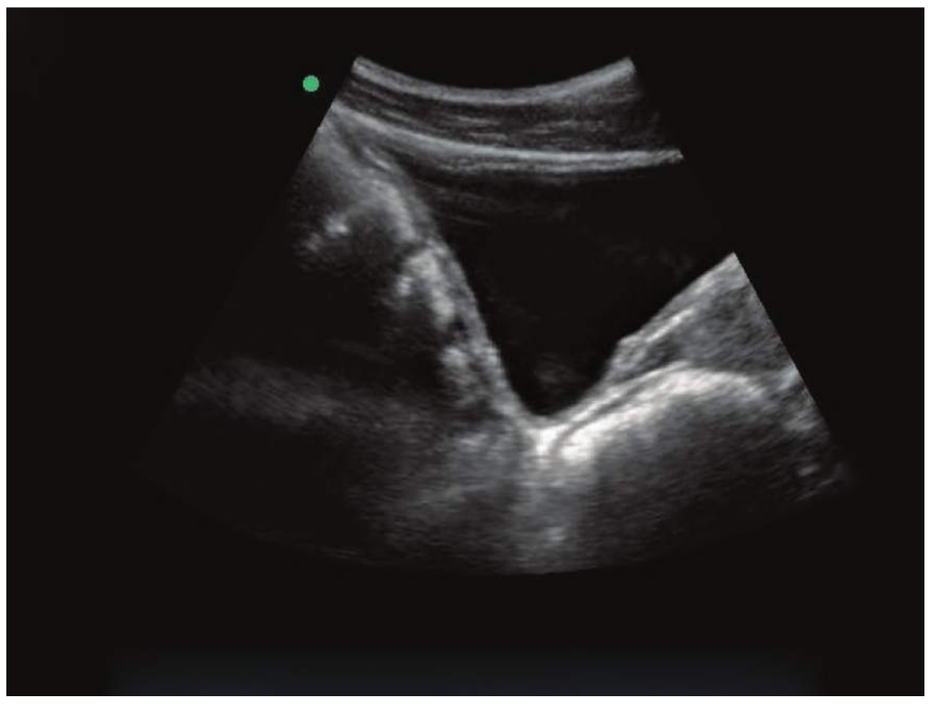
- With the indicator oriented toward the patient's head, the transducer is placed just above the symphysis pubis and is directed into the pelvis (Fig. 24.17).
- Identify the bladder (triangular in this view when fully distended), uterus (pear-shaped if present), prostate, seminal vesicles, and rectum (Figs. 24.18 and 24.19).

Technique: Transverse View

- With the indicator oriented toward the patient's right, the transducer is placed about 1 to 2 cm above the symphysis pubis and the beam is angled through the bladder into the peritoneum (Fig. 24.20).



FIGURE 24.17 ■ **Suprapubic Sagittal View.** The transducer indicator is oriented toward the patient's head and the transducer is placed just above to the symphysis pubis and is directed into the pelvis. (Photo contributor: Lawrence B. Stack, MD.)



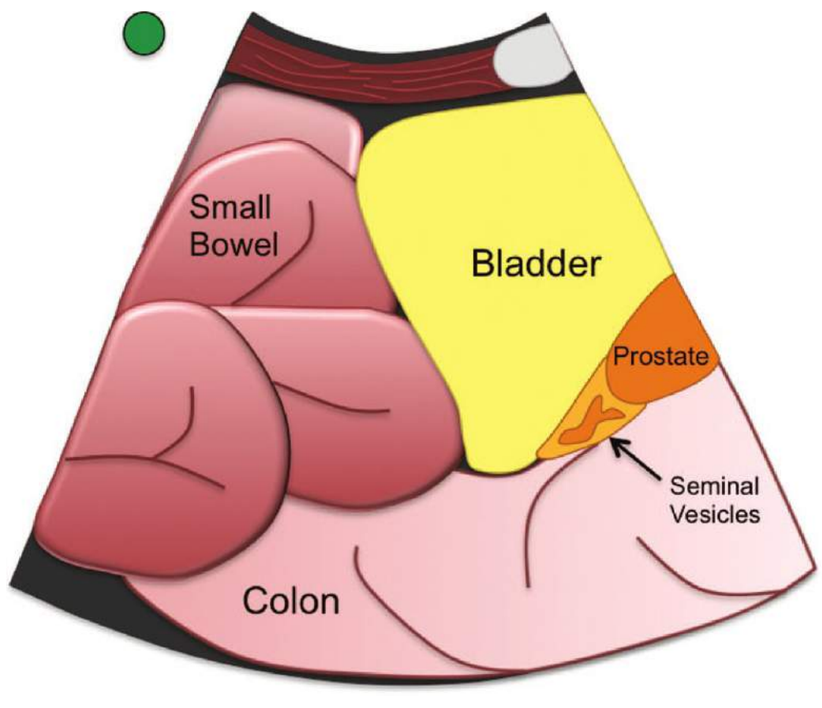


FIGURE 24.18 ■ **Normal Male Pelvis, Sagittal View.** The bladder appears triangular in this view. The prostate and seminal vesicles can be seen to the right of the screen, just deep to the bladder. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)



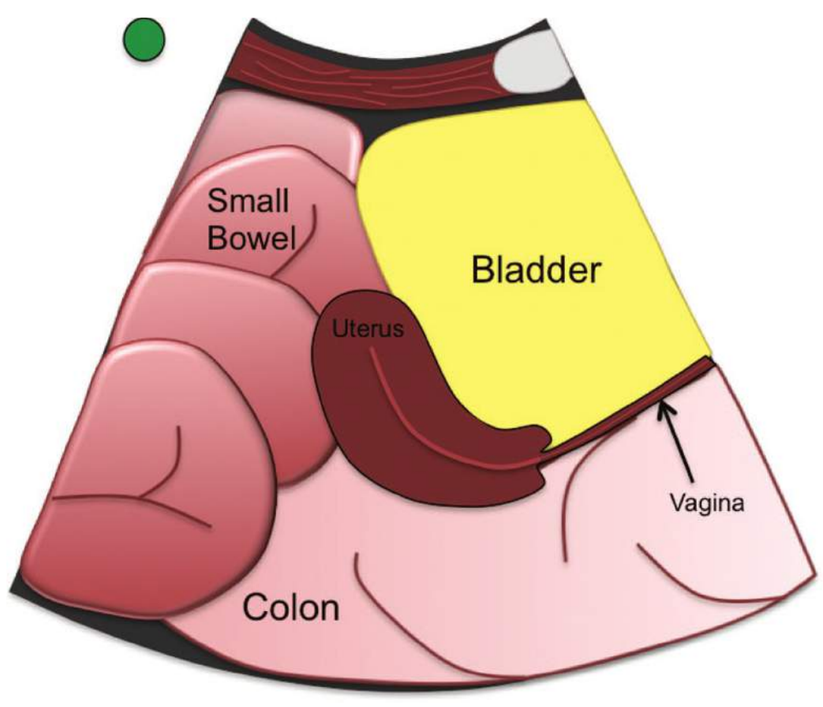


FIGURE 24.19 ■ **Normal Female Pelvis, Sagittal View.** The uterus appears pear-shaped in this view, and may be anteroflexed or retroflexed. The "pouch of Douglas" is a potential space between the uterus and the rectum, and is deep to the uterus. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)



FIGURE 24.20 ■ **Suprapubic View, Transverse.** The indicator is oriented to the patient's right and the beam angled through the bladder into the peritoneum. (Photo contributor: Lawrence B. Stack, MD.)

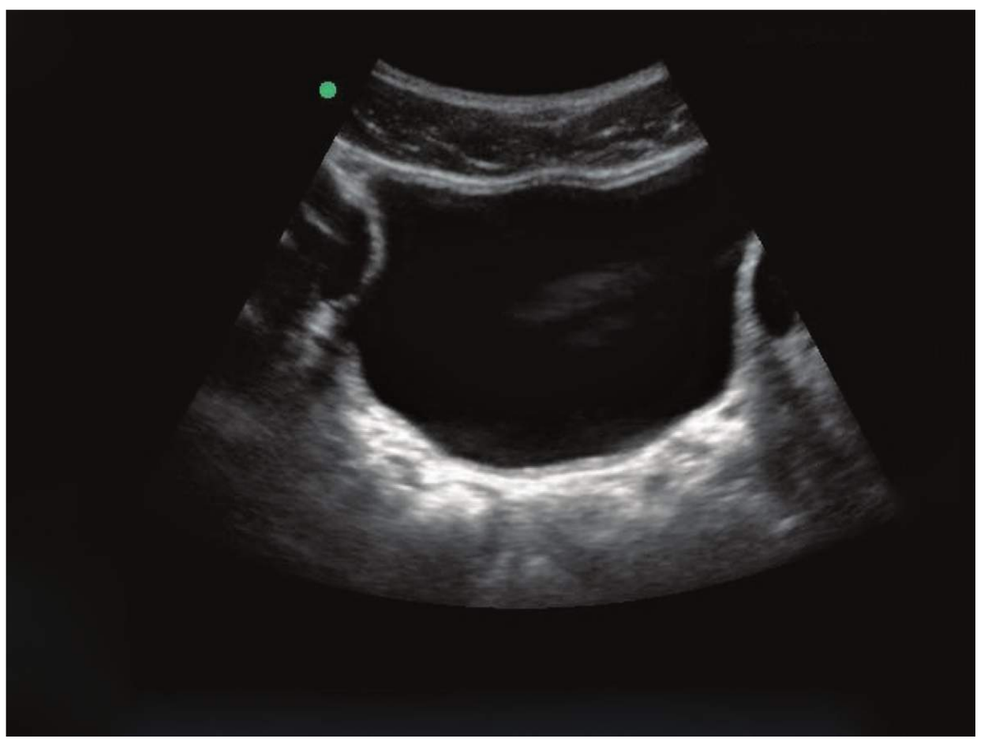
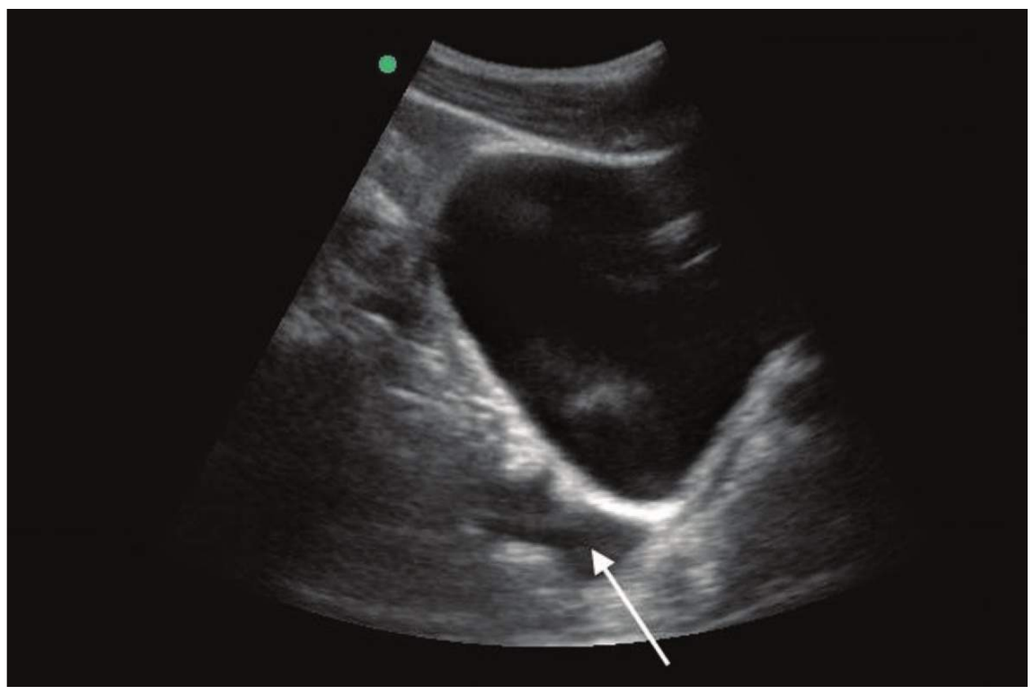


FIGURE 24.21 ■ **Suprapubic View, Transverse.** The bladder appears rectangular in this view. The uterus/cervix or prostate/seminal vesicles can be seen deep to the bladder. (Ultrasound contributor: Jeremy S. Boyd, MD.)

- Identify the bladder (rectangular in this view when fully distended), uterus (oval hyperechoic structure if present), prostate, seminal vesicles, and rectum (Fig. 24.21).

Abnormal Findings

- Hemoperitoneum (male pelvis): Free fluid in the male pelvis is seen as anechoic (black) areas filling the rectovesicular space, the potential space between the bladder and the rectum. The rectovesicular space is immediately cephalad to the extraperitoneal prostate and seminal vesicles. In the sagittal view, it is easier to see this junction that distinguishes the peritoneal cavity from the extraperitoneal pelvis. A large amount of free fluid may appear as loops of bowel floating lateral to the bladder in the transverse view and superior to the bladder in the sagittal view (Figs. 24.22 and 24.24).
- Hemoperitoneum (female pelvis): Free fluid in the female pelvis is seen as an anechoic (black) area in the rectouterine space, the potential space between the uterus and rectum. The vaginal stripe is an extraperitoneal structure and intraperitoneal fluid will not be seen collecting beneath it. A large amount of free fluid may appear as loops of bowel floating lateral to the bladder in the transverse view and superior to the bladder in the sagittal view (Figs. 24.23 and 24.24).



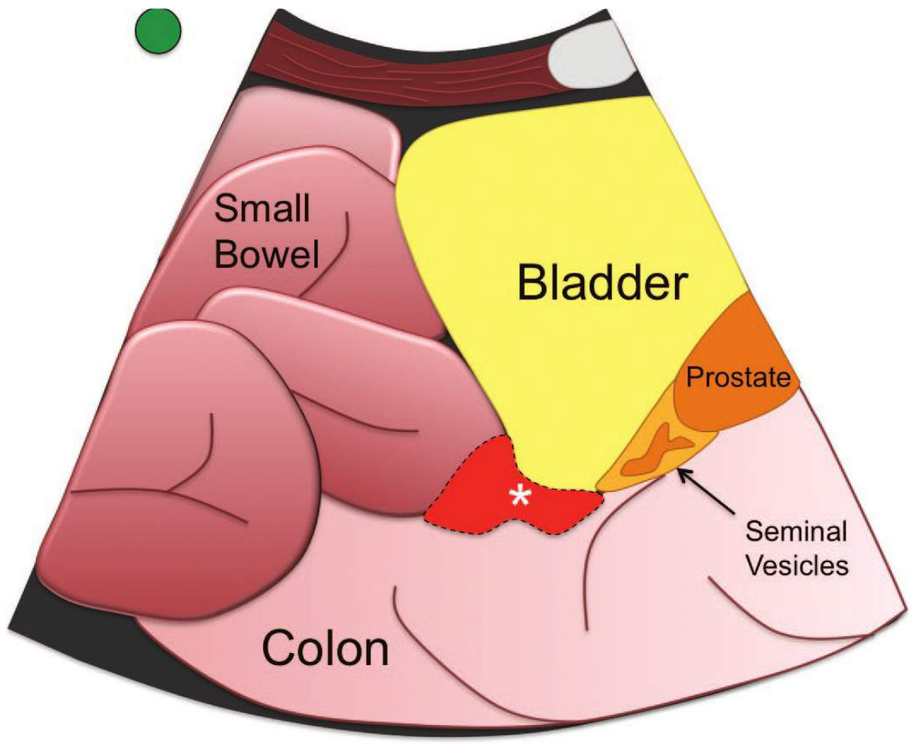


FIGURE 24.22 ■ **Hemoperitoneum, Sagittal Male.** Hemoperitoneum is seen deep to the bladder in the rectovesicular area on this sagittal view (arrow). Free fluid is seen as an anechoic (black) area in the rectovesicular space as demarcated by the asterisk. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

Pearls

1. It is important to remember that the bladder is within the pelvis; therefore, the transducer must be directed cephalad through the bladder into the peritoneum to evaluate for intraperitoneal fluid or hemorrhage in the rectovesicular or rectouterine space. If the prostate or vaginal stripe can be seen on a transverse suprapubic view, then the beam is directed caudal to the peritoneum and is missing from the peritoneal cavity entirely.
2. When in the sagittal plane, rotate the transducer 90 degrees counterclockwise directing the transducer indicator toward the patient's right to obtain a transverse view.
3. An empty bladder will not be able to sufficiently displace the small bowel from the pelvis. This often prevents an adequate view of the dependent portions of the pelvis to assess for free peritoneal fluid. Filling the bladder through a Foley catheter or waiting for the bladder to distend will allow a better view for free intraperitoneal fluid that has collected in the pelvis.

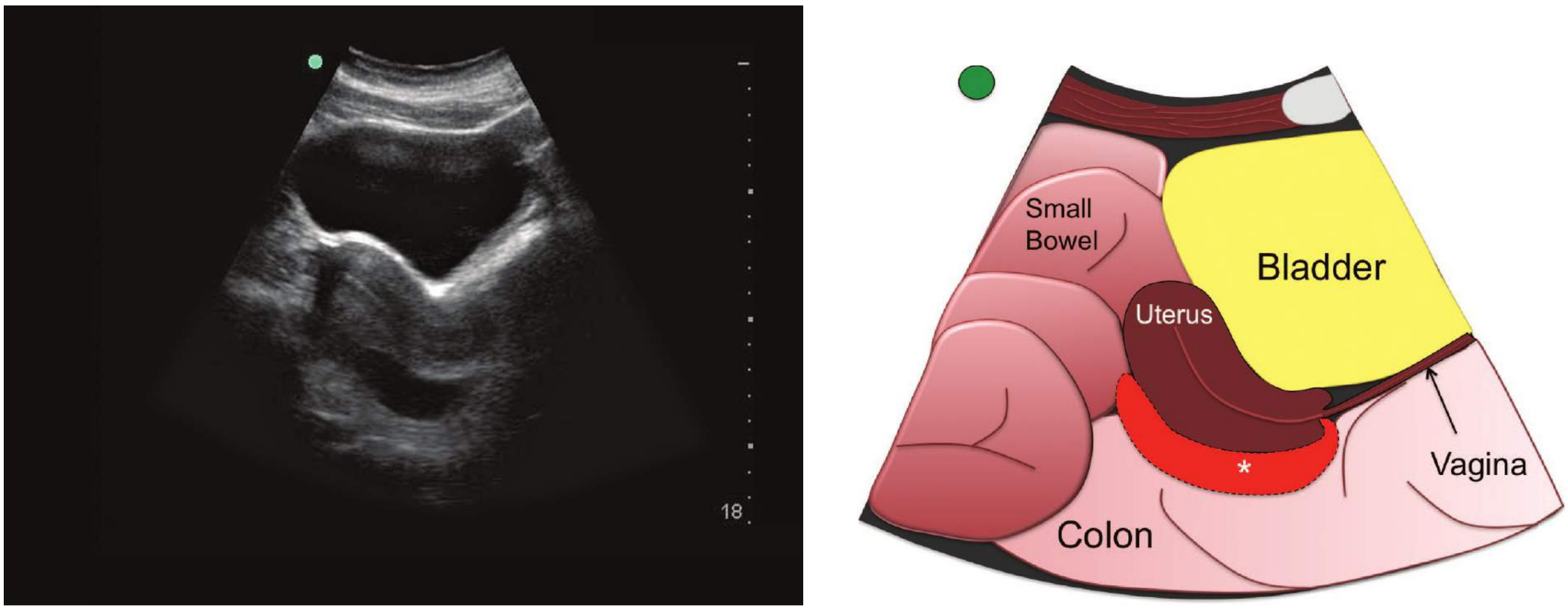


FIGURE 24.23 ■ **Hemoperitoneum, Sagittal Female.** Hemoperitoneum is seen deep to the uterus, in the pouch of Douglas. Free fluid is seen as an anechoic (black) area in the rectouterine space as demarcated by the asterisk. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

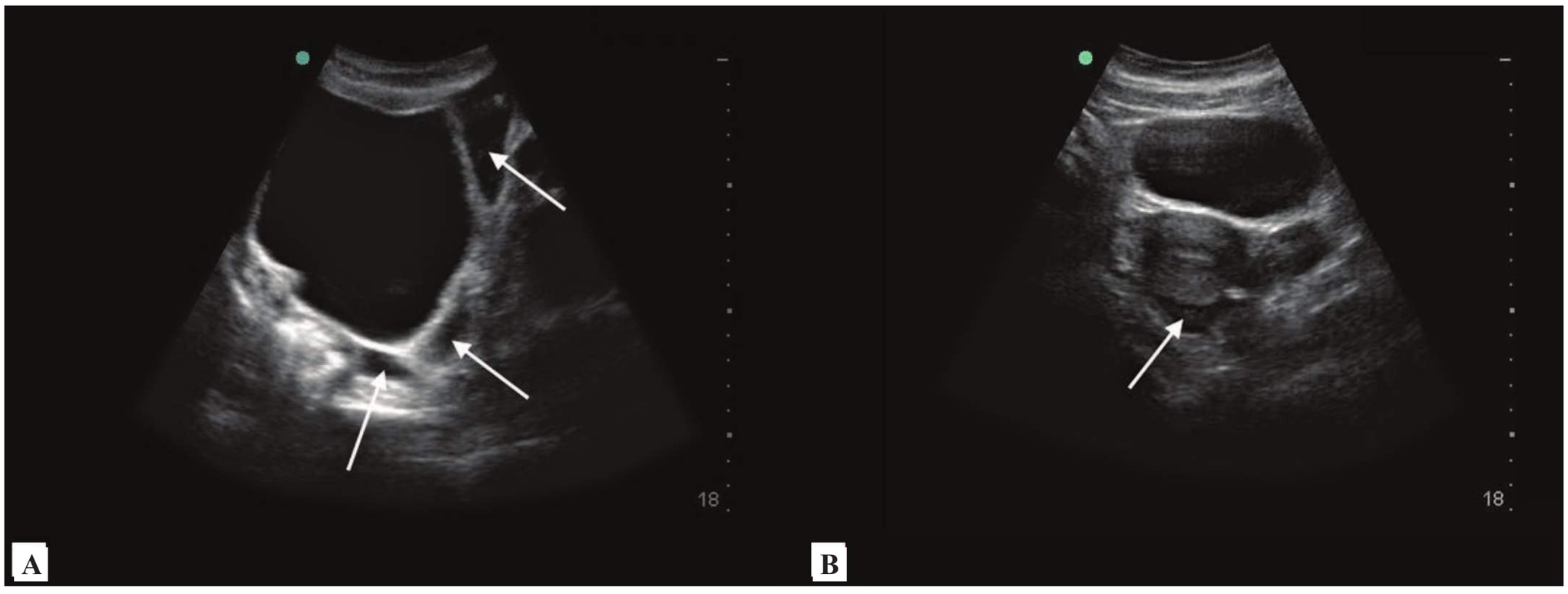


FIGURE 24.24 ■ **Hemoperitoneum, Transverse Male (A) and Female (B).** Hemoperitoneum (arrows) is seen deep and lateral to the bladder in the male pelvis (**A**), and deep to the uterus in the female pelvis (**B**). (Ultrasound contributor: Jeremy S. Boyd, MD; Robinson M. Ferre, MD.)

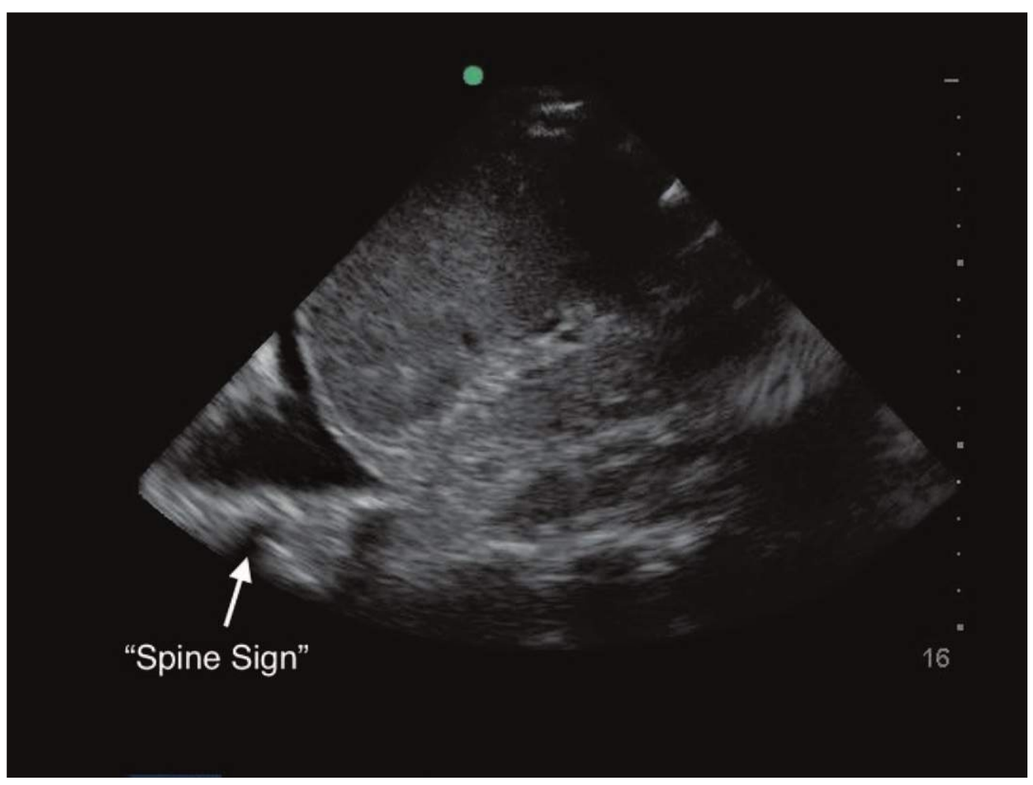
- Note that evaluation for thoracic fluid is best performed as an extension of the RUQ and LUQ views by sliding the transducer up into the chest in a coronal plane.

Technique

- A linear or convex transducer is preferred for evaluation of pneumothorax.
- A convex or phased array transducer is preferred for evaluation of hemothorax.
- The transducer indicator is oriented toward the patient's head.

Suspected Pleural Fluid (Hemothorax)

- With the indicator directed toward the patient's head, the transducer is placed in the fifth or sixth intercostal space in the midaxillary line.



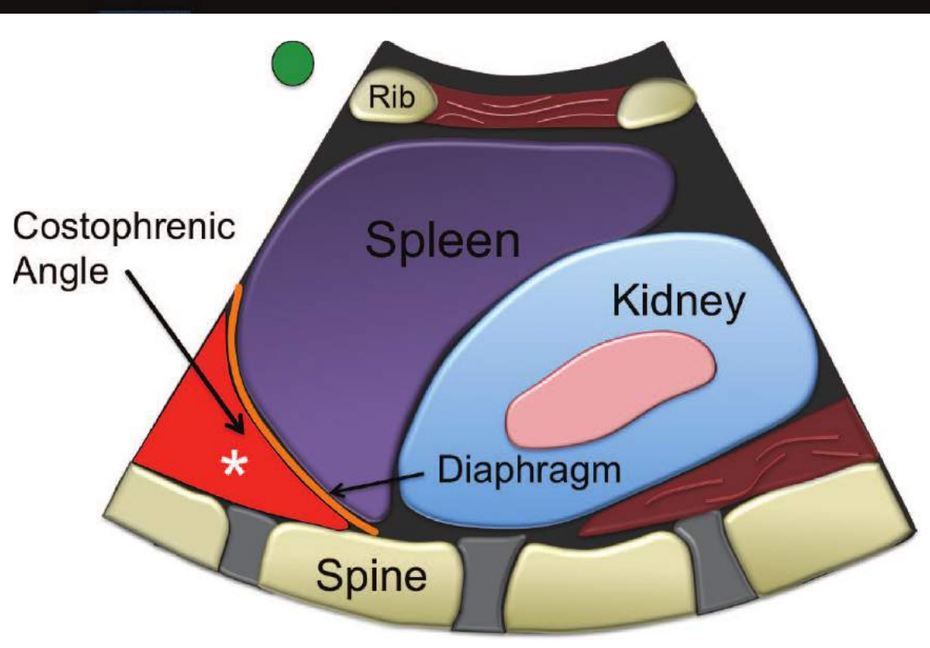


FIGURE 24.25 ■ **Hemothorax.** Fluid (in this case blood) is identified as an anechoic "wedge" above the diaphragm with loss of the normal mirroring artifact of the diaphragm allowing visualization of the spine extending beyond the costophrenic angle, also known as the "spine sign." (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

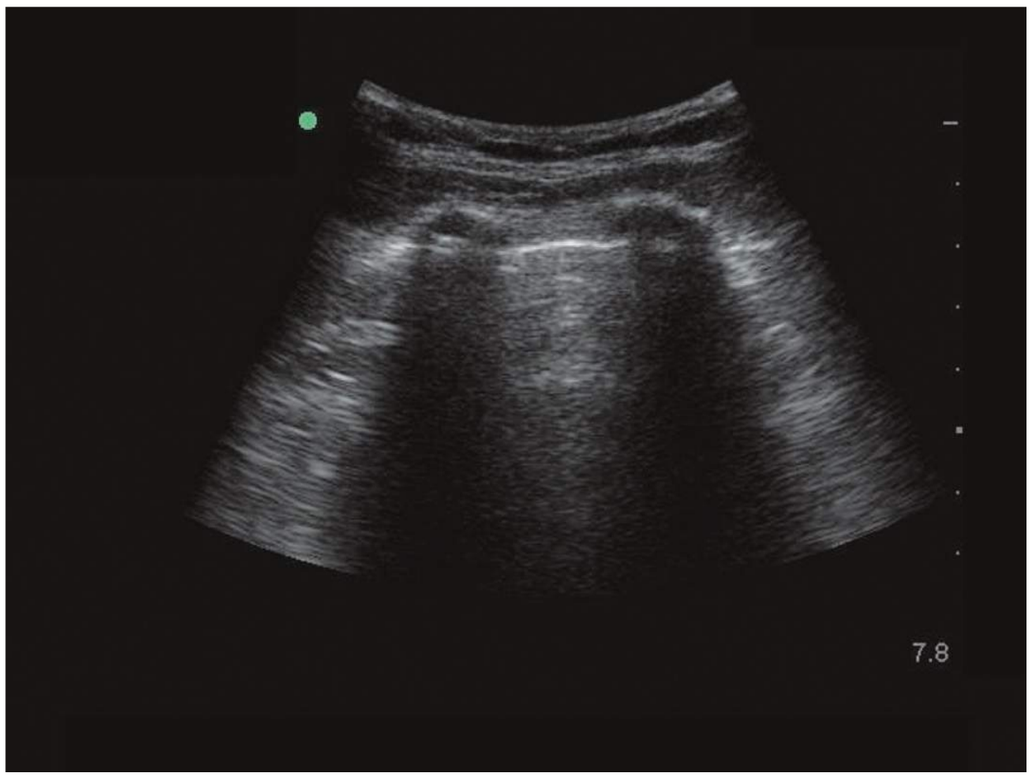
- Identify the liver (on the right) or the spleen (on the left), the diaphragm, and the vertebral bodies that lie at the bottom of the screen.
- In the fully inflated healthy lung, air prevents direct visualization of structures deep to the interface of the diaphragm and the visceral pleura of the lung. If fluid is present, it is identified as an anechoic (black) area that is caudal to the lung in the costophrenic recess. A hemothorax may have a simple anechoic appearance, but may also contain heterogeneous echoes from clotted blood or portion of lacerated lung tissue.
- The presence of pleural fluid will allow direct visualization of the lung and posterior structures, including the vertebral bodies of the thoracic spine often referred to as the "spine sign" (Fig. 24.25).

Suspected Pneumothorax

- When using ultrasound to evaluate for a pneumothorax, the transducer is placed in the superior most portion of the chest wall in a supine patient to assess for normal apposition of the lung. Unlike chest radiography, ultrasound must look for normal apposition of the visceral and parietal portions of the lung in a stepwise fashion. Indeed, it is important to note that ultrasound can only assess for the presence or absence of a pneumothorax on the portion of the lung, where the transducer is currently imaging. Thus, when using ultrasound, we assume that there is no prior history of pleural scarring or pleural adhesions.



FIGURE 24.26 ■ **Anterior Thorax View.** A linear transducer is placed below the clavicle in the midclavicular line dragged inferiorly to assess multiple rib spaces for pneumothorax. Other probe types may be used as well. (Photo contributor: Lawrence B. Stack, MD.)



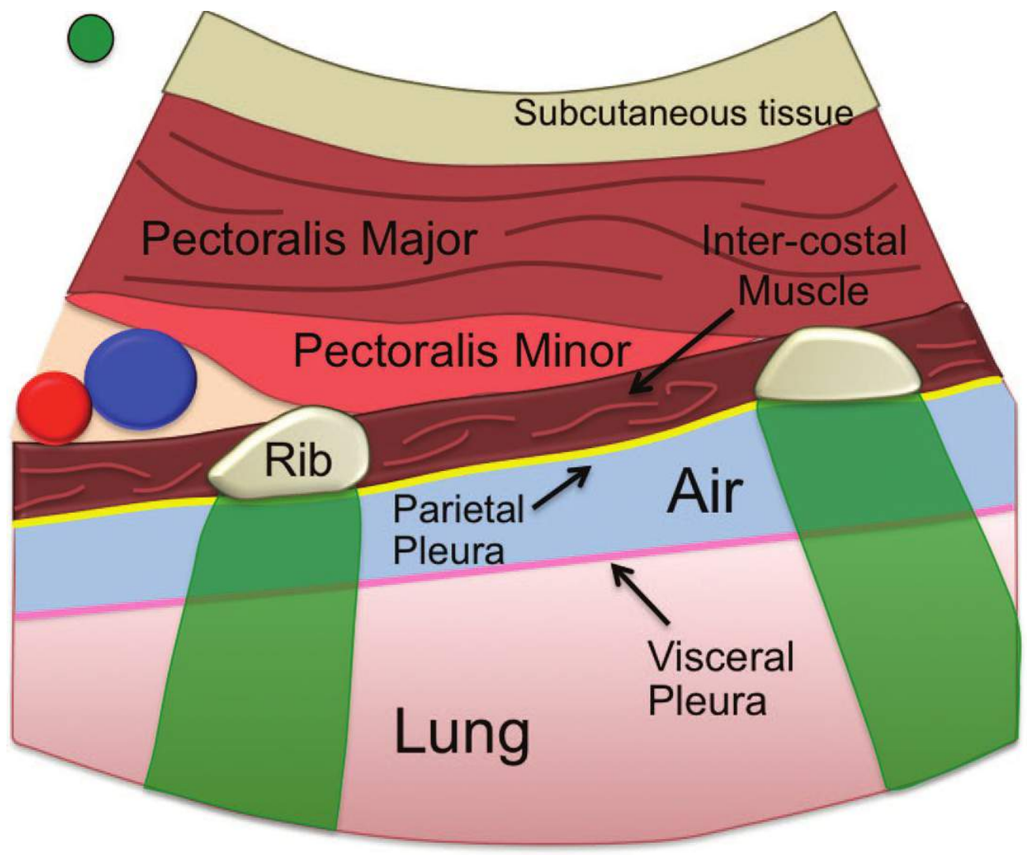


FIGURE 24.27 ■ **Pneumothorax**. A still image of a pneumothorax as seen on ultrasound using a curvilinear or convex array transducer. The diagram illustrates the layer of air between the pleura causing the absence of "lung sliding" seen in real-time imaging. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

- With the indicator directed toward the patient's head, the transducer is placed just below the clavicle in the midclavicular line. To provide maximum sensitivity for the presence of a pneumothorax, the probe should be dragged caudal in the midclavicular line until the diaphragm is seen just below the costal margin (Fig. 24.26).
- Once the probe is placed on the chest wall, identify the pectoralis and intercostal muscles and ribs with associated acoustic shadows that project to the bottom of the screen. The interface of the visceral and parietal pleura, known as the pleural line, will be seen immediately deep to the rib and intercostal muscle.
- The depth should be adjusted so that the pleural line is positioned in the middle or upper half of the screen.
- If there is normal apposition of the visceral and parietal pleura (no pneumothorax), sliding will be seen along the

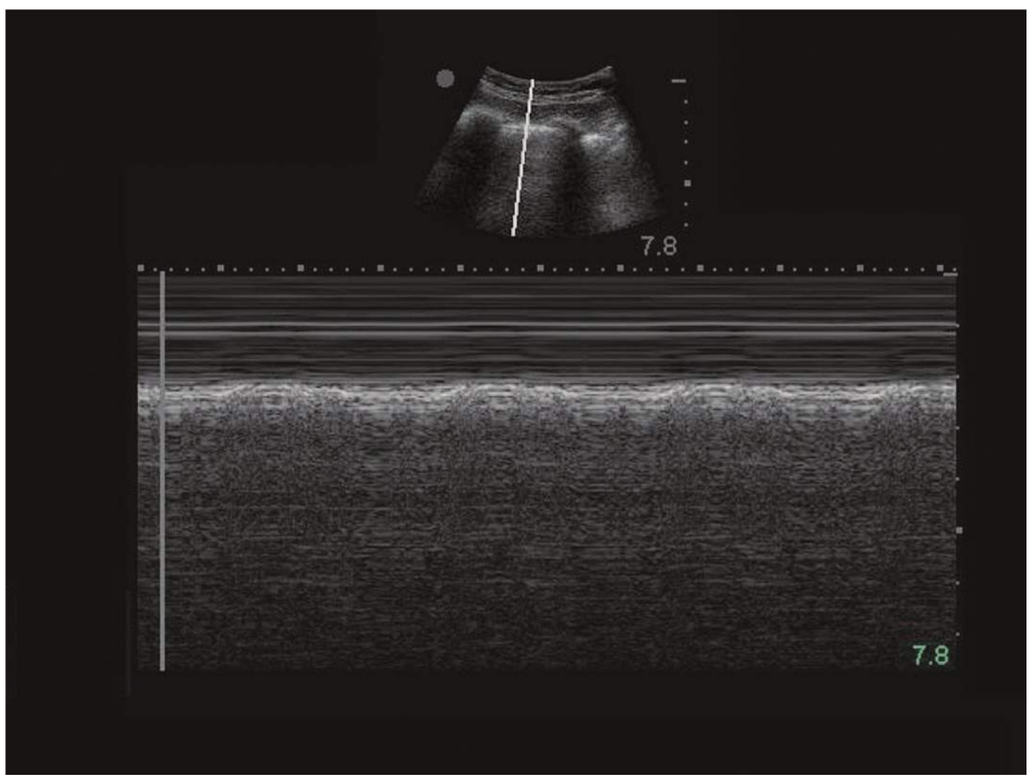


FIGURE 24.28 ■ **Seashore Sign.** The "seashore sign" describes the M-mode appearance on thoracic ultrasound when there is no pneumothorax. The near-field image resembles waves on a beach, and the far-field image resembles a sandy beach, an artifact caused by lung sliding. (Ultrasound contributor: Jeremy S. Boyd, MD.)

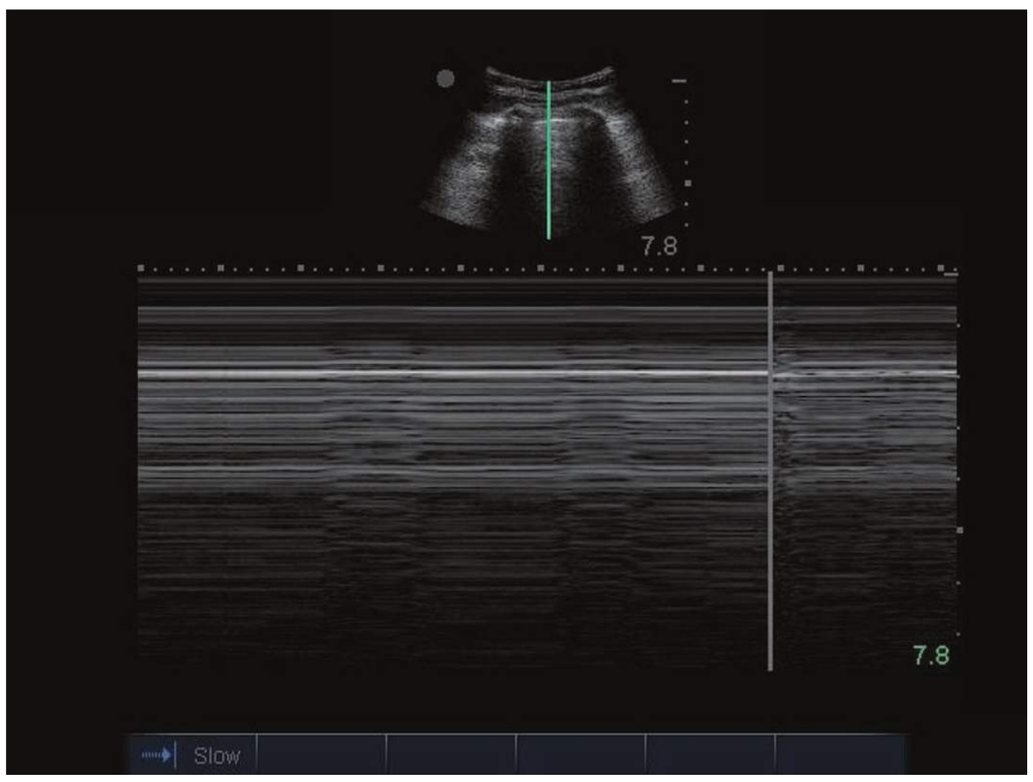


FIGURE 24.29 ■ **Barcode or Stratosphere Sign (Pneumothorax).** This describes the M-mode appearance on thoracic ultrasound when pneumothorax is present. The "sandy beach" is now gone due to the absence of lung sliding. (Ultrasound contributor: Jeremy S. Boyd, MD.)

pleural line during normal respiration. If a pneumothorax is present, no sliding will be seen (Fig. 24.27).

- B-lines, a type of comet tail artifact, are occasionally seen arising from the pleural line and will project to the bottom of the screen (see Lung Ultrasound). B-lines are only present if there is normal apposition of the visceral and parietal pleura. In essence, they "rule out" a pneumothorax at the portion of lung that is being imaged. If more than two B-lines are present in any one view, it indicates the presence of fluid within the extravascular lung tissue, which may indicate pulmonary contusion in the setting of trauma.
- M-mode can assist with the detection and documentation of normal lung sliding. The "seashore sign," described as "waves on the beach," is noted with M-mode when no pneumothorax is present (Fig. 24.28). Conversely, a "stratosphere sign" is seen when a pneumothorax is present, the result of the M-mode tracing only a reverberation artifact (Fig. 24.29).

Abnormal Findings

- Hemothorax: As visualized at the upper quadrants of the lateral chest, a hemothorax will appears as an anechoic (black) area between the diagphram and the lung within the costophrenic recess. Heterogeneous echoes may be present due to clotted blood, lacerated lung, or other material. The presence of pleural fluid will allow direct visualization of the lung and posterior structures, including the vertebral bodies of the thoracic spine often referred to as the "spine sign"(Fig. 24.25).
- Pneumothorax: The absence of sliding along the pleural line is indicative of a pneumothorax. If lung sliding is absent, one should search for the leading edge of the pneumothorax, also known as lung point. Estimation of the pneumothorax size is possible by evaluating progressively more lateral views of the thorax in the supine patient.

Pearl

1. Lung point is the point of transition from normal lung sliding to the absence of lung sliding that occurs at the leading edge of the pneumothorax. The finding of a "lung point" is 100% diagnostic of a pneumothorax (Fig. 24.30).

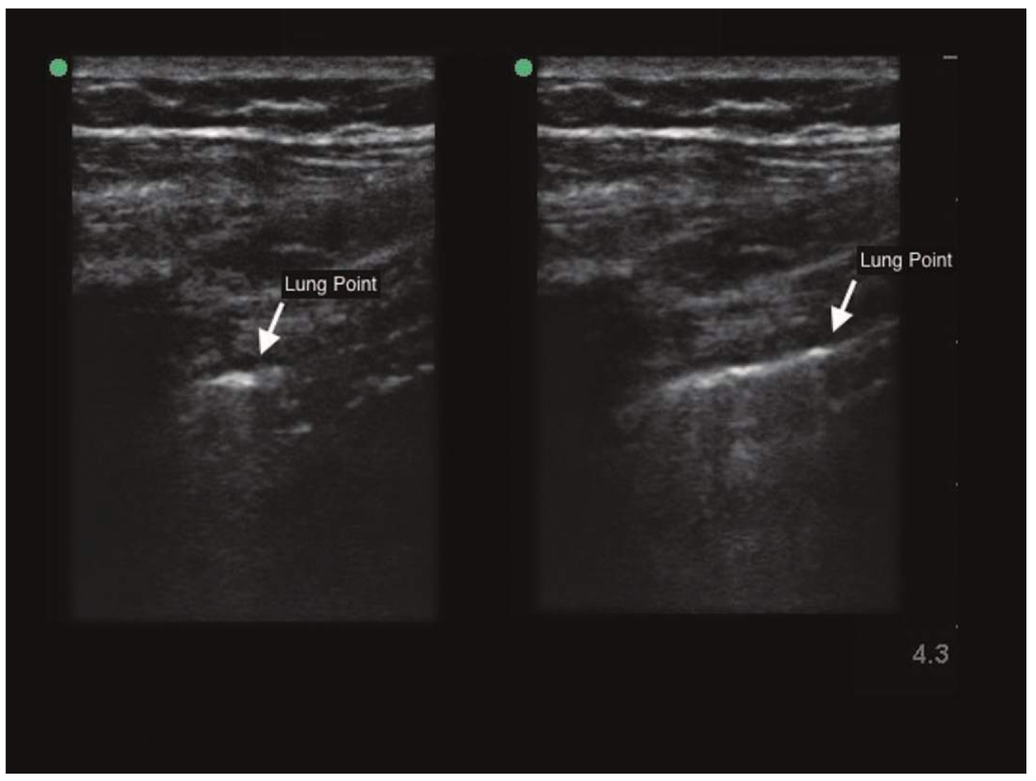


FIGURE 24.30 ■ **Lung Point.** The point of transition from normal lung sliding to the absence of lung sliding occurs at the leading edge of a pneumothorax. It is seen in this pair of images to move from left to right as the patient inhales. (Ultrasound contributor: Jeremy S. Boyd, MD.)

Clinical Summary

Focused cardiac ultrasound can yield significant diagnostic information for the patient presenting with cardiac arrest, shock, shortness of breath, and a host of other complaints or physical findings. Although the intricacies of comprehensive echocardiography are beyond the scope of practice of most emergency medicine providers, with experience one can incorporate focused bedside cardiac ultrasound into the diagnostic armamentarium safely to answer specific diagnostic questions such as cardiac activity, volume status, gross cardiac function, right heart strain, and the presence of pericardial effusion.

It is important to note that by convention, unlike abdominal sonography, cardiac ultrasound is viewed with the transducer indicator displayed on the right of the screen. This will require the indicator on the transducer to be directed toward the patient's left in an anatomically transverse view. This may be disorienting for many who have not performed cardiac ultrasound before. Most ultrasound systems include cardiac presets that automatically reverse the indicator orientation to the right of the display screen. The following section describes a sonographic approach for a conventionally oriented image using standard cardiac windows.

Indications

- Cardiac arrest, pulseless electrical activity (PEA)
- Penetrating thoracic/abdominal trauma
- Unexplained hypotension or shock
- Dyspnea
- Chest pain
- Acute myocardial infarction
- Suspected aortic dissection

Specific pathologic states investigated with bedside cardiac ultrasound include asystole, cardiac activity, pericardial effusion, acute heart failure, aortic root dilatation/dissection, and right heart enlargement.

The sonographic windows for focused cardiac ultrasound include the subxiphoid view presented within the trauma/FAST examination as well as parasternal and apical views. Focused cardiac ultrasound utilizes standard views that are familiar to cardiologists and sonographers alike. These five windows allow the emergency physician to effectively and efficiently evaluate emergent cardiopulmonary pathology.

Views for Focused Cardiac Ultrasound (Fig. 24.31)

1. Subxiphoid/subcostal (SUBX; see E-FAST Examination, Subxiphoid View)
2. Parasternal long-axis view (PSLA)
3. Parasternal short-axis view (PSSA)
4. Apical four-chamber view (AP4)
5. Subxiphoid long-axis view (IVC)

The technique and common findings for each of these views are presented in the next five topics.

Patient Positioning

- The patient should be optimally positioned in the left lateral decubitus position with the left arm above the head (allowing better access to the intercostal spaces) for all but the SUBX and IVC views, which should be performed with the patient supine. If this is not feasible, the patient may be supine for the entire study.

Recommended Transducer

- Phased array

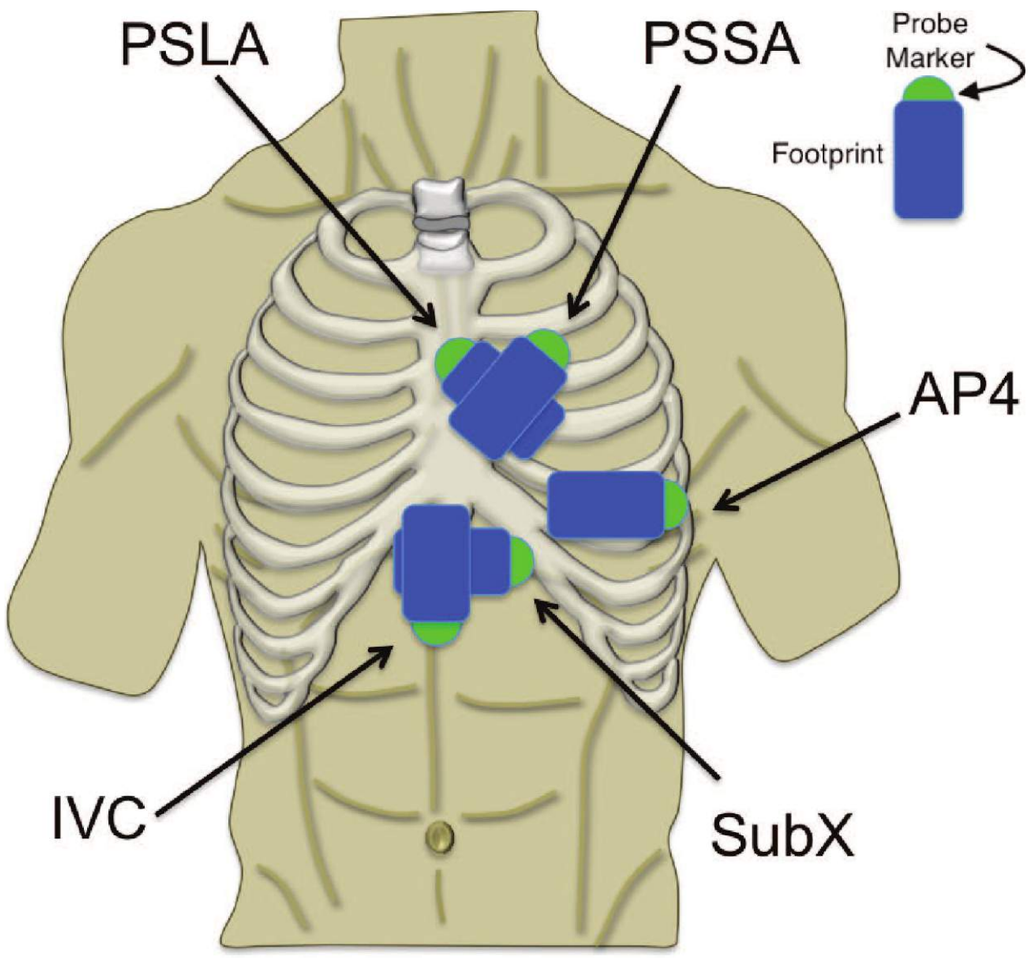


FIGURE 24.31 ■ **Focused Cardiac Ultrasound**. Transducer and probe marker positions for evaluation of the heart and IVC. (Illustration courtesy of Robinson M. Ferre, MD.)

Technique

- The transducer indicator should be directed toward the left in a cardiac preset.
- With the transducer indicator pointing to the patient's left, the transducer is placed inferior to the xiphoid process and directed cephalad toward the left shoulder in a horizontal plane (see Fig. 24.32).



FIGURE 24.32 ■ **Subxiphoid View.** With the transducer indicator pointing to the patient's left, the transducer is placed inferior to the xiphoid process and directed cephalad toward the left shoulder in a horizontal plane. (Photo contributor: Lawrence B. Stack, MD.)

- Pivot, sweep, and tilt the transducer as needed to view all four cardiac chambers.
- Identify the heart, four cardiac chambers, and surrounding pericardium (see Fig. 24.33).

Abnormal Findings

- Pericardial effusion: Pericardial fluid will appear as an anechoic (black) region noted between the pericardium and the right ventricle. As more fluid collects, the fluid will be seen completely surrounding all four chambers of the heart. Occasionally, internal echoes representing fibrin, clot, or cardiac tissue may be present within the pericardial space (Fig. 24.34).
- Asystole: No cardiac activity present.
- Hyperdynamic cardiac activity: Extensive cardiac contraction with near-total or complete collapse of the cardiac chambers, often associated with tachycardia and hypovolemia.

Pearl

See "Pearls" for the "E-FAST Examination, Subxiphoid View."

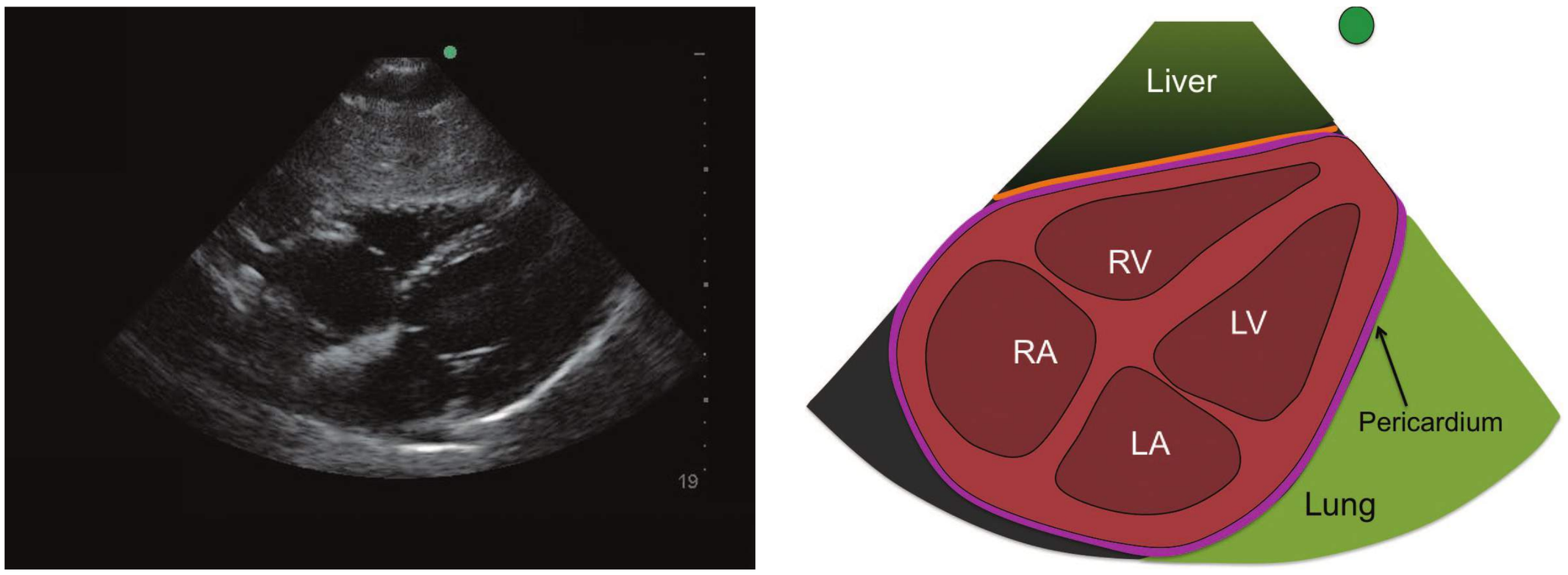
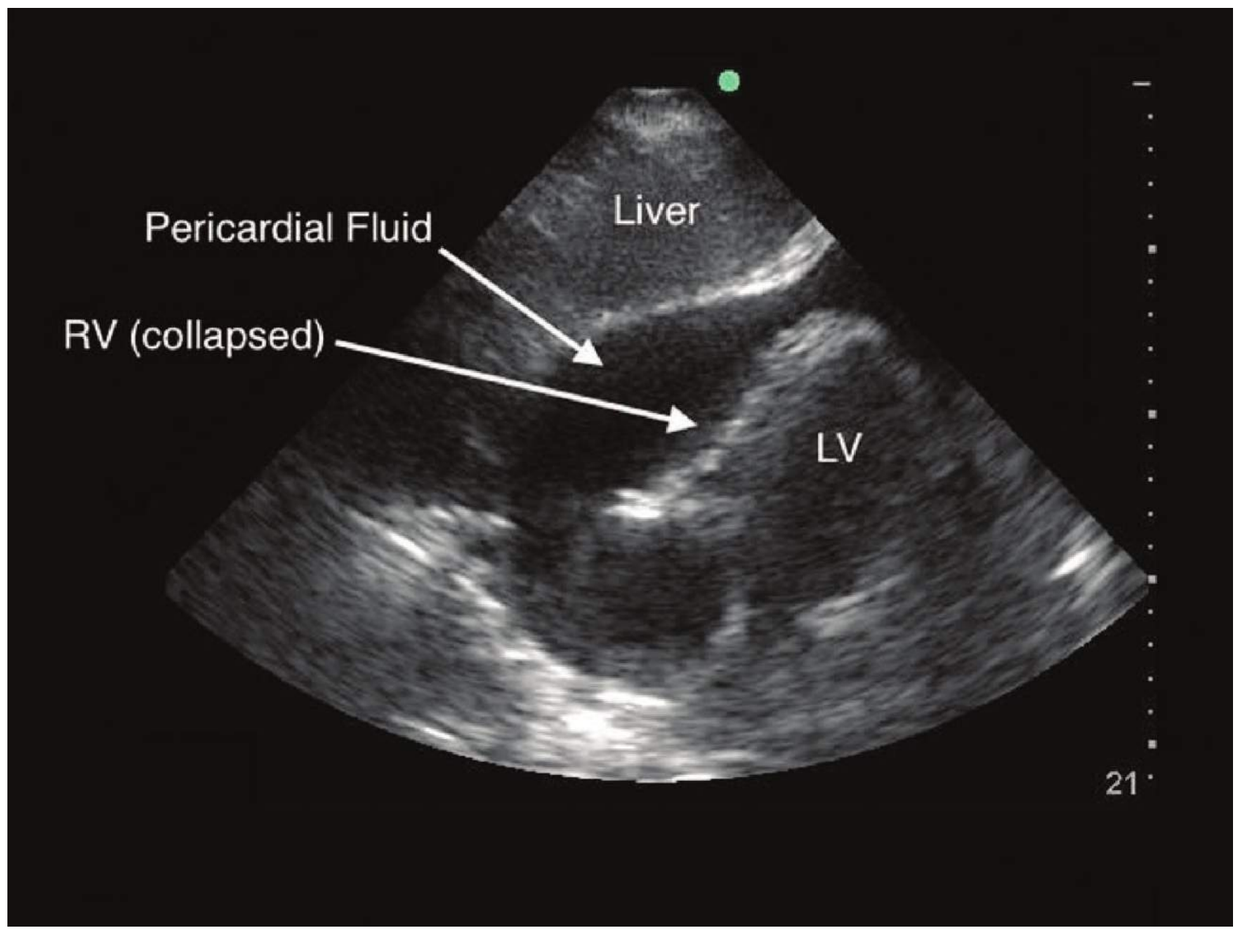


FIGURE 24.33 ■ **Subxiphoid View.** A normal view demonstrating the heart, four cardiac chambers, and surrounding pericardium. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)



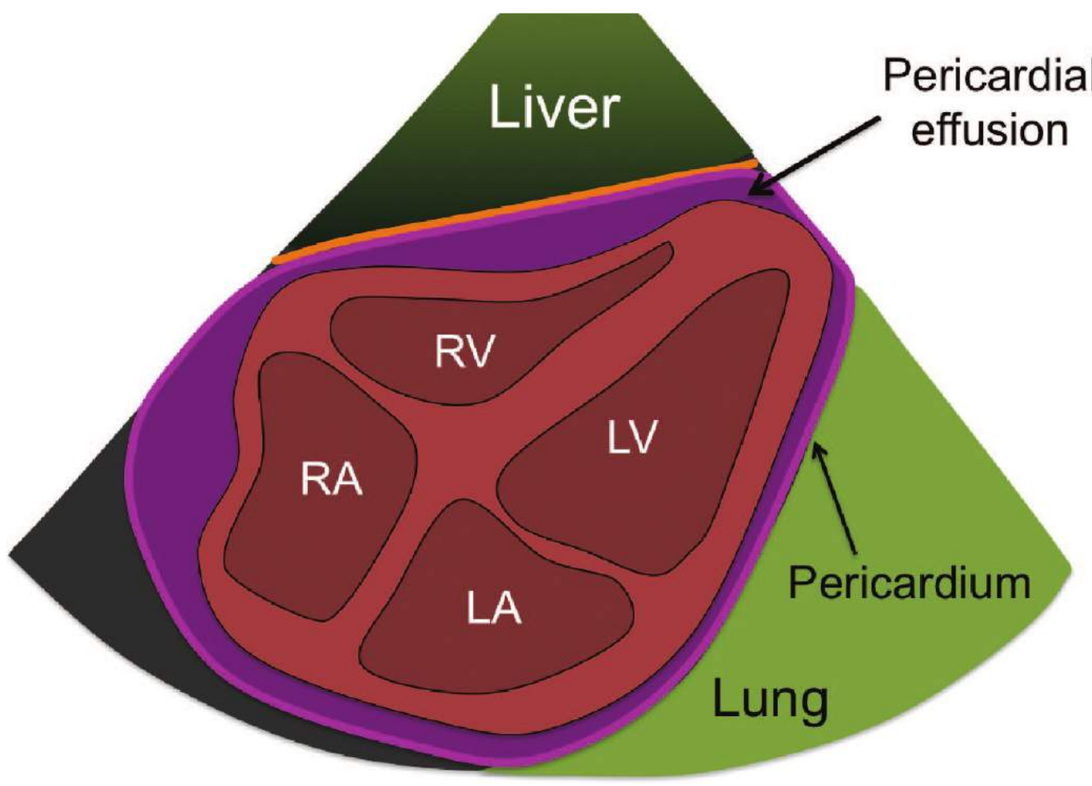


FIGURE 24.34 ■ **Pericardial Effusion**. Pericardial fluid will appear as an anechoic (black) region noted between the pericardium and the right ventricle. Hypotensive patients with a pericardial effusion and findings such as right ventricular collapse (illustration) and/or a distended IVC warrant consideration of emergent pericardiocentesis. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

Technique

- The transducer indicator directed at the right clavicle or shoulder.
- The transducer is placed in the fourth or fifth left parasternal intercostal space and the beam is directed posteriorly (Fig. 24.35).
- Identify the right ventricle, left atrium, left ventricle, mitral valve, aortic valve, aortic root, aortic outflow tract, and surrounding pericardium (Fig. 24.36).

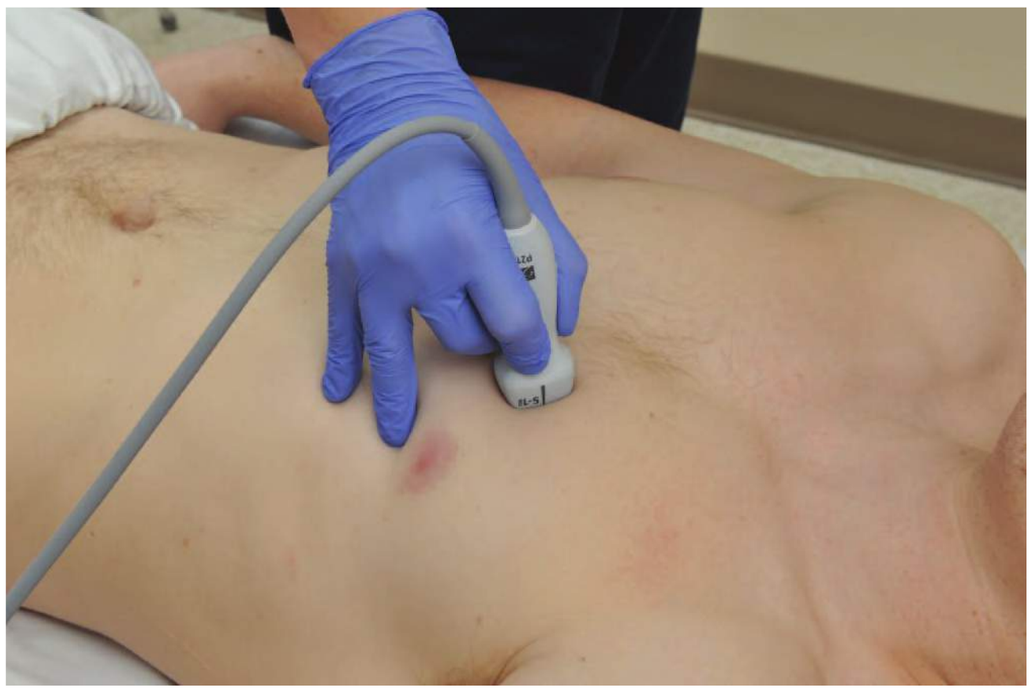


FIGURE 24.35 ■ **Parasternal Long-Axis View.** The transducer is placed in the fourth or fifth left parasternal intercostal space and the beam is directed posteriorly. (Photo contributor: Lawrence B. Stack, MD.)

Abnormal Findings

- Pericardial effusion: Anechoic (black) region noted between the hyperechoic (bright) pericardium and the walls of the heart (see Fig. 24.37). In this view, pericardial effusion may be distinguished from a left-sided pleural effusion by its relationship to the descending thoracic aorta.
- Aortic root dilatation: An aortic root measurement greater than 3.8 to 4.0 cm is abnormal and indicates aneurismal dilatation that may suggest aortic dissection in the appropriate clinical setting. Further evaluation is recommended.
- Dilated descending aorta: The transverse descending thoracic aorta can be seen in the far field in this view posterior to the left atrium. A descending thoracic aorta greater than 4.0 cm is abnormal and indicates aneurismal dilatation that may suggest aortic dissection in the appropriate clinical setting. Further evaluation is recommended.
- Poor left ventricular function: Gross estimates of left ventricular function may be made by observing the anteroposterior motion of the mitral valve, the increase in myocardial thickness with contraction, and the extent to which the anterior and posterior walls of the left ventricle approach each other in systole.

Pearls

1. A true parasternal long-axis view (a sagittal image through the heart) will visualize the aortic root within the image. If the aortic root is not present, you are likely in

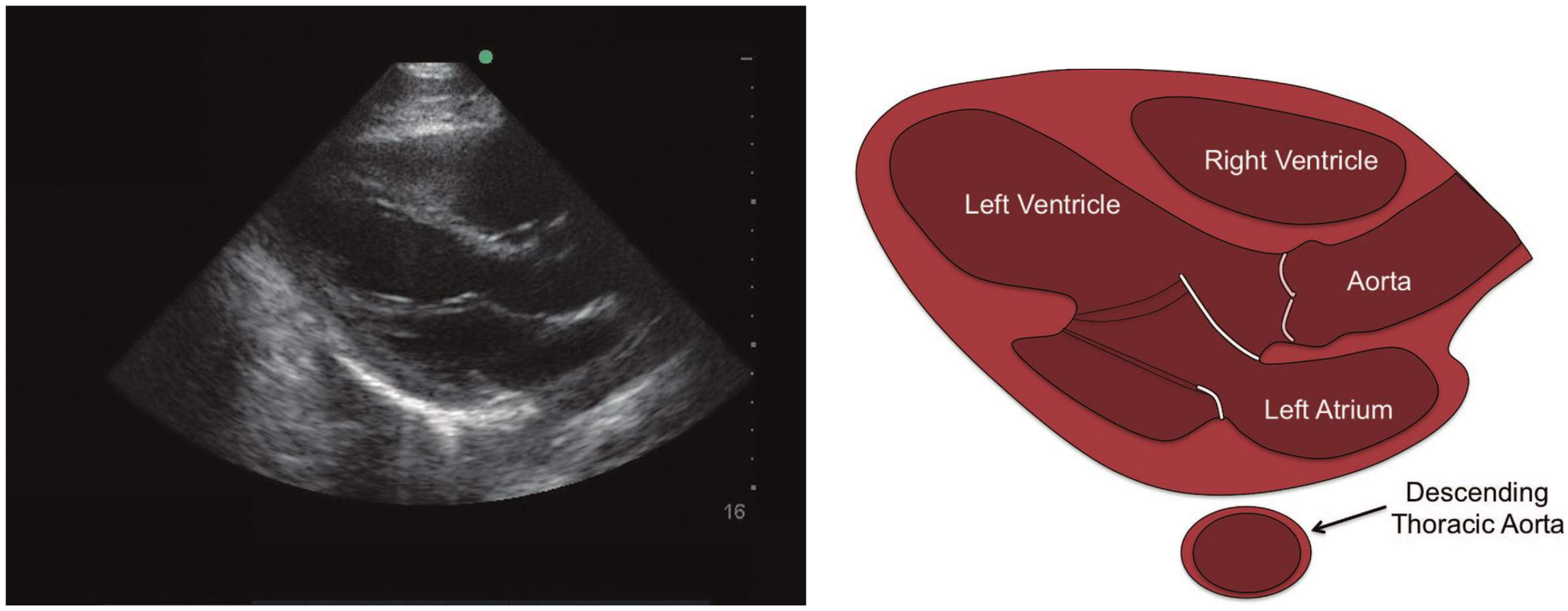


FIGURE 24.36 ■ **Parasternal Long-Axis View.** The right ventricle, left atrium, left ventricle, mitral valve, aortic valve, aortic root, aortic outflow tract, and surrounding pericardium are seen. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Shannon Snyder, MD, RDMS.)

an oblique plane and will need to angle the transducer to optimize the image.

2. It is critical to make deliberate, slow, small adjustments of the transducer in imaging the heart, since even small movements at the skin surface can translate into large changes in beam angle at just 5 to 10 cm deep from the surface.
3. Normal spontaneous respiration is usually fine for cardiac imaging. Patients who are tachypneic can be very challenging, and verbally coaching the patient's breathing patterns is best. If you note a great deal of artifact due to lung interposition, place the patient in the left lateral decubitus position; have him or her inhale and slowly exhale while you scan. When you have an acceptable window, ask the patient to stop exhaling and hold his or her breath while you capture your images.
4. Remember that the parasternal long axis is approximated by a line running from the right acromioclavicular joint and the left antecubital fossa (when the arm is lying by the patient's side).

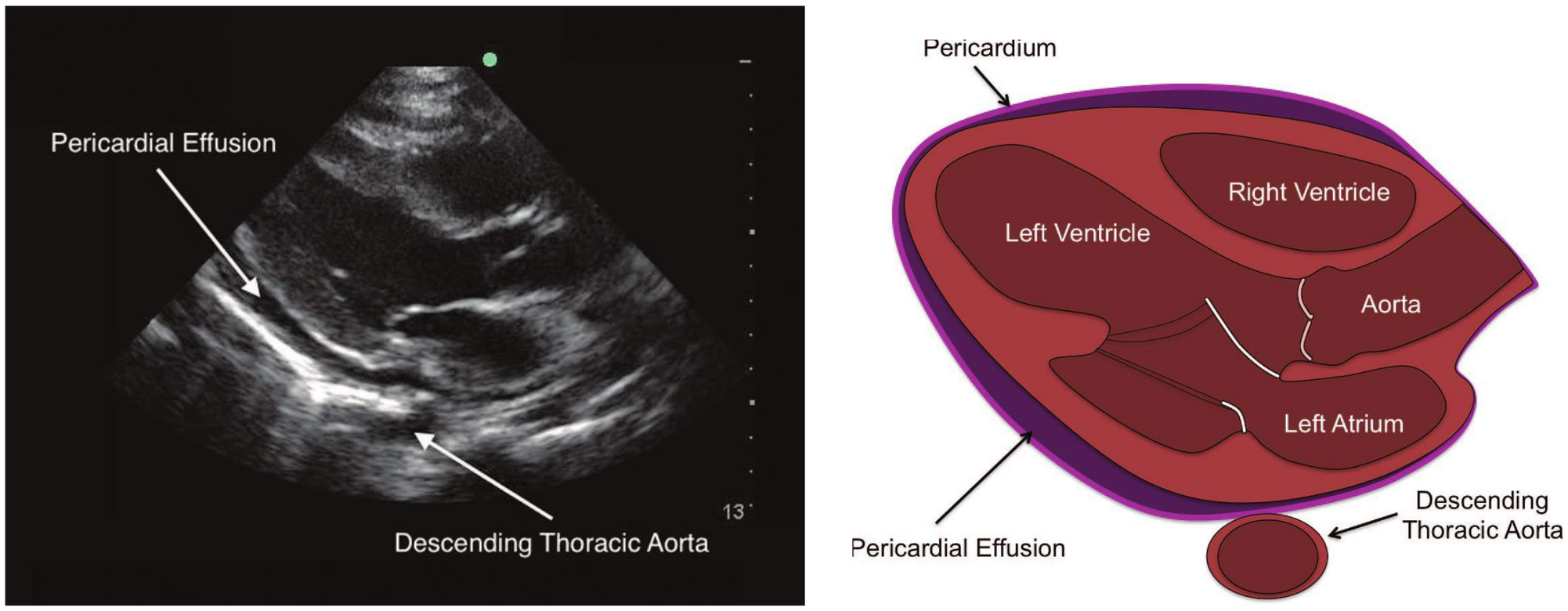


FIGURE 24.37 ■ **Parasternal Long-Axis View.** A pericardial effusion is seen as an anechoic (black) region between the hyperechoic (bright) pericardium and the walls of the heart. The image demonstrates a small pericardial effusion, while the illustration demonstrates the location of a larger (circumferential) effusion. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

PARASTERNAL SHORT-AXIS VIEW (PSSA)

Technique

- From the parasternal long-axis position, rotate the transducer 90 degrees clockwise (to the patient's left) or place the transducer in the fourth or fifth left parasternal intercostal space in a line connecting the left clavicle/shoulder and the right hip (Fig. 24.38).

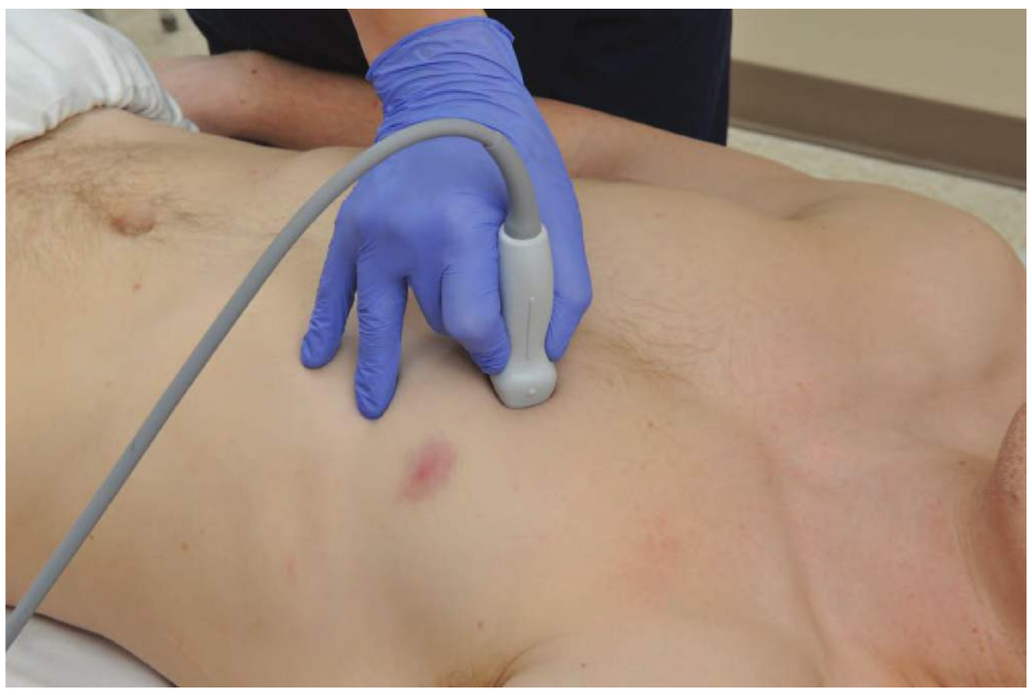


FIGURE 24.38 ■ **Parasternal Short-Axis View.** Place the transducer in the fourth or fifth left parasternal intercostal space in a line connecting the left clavicle/shoulder and the right hip, with the transducer rotated 90 degrees clockwise from the parasternal long-axis view. (Photo contributor: Lawrence B. Stack, MD.)

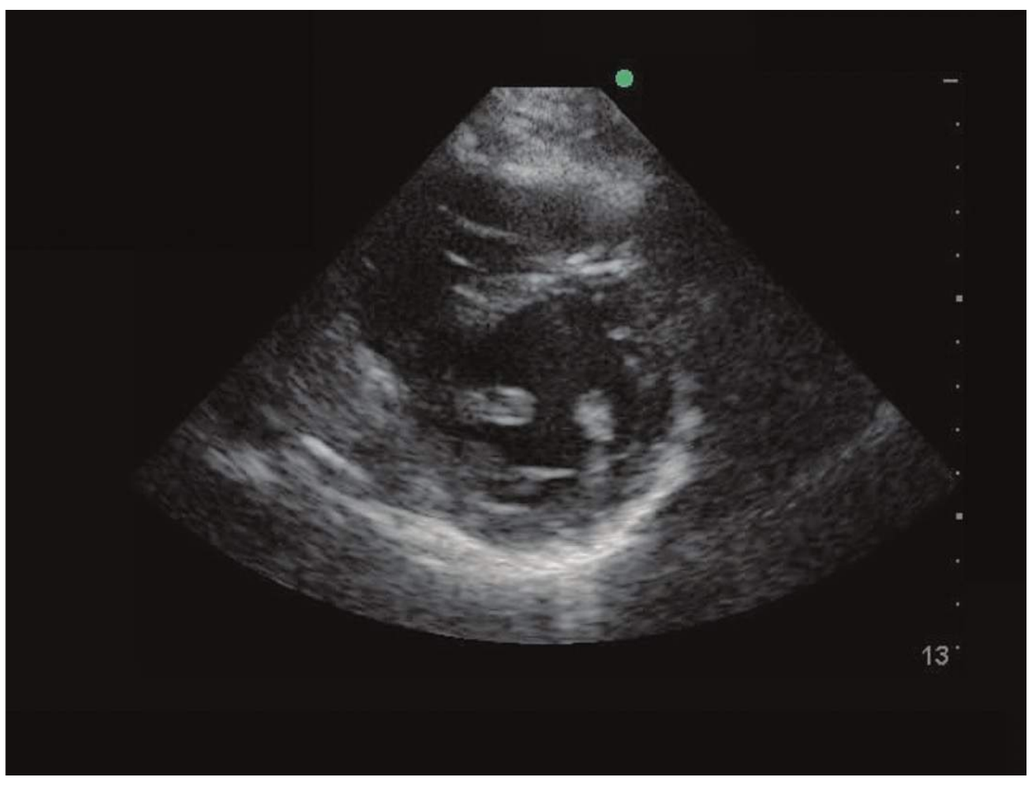
- Identify the left ventricle (circular), right ventricle (crescent-shaped), and surrounding pericardium (Fig. 24.39).

Abnormal Findings

- Pericardial effusion: Anechoic (black) region noted between the bright pericardium and the walls of the heart (Fig. 24.37).
- Dilated right ventricle: The right ventricle is normally a crescent-shaped structure. A rounded, dilated right ventricle that deforms the rounded left ventricle (creating an "OD" sign) suggests elevated right-sided pressures, as seen with pulmonary emboli and severe pulmonary hypertension (Fig. 24.40).

Pearl

1. The standard parasternal short-axis view is obtained with the image plane at the level of the papillary muscles. Visualization of the papillary muscles should ensure a true transverse section through the left ventricle, and provides a prime location for the evaluation of left ventricular contraction and motion.



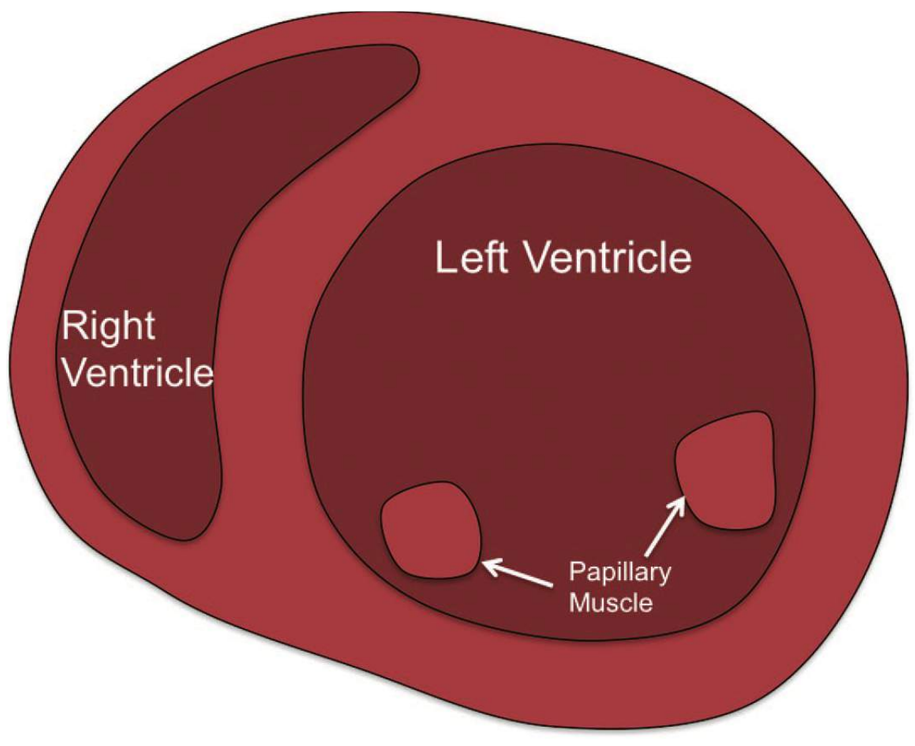
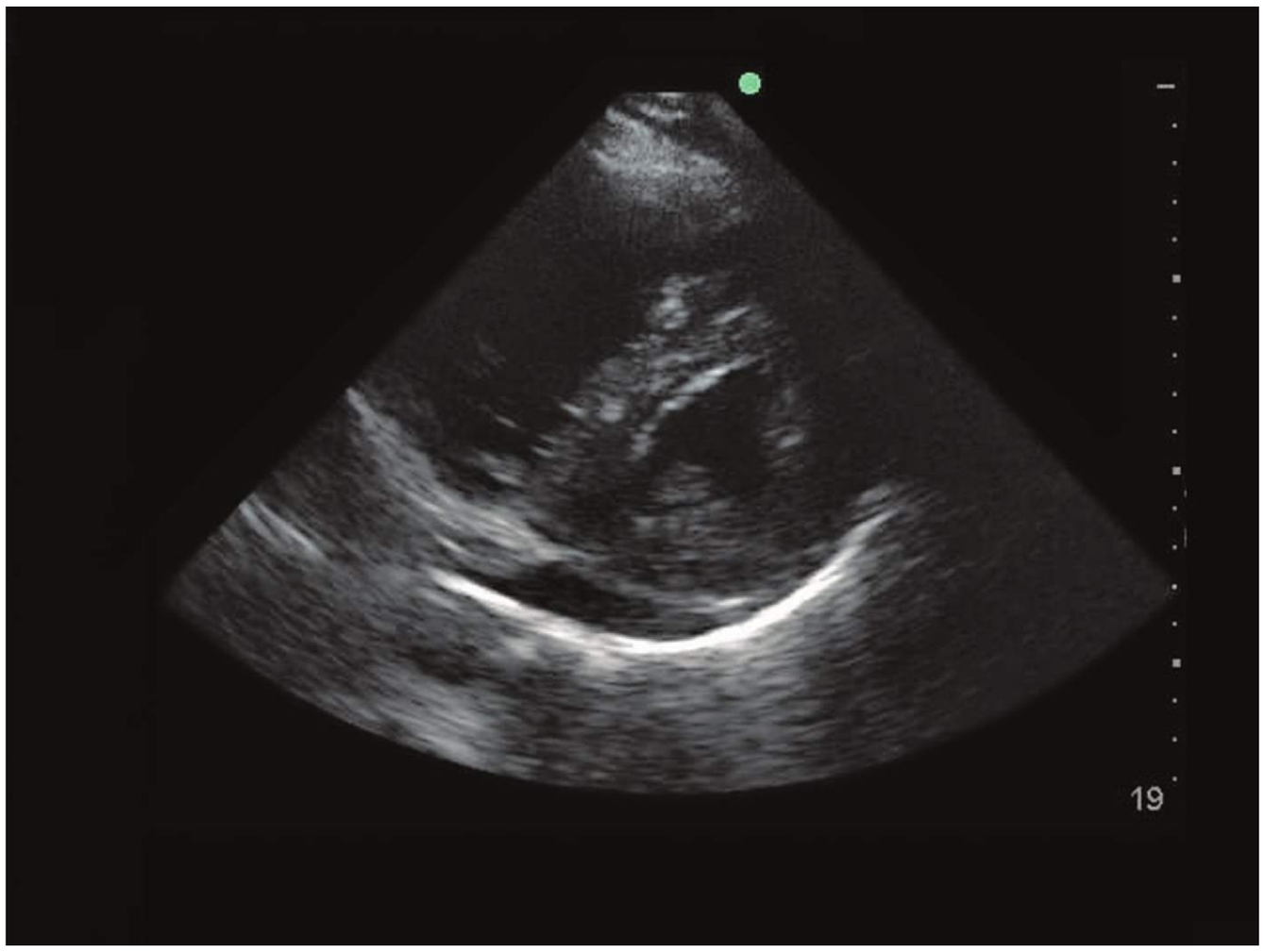


FIGURE 24.39 ■ **Parasternal Short Axis.** The left ventricle (circular) and right ventricle (crescent-shaped) and surrounding pericardium are seen. The papillary muscles are seen in cross-section. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Shannon Snyder, MD, RDMS.)



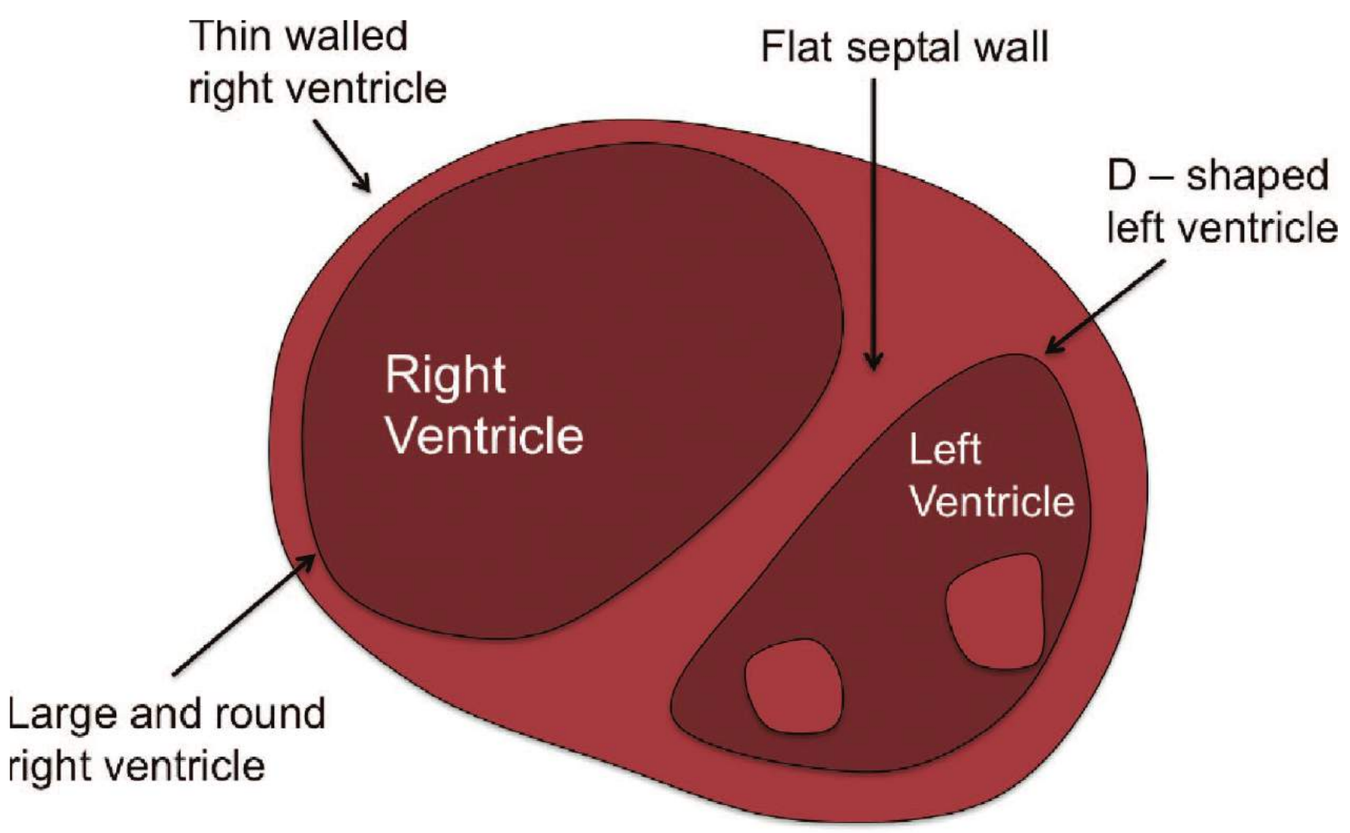


FIGURE 24.40 ■ **Parasternal Short Axis.** A rounded, dilated right ventricle that deforms the rounded left ventricle (creating an "OD" sign) suggests elevated right-sided pressures. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

Technique

- The transducer indicator is directed toward the left axilla or directly posterior to the patient.
- The transducer is placed over the cardiac apex or the point of maximal intensity with the beam directed toward the right clavicle/shoulder in a plane coronal to the heart (Fig. 24.41).
- Identify the left ventricle, right ventricle, left atrium, right atrium, tricuspid valve, mitral valve, and surrounding pericardium (Fig. 24.42).

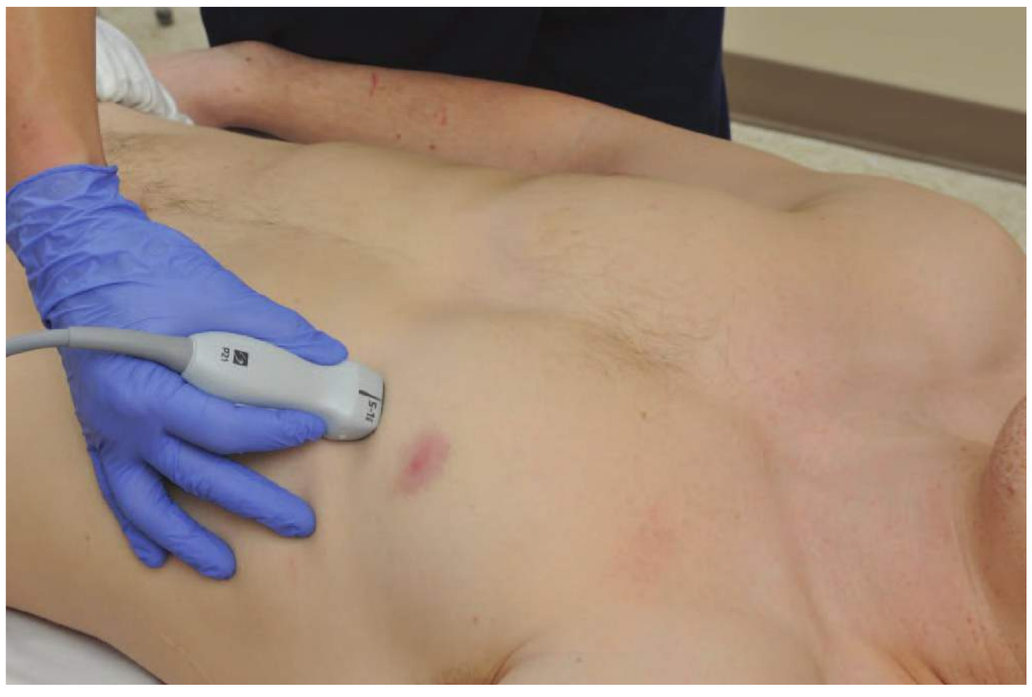


FIGURE 24.41 ■ **Apical Four-Chamber View.** The transducer is placed over the cardiac apex with the beam directed toward the right clavicle/shoulder in a plane coronal to the heart. (Photo contributor: Lawrence B. Stack, MD.)

Abnormal Findings

- Pericardial effusion: Anechoic (black) region noted between the hyperechoic pericardium and the walls of the heart (see Figs. 24.37 and 24.43).
- Dilated right atria/ventricle: If the right ventricle is equal to or larger than the left ventricle in size, or there is poor contractility of the right heart in comparison with the left, it may suggest elevated right-sided pressures as seen with pulmonary emboli and severe pulmonary hypertension (Fig. 24.44).
- Dilated cardiomyopathy: As cardiac remodeling occurs in states of dilated cardiomyopathy, the left ventricle begins to take on more of a dilated balloon-like shape rather than its normal more streamlined bullet-like shape (Fig 24.45).

Pearls

1. See "Pearls for the FAST Examination, Subxiphoid-Cardiac," item 4, above.
2. It is critical to realize the variance in the resting position of the heart. There can be significant differences in acoustic windows from patient to patient with the four-chamber apical view.



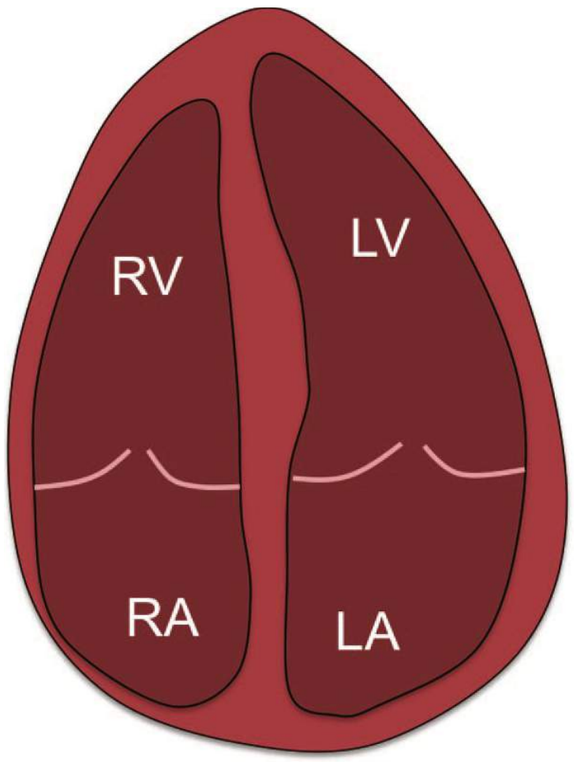


FIGURE 24.42 ■ **Apical Four-Chamber View.** The left ventricle, right ventricle, left atrium, right atrium, tricuspid, and mitral valves are seen. Ultrasound contributor: Stephen J. Leech, MD, RDMS; illustration courtesy of Robinson M. Ferre, MD.)

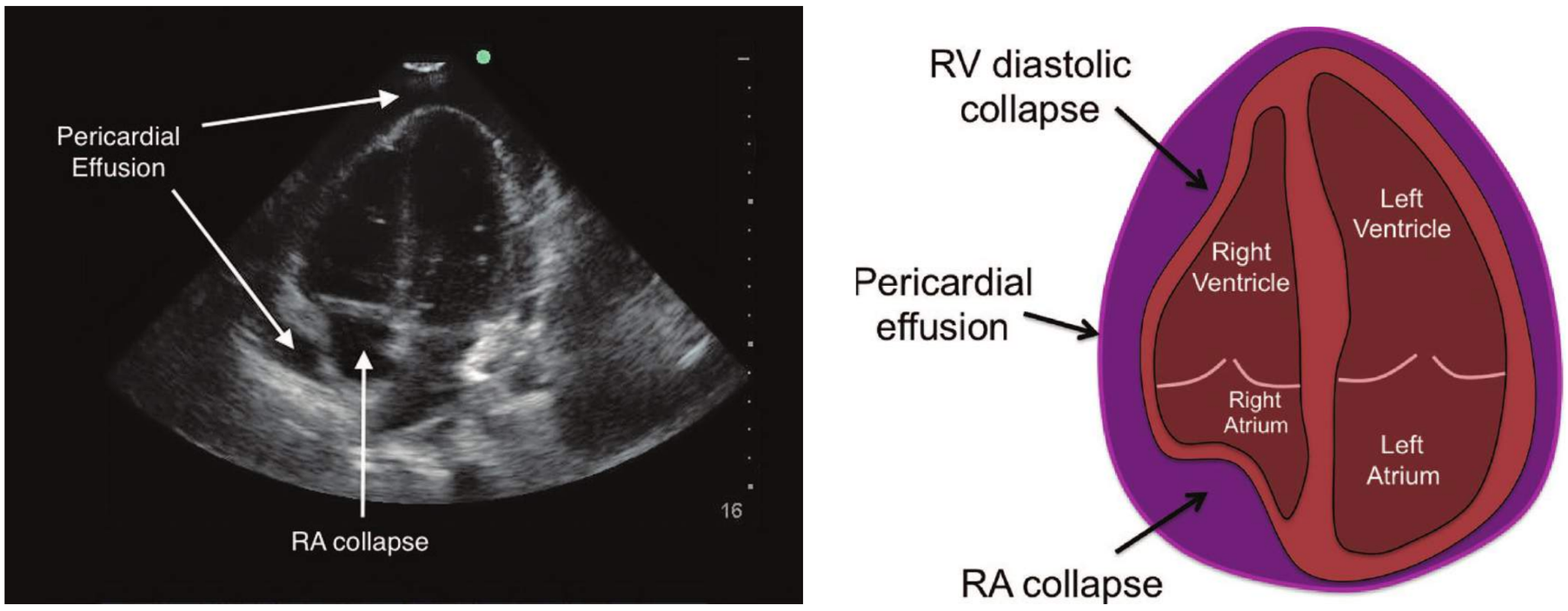


FIGURE 24.43 ■ **Apical Four-Chamber View.** A pericardial effusion appears as an anechoic (black) region between the hyperechoic pericardium and the walls of the heart. When tamponade physiology is present, right atrial and right ventricular collapse may be seen. Right atrial collapse is seen in the ultrasound image. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

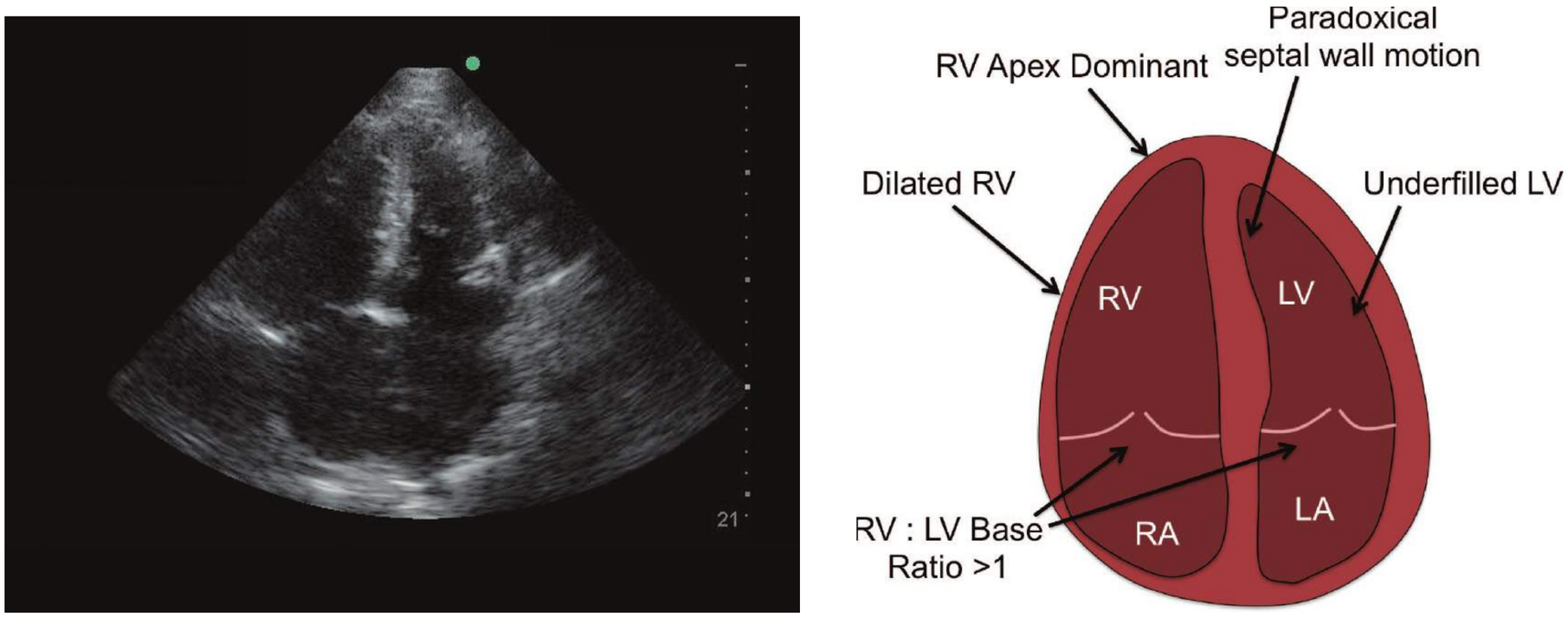


FIGURE 24.44 ■ **Apical Four-Chamber View.** A right ventricle that is equal in size or larger than the left ventricle suggests elevated right-sided pressures. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Robinson M. Ferre, MD.)

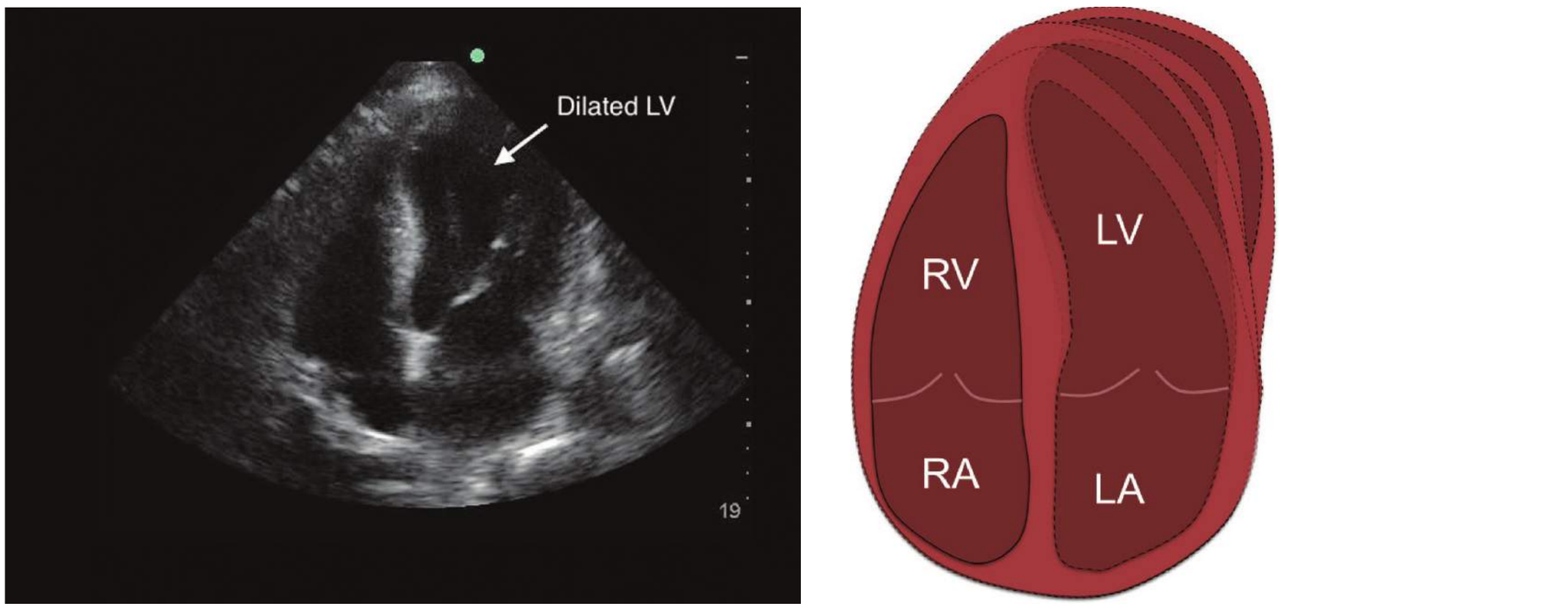


FIGURE 24.45 ■ **Apical Four-Chamber View.** As cardiac remodeling occurs in states of dilated cardiomyopathy, the left ventricle begins to take on more of a dilated balloon-like shape rather than its normal streamlined bullet-like shape. The diagram shows the progression of remodeling. (Ultrasound contributor: Jeremy S. Boyd, MD; illustration courtesy of Robinson M. Ferre, MD.)

Technique

- The transducer indicator is directed toward the patient's feet.
- The transducer is placed subxiphoid in a midline plane and tilted toward the patient's right to identify the IVC as it enters the right atrium (Fig. 24.46).
- Identify the liver, IVC, right atrium, and right hepatic vein (Fig. 24.47).
- Measure the IVC 2 to 3 cm distal to the entry point to the right atrium. Measure the IVC through the respiratory cycle at end inspiration and expiration (Fig. 24.48).

Abnormal Findings

- IVC collapse: The IVC should partially collapse during the normal respiratory cycle in nonventilated patients. A totally collapsed IVC is indicative of low central venous pressure (CVP). More than 50% collapse is less specific but also indicative of low CVP (Fig. 24.49).
- IVC size: The size of the IVC may be measured throughout the respiratory cycle. In general, an IVC that measures greater than 2 cm or greater with poor respiratory variation is indicative of elevated CVPs due to volume overload, RV failure, and/or tamponade physiology (Fig. 24.50A,B).
- IVC distensibility: In patients receiving positive pressure ventilation, the IVC will distend rather than collapse during inspiration. Measurements of IVC distensibility in ventilated patients can be used to calculate volume responsiveness to intravenous fluid challenge.

Pearls

1. Consider using M-mode through the IVC to aid in measurement for patients with high respiratory rates.
2. Some patients require a low anterior intercostal view to image the IVC as it enters the right atrium.

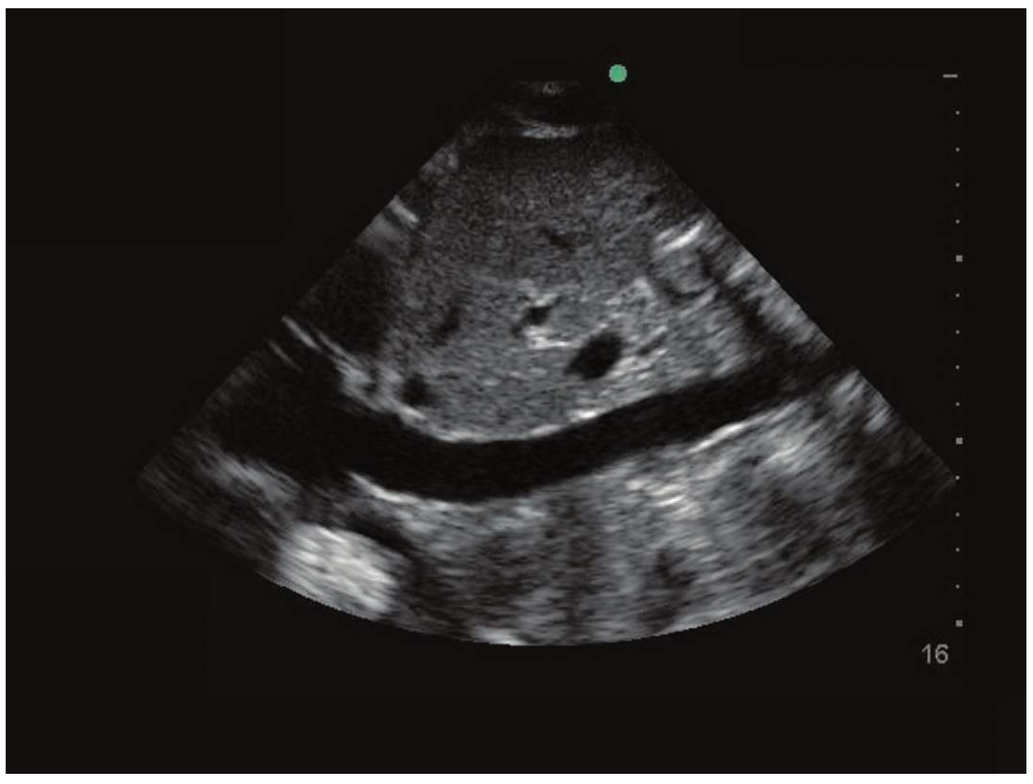


FIGURE 24.47 ■ **IVC View.** The liver is seen with the IVC entering the right atrium on the left. Probe marker (on right of the screen) is directed inferiorly here. (Ultrasound contributor: Jeremy S. Boyd, MD.)



FIGURE 24.46 ■ **IVC View.** The transducer is placed subxiphoid in a midline plane and tilted toward the patient's right to identify the IVC as it enters the right atrium. (Photo contributor: Lawrence B. Stack, MD.)

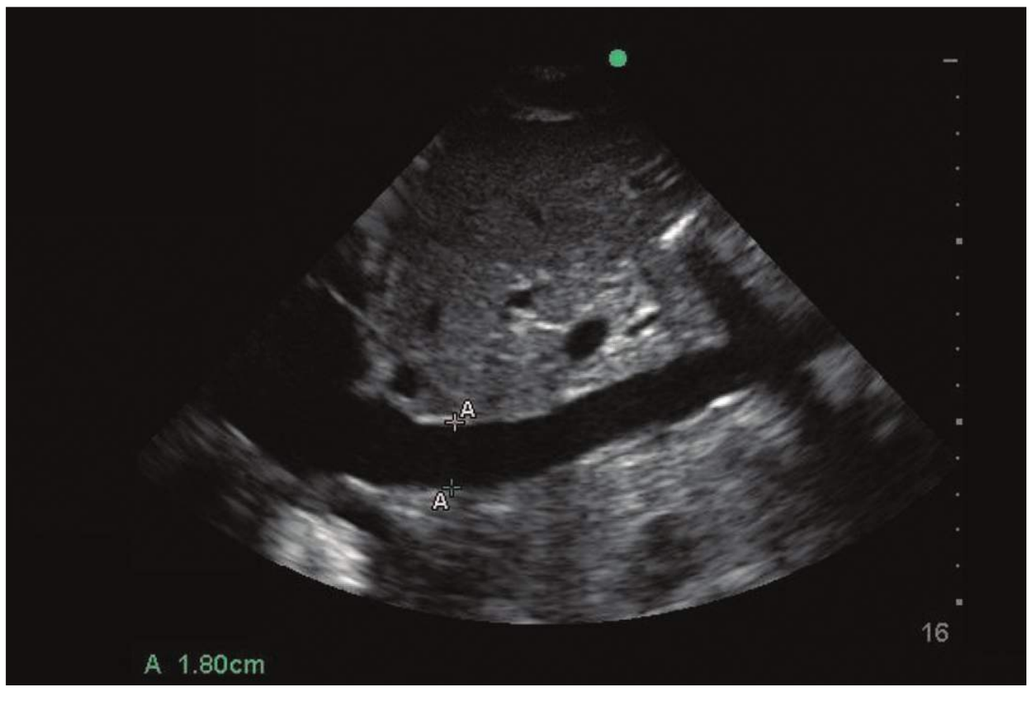


FIGURE 24.48 ■ **IVC View.** Measure the IVC 2-3 cm distal to the entry point to the RA. 1.5-2.5 cm is considered a normal IVC diameter. Greater than 2.5 cm is considered an enlarged or "plethoric" IVC. (Ultrasound contributor: Jeremy S. Boyd, MD.)

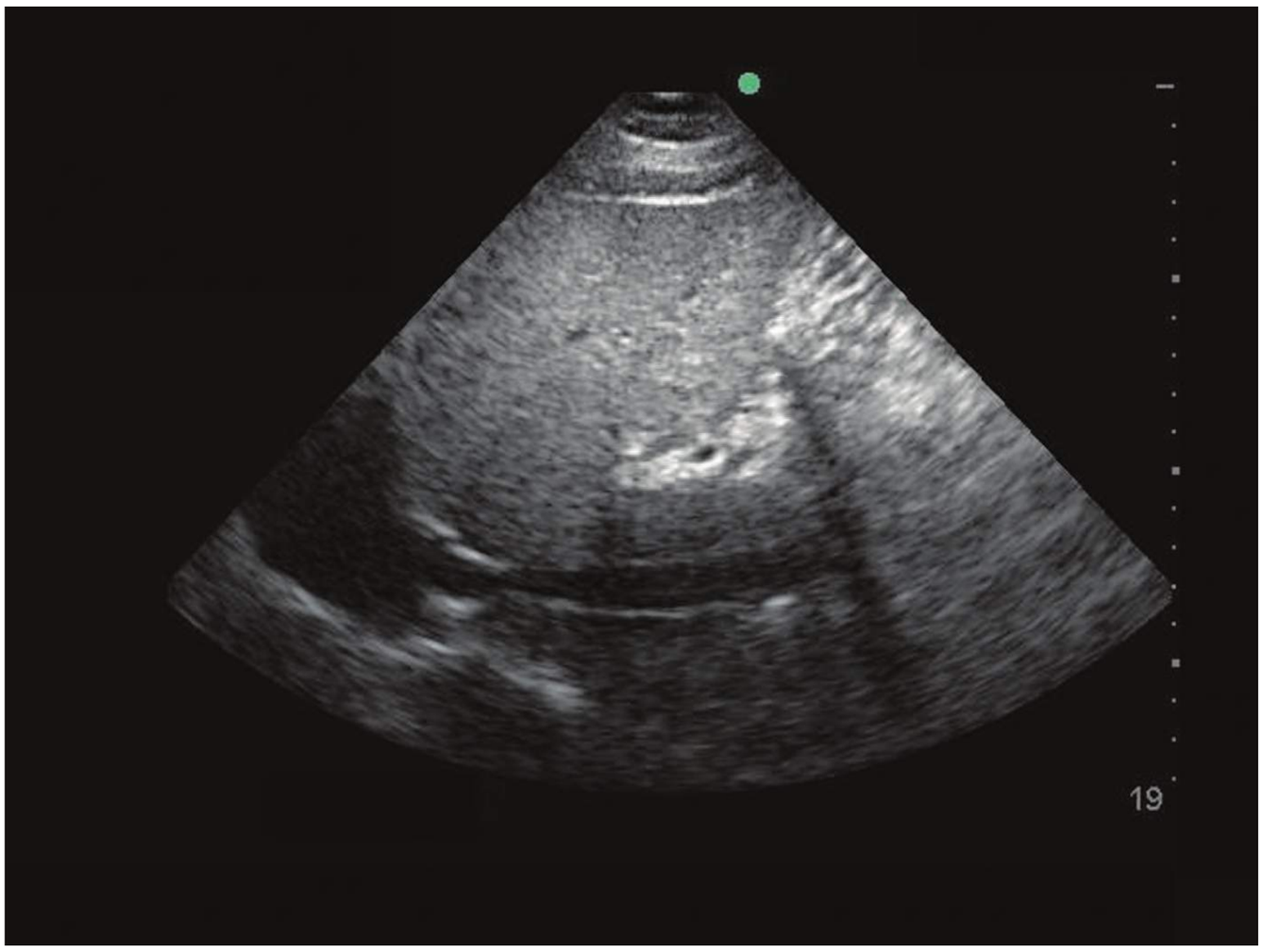


FIGURE 24.49 ■ **IVC View.** A totally collapsed IVC is indicative of low CVP. (Ultrasound contributor: Jeremy S. Boyd, MD.)

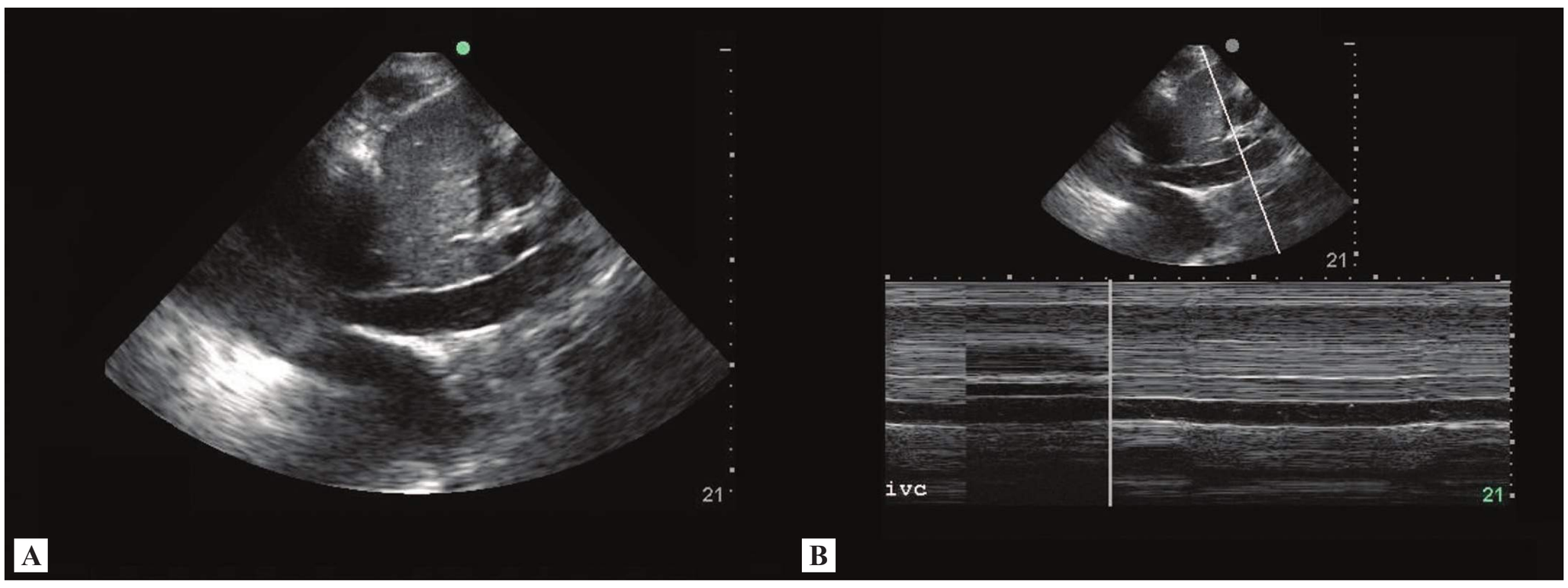


FIGURE 24.50A, B ■ **IVC View.** An IVC with poor respiratory variation (demonstrated in M-mode) is indicative of elevated central venous pressures due to volume overload, right ventricular failure, and/or tamponade physiology. (Ultrasound contributor: Jim Fiechtl, MD.)

Clinical Summary

Aerated lungs often interfere with sonographic visualization of the organs deep to them, such as often occurs in cardiac and biliary exams. This is due to the scattering effect of gas molecules on the ultrasound beam. For this reason, ultrasound of the lungs themselves was long thought to be of no diagnostic use. However, in recent years researchers and clinicians have discovered that lung ultrasound can be used to assess for pathologic conditions that occur at the interface of the lung and the chest wall such as pneumothorax, pulmonary edema, and pneumonia. The evaluation of the pleura and the finding of lung sliding to evaluate for pneumothorax was described earlier in the section on trauma ultrasound. Contemporary point-of-care lung ultrasound often evaluates for the presence of alveolar interstitial syndrome which occurs with the collection of fluid within the interlobular and intralobular lymphatics of the lung. This occurs in various pathologic states including cardiogenic pulmonary edema, acute respiratory distress syndrome, pneumonia, and diffuse parenchymal lung disease. Lung ultrasound can also effectively be used to visualize lung consolidation and pleural effusions.

Indications

- Shortness of breath and acute dyspnea
- Hypoxia
- Pleuritic chest pain
- Congestive heart failure
- Chest trauma

Required Views

- For comprehensive evaluation, current consensus recommendations include scanning the thorax in eight separate zones including the costophrenic angles of each chest (Fig. 24.51). Each hemithorax is divided into four different zones as shown in the figure.
- A more focused lung exam can be performed depending on the pathology suspected. For example, if assessing for a pneumothorax following a subclavian central venous line, anterior views along the midclavicular line would be sufficient.

Recommended Transducers

- Convex array (anterior and costophrenic angles)
- Phased array (costophrenic angles; poor for assessing lung sliding)
- Linear array (anterior chest only)

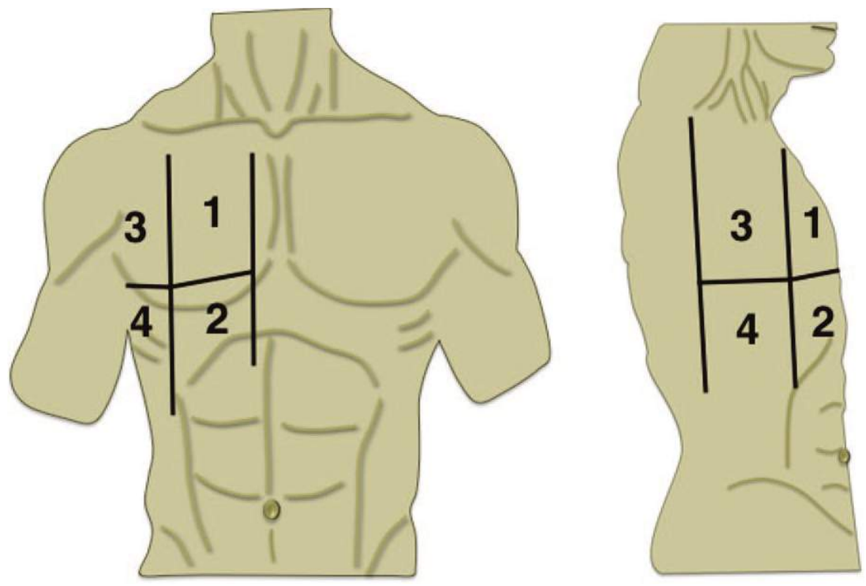


FIGURE 24.51 ■ **Lung Zones.** Current consensus recommendations include scanning the thorax in eight separate zones including the costophrenic angles of each chest. (Illustration courtesy of Robinson M. Ferre, MD.)

Patient Position

- The patient is traditionally imaged in a supine position, which is necessary if assessing for a pneumothorax.
- If assessing for conditions other than pneumothorax, a supine, semi-recumbent, or upright position may be used.

Technique

- To assess for alveolar interstitial syndrome or consolidation: With the indicator pointed toward the patient's head, each of the four zones of each hemithorax should be imaged. The depth on the screen should be between 13 and 15 cm deep. Each zone should be imaged thoroughly for evidence of A-lines, B-lines, or consolidation.
- To assess for pleural fluid: The technique for evaluating costophrenic angles for pleural effusion is identical to that employed in the E-FAST when evaluating for hemothorax.
- To assess for a pneumothorax: The technique for evaluating costophrenic angles for pleural effusion is identical to that employed in the E-FAST when evaluating for hemothorax.

Findings

- A-lines: Normal aerated lung is indicated by the presence of "A-lines" and lung sliding. A-lines are echogenic horizontal lines deep and parallel to the pleural line that occur at regular intervals and do not move with respiration or lung sliding. (Fig. 24.52)
- B-Lines: The presence of "B-lines" indicates alveolar interstitial syndrome. B-lines are defined as laser-like

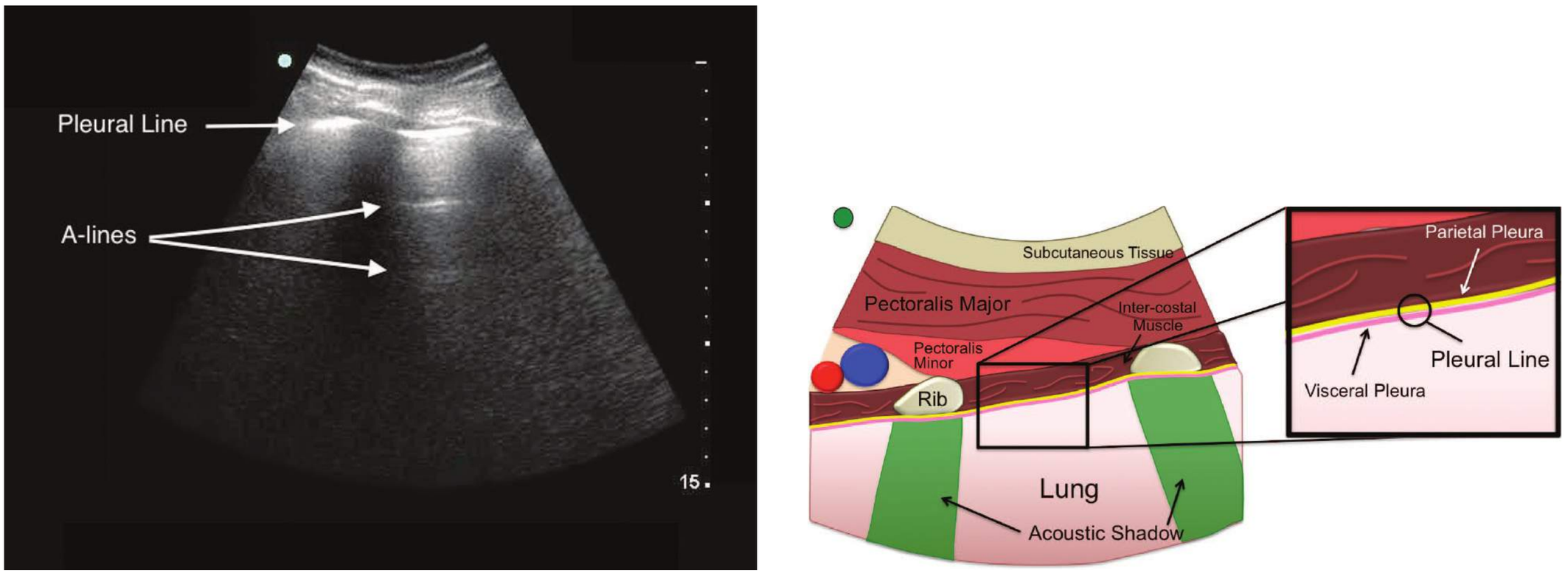


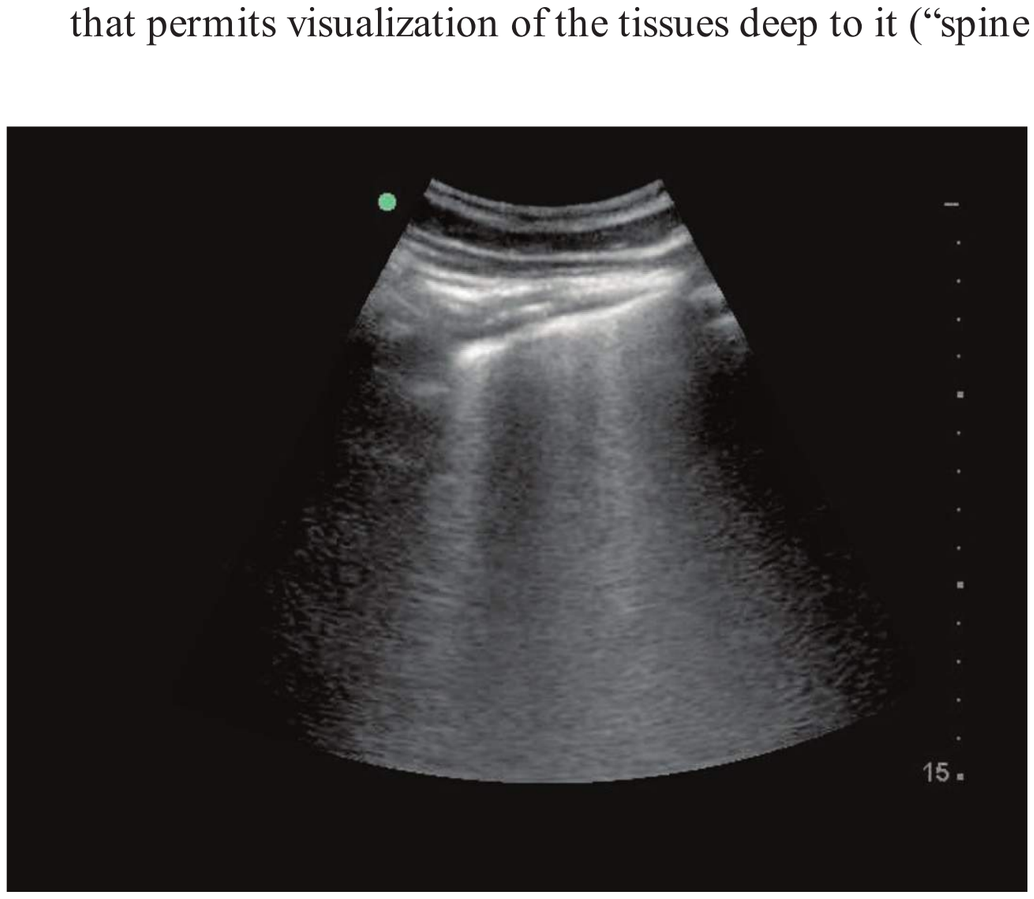
FIGURE 24.52 ■ **Lung Ultrasound: A-Lines.** Normal aerated lung is indicated by the presence of "A-lines" and lung sliding. A-lines are echogenic horizontal lines deep and parallel to the pleural line that occur at regular intervals and do not move with respiration. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

artifacts that begin from the pleural line and extend to the bottom of the screen without fading and move in conjunction with respiration or lung sliding (Fig. 24.53).

- Consolidation: Consolidation of lung tissue appears as a subpleural area of tissue-like echotexture. It may have comet-tail artifacts extending from the far-field border, a pattern called the "shred sign." Air and/or fluid bronchograms may also be noted within the consolidation (Fig. 24.54).
- Pleural effusion: A hypoechoic area located in the diaphragmatic recess superior/cephalad to the diaphragm that permits visualization of the tissues deep to it ("spine sign") is a pleural effusion. Consolidated lung can often be seen moving within the effusion in synchrony with the patient's respirations (Fig. 24.55).

Pearls

- The distribution of B-lines, whether diffuse, focal, unilateral, or bilateral, in conjunction with the clinical exam and history can help to determine the etiology of the patient's symptoms. Cardiogenic pulmonary edema will be found diffusely and bilaterally, whereas a focal pneumonia or



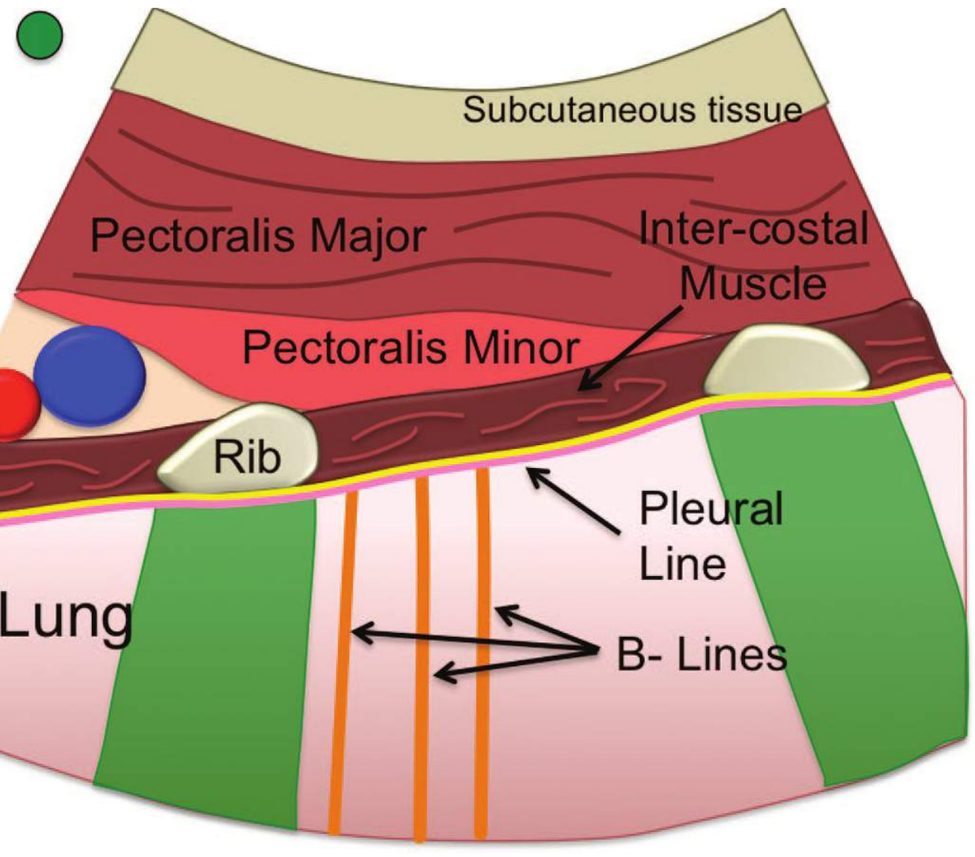


FIGURE 24.53 ■ **Lung Ultrasound: B-Lines.** The presence of "B-lines" indicates alveolar interstitial syndrome. B-lines are defined as laser-like artifacts that begin from the pleural line and extend to the bottom of the screen without fading and move in conjunction with respiration. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jordan Rupp, MD.)

pulmonary contusion may only present within a focal region of the ultrasound lung exam.

- In patients with cardiogenic pulmonary edema, the number and frequency of B-lines correlate with the severity of edema and can also be used to monitor the response to therapy.
- When performing an E-FAST exam, the linear probe is frequently used as it is most sensitive for lung sliding and visualization of the pleura. When using a different probe to evaluate the lungs, adjust the depth to a shallow setting to ensure adequate evaluation of the pleura for lung sliding. The remainder of the exam should be performed with a depth of at least 13 cm when assessing for B-lines and consolidation.

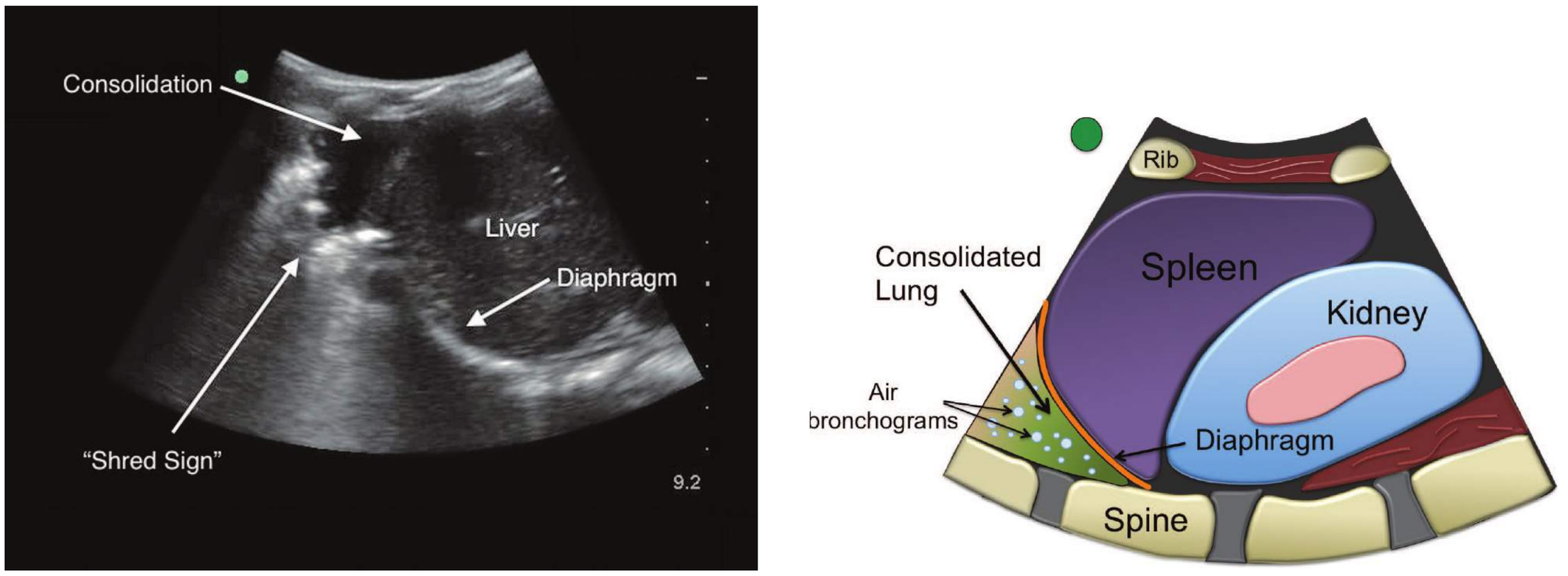


FIGURE 24.54 ■ **Lung Ultrasound**. Consolidation of lung tissue appears as a subpleural area of tissue-like echotexture. It may have comet-tail artifacts extending from the far field border, a pattern called the "shred sign," seen in this image of a pneumonia. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

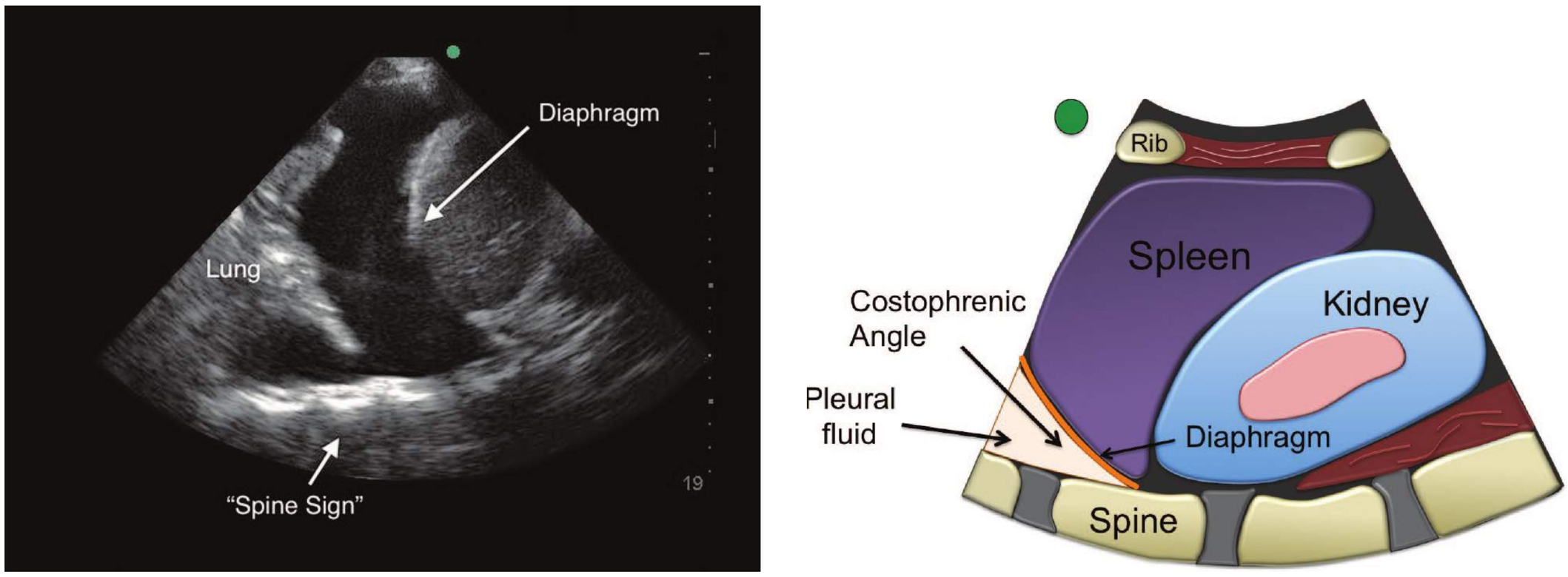


FIGURE 24.55 ■ **Lung Ultrasound.** A pleural effusion is seen as a hypoechoic area located in the diaphragmatic recess superior/cephalad to the diaphragm with the spine sign seen extending beyond the diaphragm into the thorax. Consolidated lung is seen within the effusion. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Jeremy S. Boyd, MD.)

ABDOMINAL AORTA ULTRASOUND

Clinical Summary

EUS of the abdominal aorta is used to diagnose or exclude an abdominal aortic aneurysm (AAA). Though the sensitivity of abdominal ultrasound for the detection of aortic dissection is limited, the presence of an intra-aortic flap is diagnostic for aortic dissection.

Indications

- Abdominal, back, or flank pain
- Pulsatile abdominal mass
- Undifferentiated hypotension

When a patient is unstable, there is no bedside test superior to an EUS of the abdominal aorta to diagnose an AAA. Early diagnosis can improve patient survival. An abdominal aortic measurement greater than 3 cm in diameter is abnormal and diagnostic of an AAA. While an AAA may rupture at any size, the risk of rupture is much greater starting with measurements greater than 5 cm. AAAs occur as both fusiform (more common) and saccular types. It is essential to image the aorta in both sagittal and transverse planes.

Views for Emergency Department Abdominal Aortic Ultrasound

1. Transverse view
2. Sagittal view

Equipment: Recommended Transducer for Abdominal Aortic Ultrasound

- Convex array

Patient Position

The patient is supine.

Techniques

Transverse View

- Place the transducer in the epigastrium with the transducer indicator oriented to the patient's right (Fig. 24.56).
- Identify the acoustic shadow caused by the vertebral body. The abdominal aorta is the circular, pulsating, anechoic structure anterior and slightly to the patient's left.
- Identify and measure the aorta at the proximal (at or above the origin of the superior mesenteric artery [SMA]), middle (near the renal arteries), and distal (immediately proximal to the bifurcation) in the anteroposterior plane (Figs. 24.57 and 24.58).
- Identify the aorta in relation to the IVC, SMA, and splenic vein.
- Move down the abdominal aorta to the bifurcation (approximately the level of the umbilicus) (Fig. 24.59).

Sagittal View

- Place the transducer in the epigastrium with the transducer indicator oriented toward the patient's head. Move down the abdominal aorta to the bifurcation (Fig. 24.60).
- Identify the liver, aorta, IVC, celiac trunk, and SMA (Fig. 24.61).



FIGURE 24.56 ■ **Abdominal Aorta Transverse View.** Place the transducer in the epigastrium with the transducer indicator oriented to the patient's right. (Photo contributor: Lawrence B. Stack, MD.)

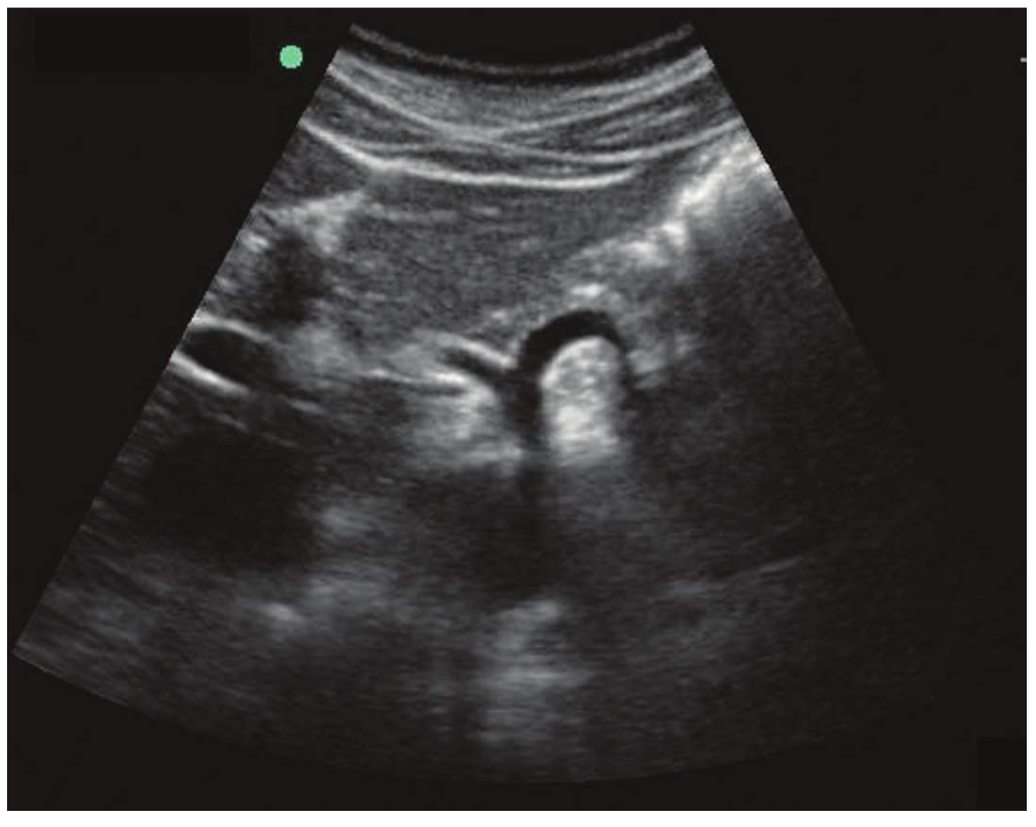


FIGURE 24.57 ■ **Abdominal Aorta: Transverse View.** Short-axis view of the proximal aorta showing the celiac artery and its branches. (Ultrasound contributor: Jeremy Boyd, MD.)

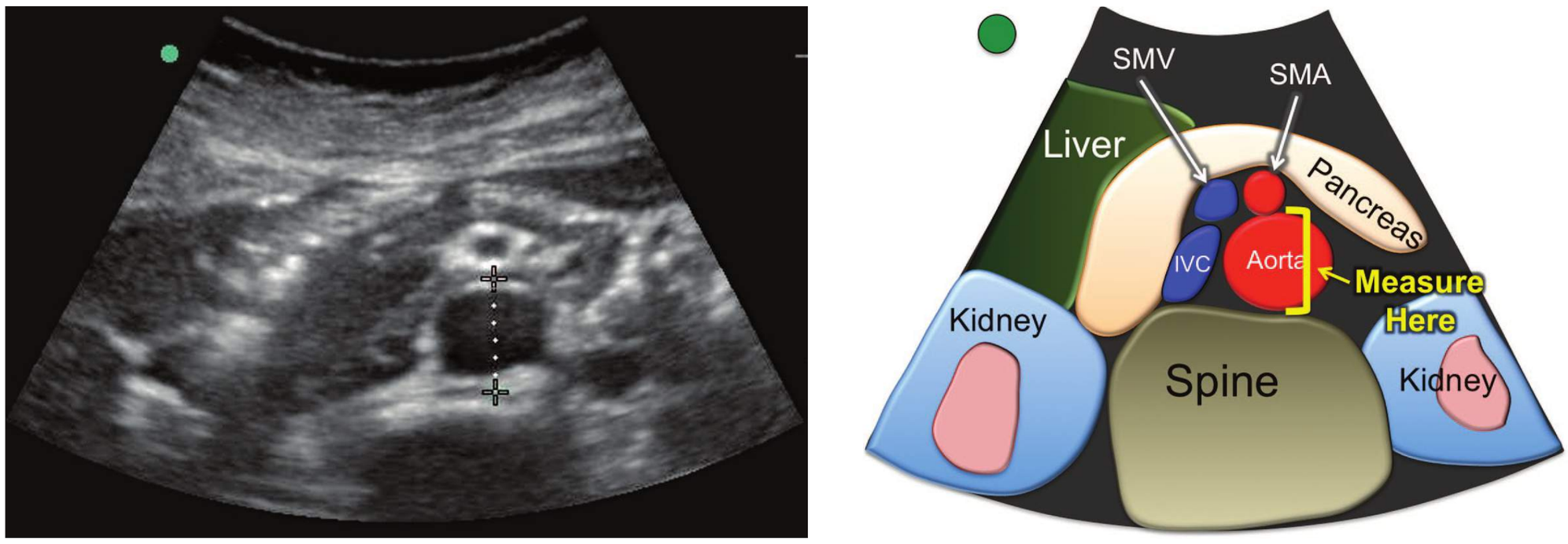


FIGURE 24.58 ■ **Abdominal Aorta: Transverse View.** The proximal aorta, superior mesenteric artery (SMA), and the surrounding anatomy are often compared to a mantle clock. (Ultrasound contributor: Eric Wu, MD; illustration courtesy of Robinson M. Ferre, MD.)

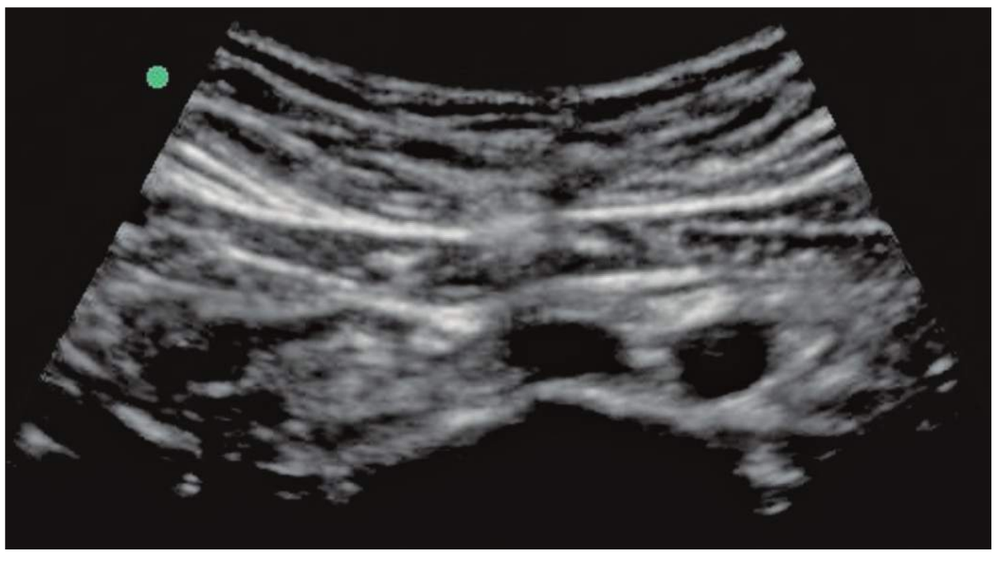
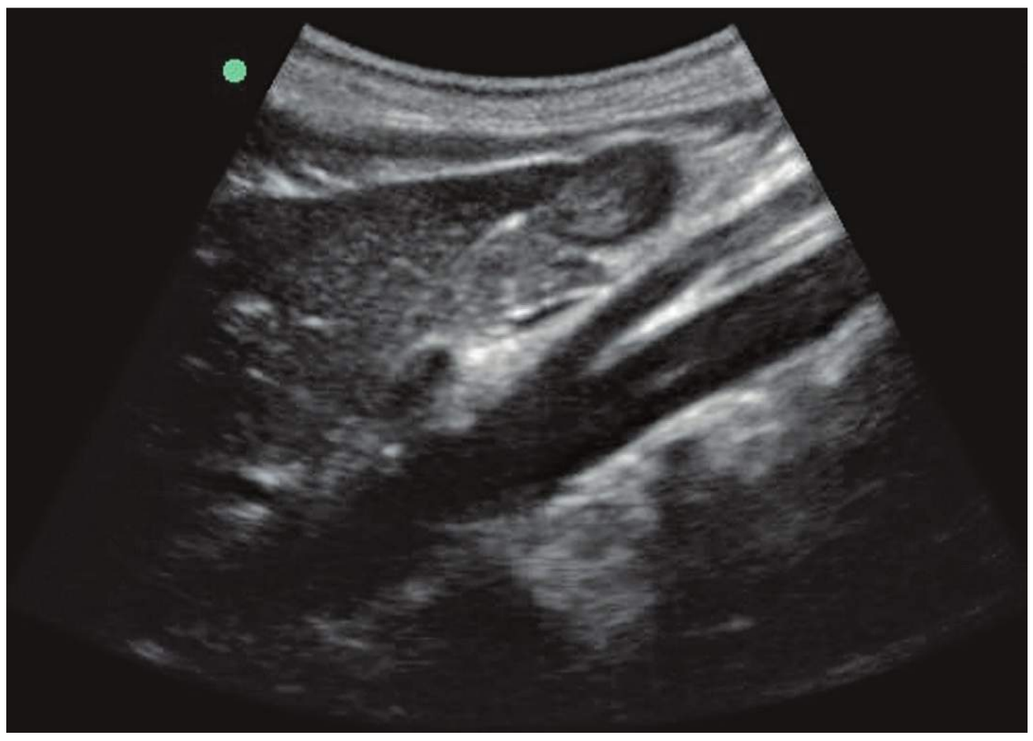


FIGURE 24.59 ■ **Abdominal Aorta: Transverse View.** Still image of the left and right iliac arteries just distal to the bifurcation of the aorta. (Ultrasound contributor: Frances Cheng, MD.)



FIGURE 24.60 ■ **Abdominal Aorta Sagittal View.** Place the transducer in the epigastrium with the transducer indicator oriented toward the patient's head. (Photo contributor: Lawrence B. Stack, MD.)



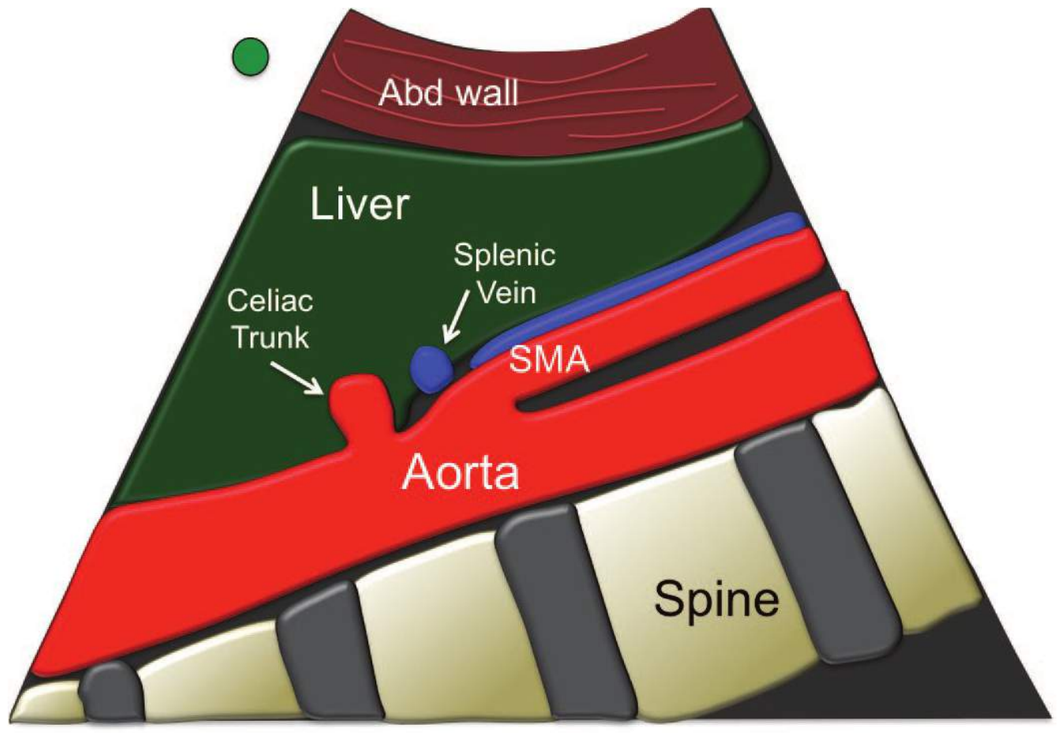


FIGURE 24.61 ■ **Abdominal Aorta.** Sagittal view, proximal aorta. (Ultrasound contributor: Jeremy Boyd, MD, RDMS; illustration courtesy of Robinson M. Ferre, MD.)

Abnormal Findings

- AAA: The abdominal aorta is aneurysmal if at any point it measures more than 3 cm. The aorta can also be aneurysmal if a distal segment measures more than 50% of a proximal segment (Fig. 24.62).
- Aortic dissection: The echogenic line of an aortic "flap" can be seen inside the lumen of the abdominal aorta when present (often moving with pulsations) (Fig. 24.63).

Pearls

1. Starting the exam in the transverse view will help orient the scanner to the anatomy.
2. Measure the entire diameter (outer wall to outer wall) of the aorta. Measuring the lumen is grossly inaccurate and will underestimate the size of an AAA when a clot is present (Figs. 24.64 and 24.65). Include measurements of the proximal, mid, and distal aorta.
3. If a significant amount of bowel gas is present, sit the patient at 45 degrees and apply constant gentle pressure.
4. The IVC may be confused for the aorta, especially in sagittal views. The "sniff test"—having the patient abruptly sniff—will cause the IVC to collapse as a result of the negative pressure transmitted to the venous system during this maneuver.
5. If spectral or color flow Doppler is available, it may be used to discriminate between the highly pulsatile flow of the aorta and the low amplitude of the IVC.
6. In patients with an aortic dissection, an intimal flap can be visible as a linear structure within the aorta. While this finding is highly specific, it is insensitive and its absence cannot be used to rule out a dissection (Fig. 24.63).

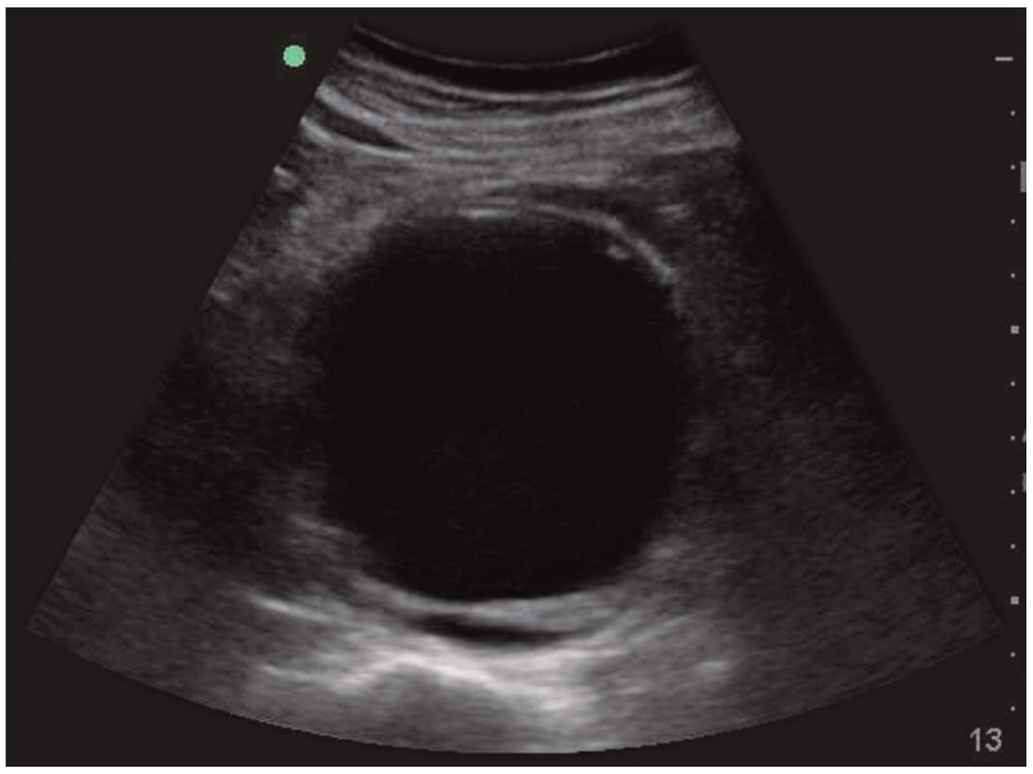


FIGURE 24.62 ■ **Abdominal Aorta: Transverse view, AAA.** Large proximal abdominal aortic aneurysm. (Ultrasound contributor: Lara Philips, MD.)

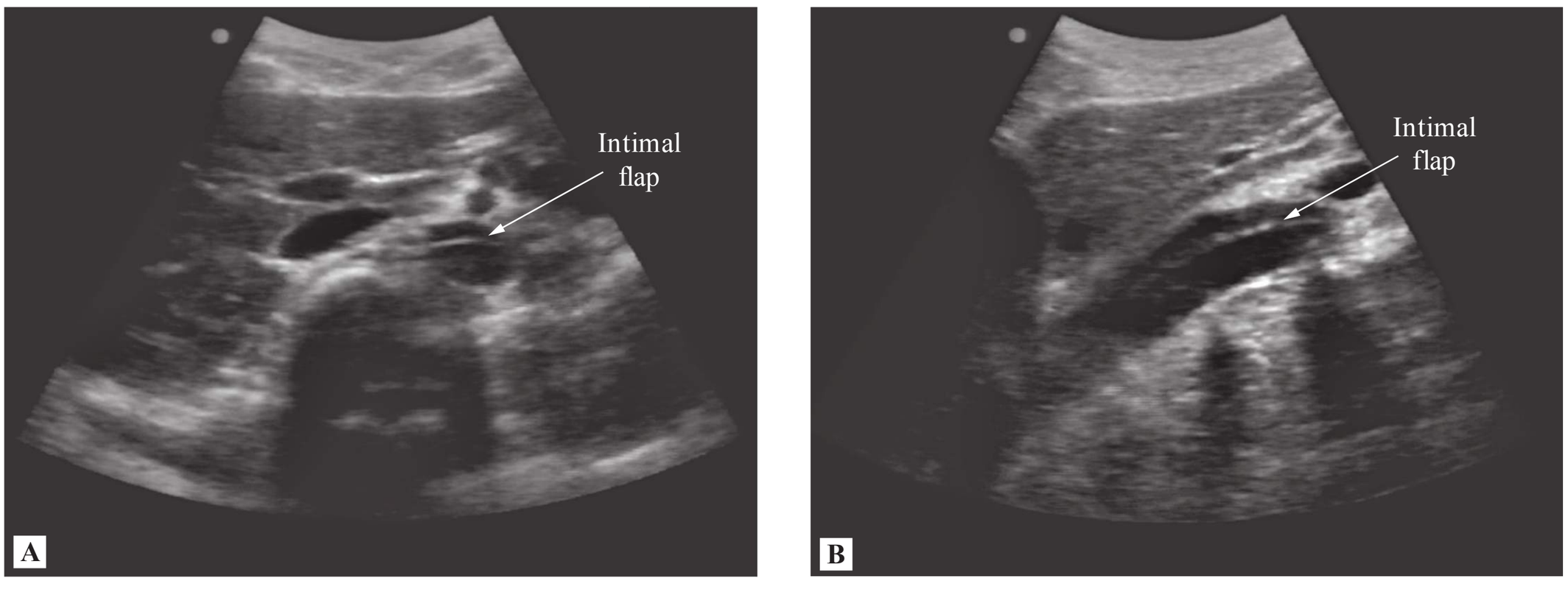


FIGURE 24.63 ■ **Abdominal Aorta: Dissection.** An aortic dissection flap is seen. (Ultrasound contributor: Paul R. Sierzenski, MD, RDMS.)

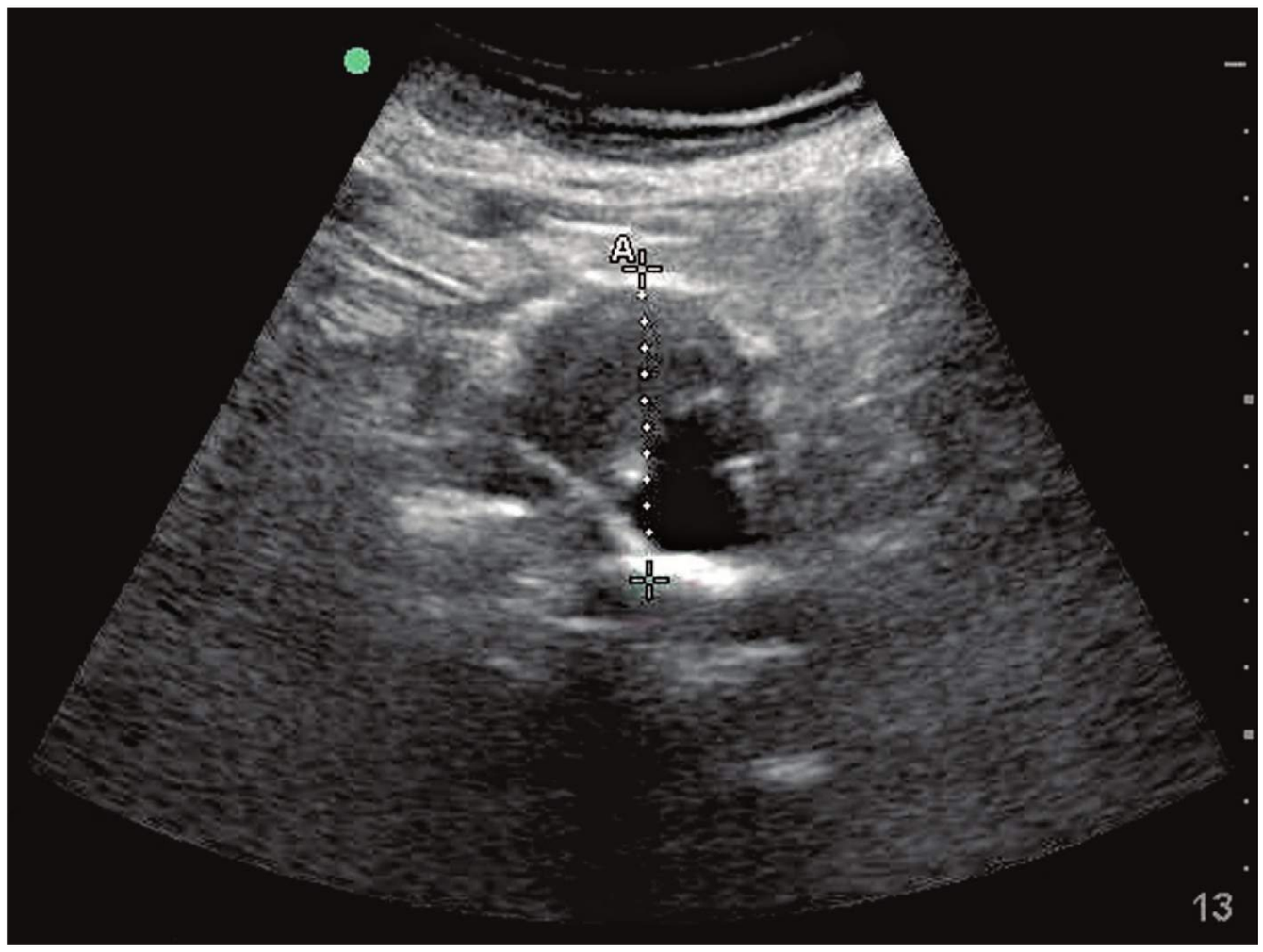


FIGURE 24.64 ■ **Abdominal Aorta: Transverse View, AAA.** Mid-aorta showing an abdominal aortic aneurysm with clot partially occluding the vessel. (Ultrasound contributor: Louis Frazier, MD.)

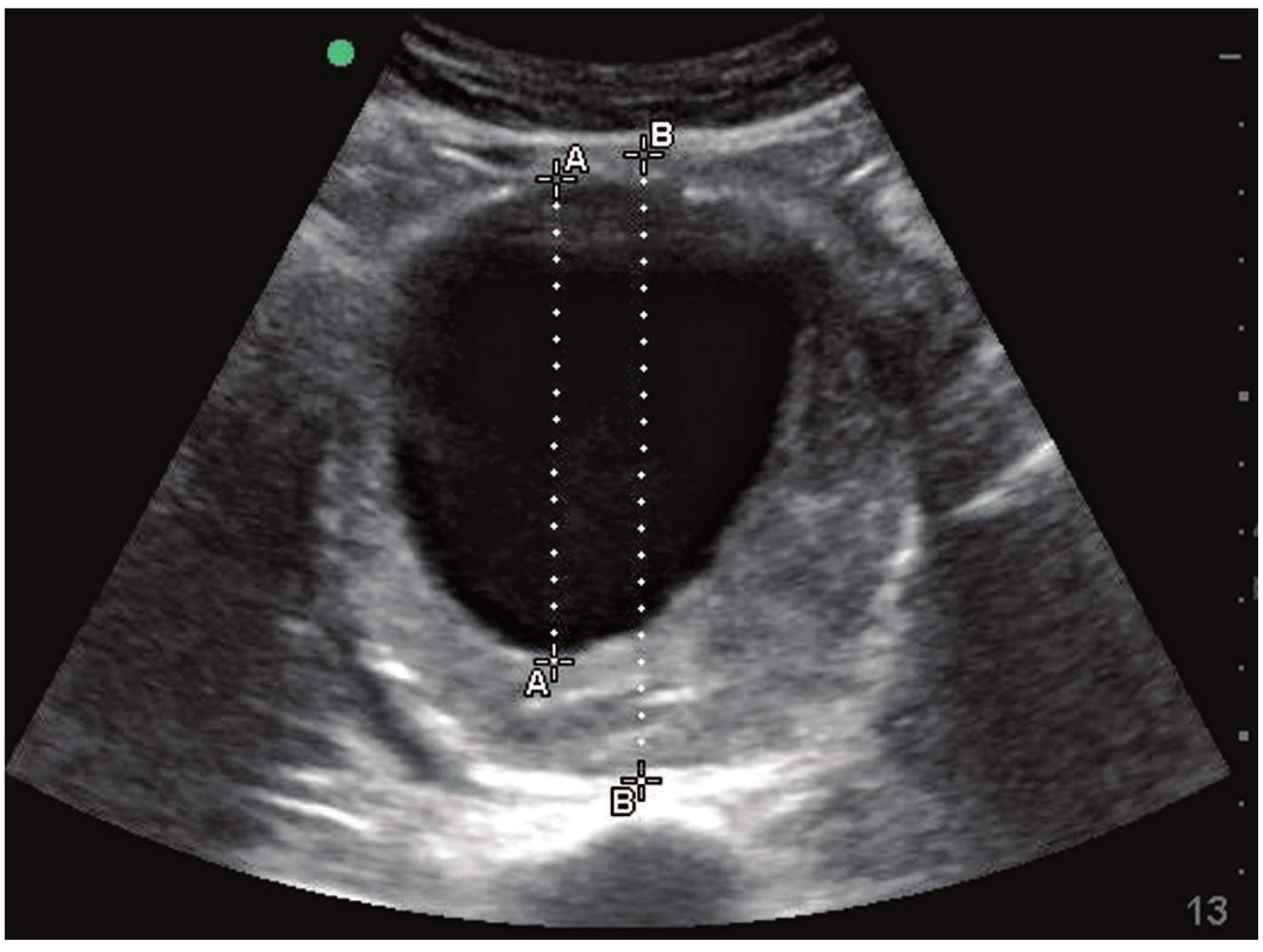


FIGURE 24.65 ■ **Abdominal Aorta: Transverse View, AAA.** Distal abdominal aortic aneurysm with clot filling a portion of the vessel. (Ultrasound contributor: Christopher Noel, MD.)

DEEP VENOUS THROMBOSIS ULTRASOUND

Clinical Summary

The presence of a deep venous thrombosis (DVT) is part of the differential diagnosis of a variety of signs and symptoms. Although clinical scoring algorithms have been developed to gauge risk, no clinical findings are conclusive of this condition and imaging is necessary to confirm the diagnosis. Ultrasound is a sensitive, noninvasive imaging modality that can be performed rapidly and essentially without contraindication. Radiology-performed studies are not always readily available emergently, thus an emergency physician properly trained in bedside ultrasonography can help guide and hasten care by performing a limited serial compression examination of the proximal leg veins. This exam has been shown to be highly sensitive for diagnosis of DVT in symptomatic ED patients when performed by properly trained individuals.

Indications

- Extremity swelling, pain, or erythema
- Dyspnea
- Chest pain

Required Views for Lower Extremity DVT Ultrasound

- Common femoral vein (CFV) (Fig. 24.66)
- Superficial femoral vein (SFV) (Fig. 24.67)
- Popliteal vein (PV) (Fig. 24.68)

Equipment: Recommended Transducers for DVT Ultrasound

- Linear
- Convex

Patient Preparation

- Ideally, the patient is recumbent with the head of the bed elevated 20 to 40 degrees for lower extremity imaging.
- External rotation of the patient's hip with partial knee flexion will aid imaging, especially the popliteal vessels. If tolerated, having the patient lie prone may also aid in imaging the popliteal vasculature.



FIGURE 24.66 ■ **DVT Ultrasound, CFV View.** The transducer should be oriented in a transverse plane just distal to the inguinal ligament. The vein is gently but firmly compressed until the walls completely collapse. (Illustration courtesy of Robinson M. Ferre, MD; photo contributor: Lawrence B. Stack, MD; ultrasound contributor: Robinson M. Ferre, MD.)

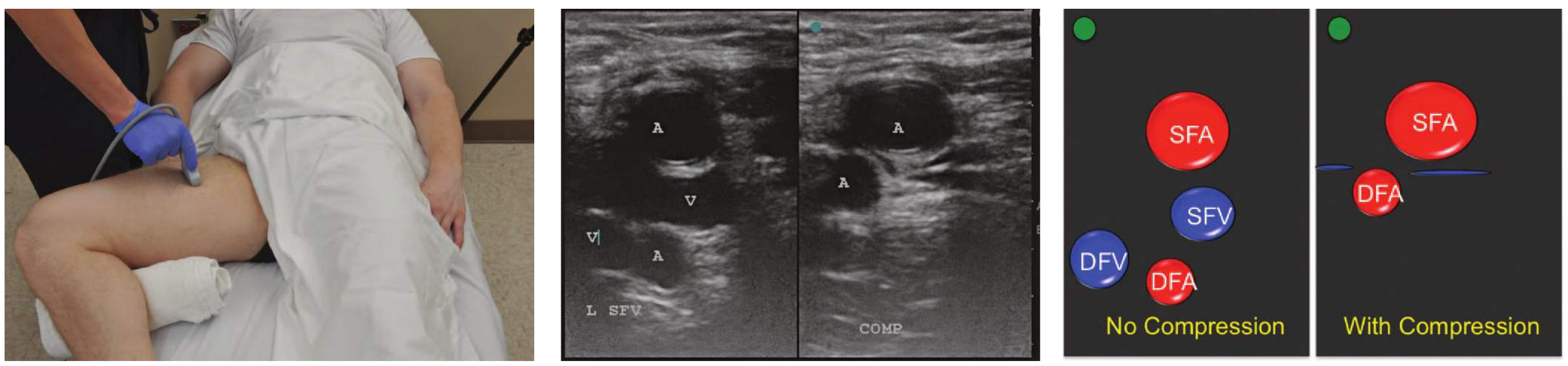


FIGURE 24.67 ■ **DVT Ultrasound, SFV View.** The transducer should be oriented in a transverse plane. The vein(s) is/are gently but firmly compressed until the walls completely collapse. (Illustration courtesy of Robinson M. Ferre, MD; photo contributor: Lawrence B. Stack, MD; ultrasound contributor: Robinson, M. Ferre, MD.)

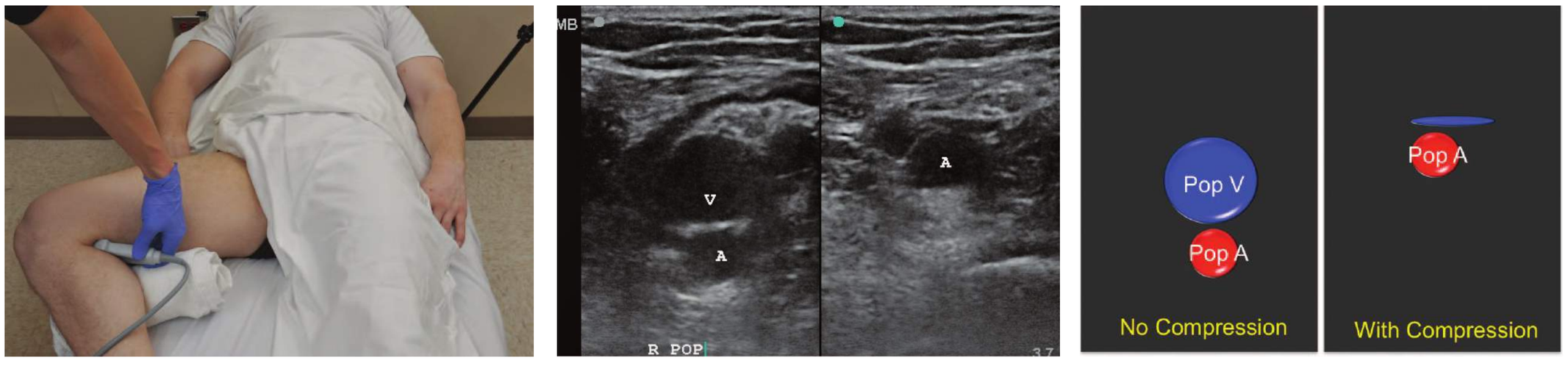


FIGURE 24.68 ■ **DVT Ultrasound, Popliteal View.** The transducer should be oriented in a transverse plane behind the popliteal fossa. The vein is gently but firmly compressed until the walls completely collapse. The popliteal vein is usually found superficial to the popliteal artery, ie, "pop on top." (Illustration courtesy of Robinson M. Ferre, MD; photo contributor: Lawrence B. Stack, MD; ultrasound contributor: Suzanne Bryce, MD.)

Technique

Lower Extremity

- The transducer should be oriented in a transverse plane through the course of the vein.
- Imaging begins at the proximal CFV moving distally to the PV. Carefully identify the CFV, SFV, and PV (Fig. 24.69).
- The vein is gently but firmly compressed until the walls completely collapse. This process is repeated every 1 to 2 cm throughout each site.
- Color or spectral Doppler can be used at the CFV and PV to document phasicity with respiration or augmentation.

Abnormal Findings

- Inability to compress the vein suggests DVT. This may be acute, chronic, or due to technical limitations (eg, unable to apply enough pressure) (Fig. 24.70).
- Poor or absent phasicity suggests occlusion at a location proximal to transducer.
- Poor or absent augmentation suggests occlusion at a location distal to transducer.

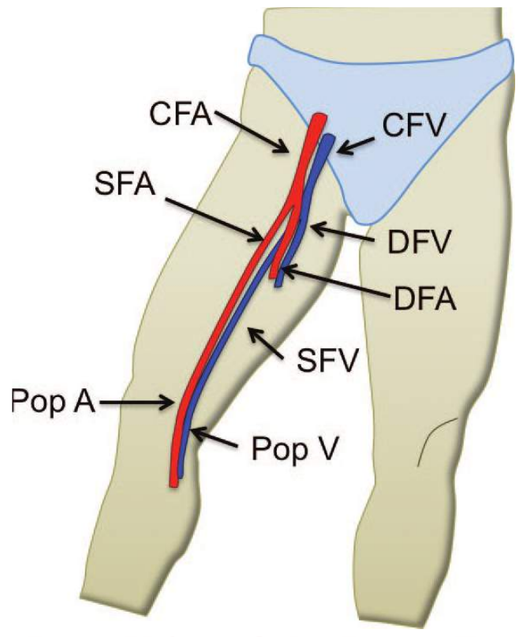


FIGURE 24.69 ■ **Deep Veins of the Lower Extremity.** Diagram illustrating the anatomy of the deep venous system and accompanying arterial system of the lower extremity. Note that the superficial femoral vein (SFV) is actually considered a deep vein of the extremity, despite its name. (Illustration courtesy of Robinson M. Ferre, MD.)

Pearls

1. The CFV is often located more proximally than new sonographers expect. Start the exam adjacent to the inguinal ligament.
2. Thoroughly evaluate the CFV at the confluence of the saphenous vein.
3. Ensure that the vascular structure is a deep vein. Deep veins are paired with arteries. Veins are easily compressible and arteries are not. Color or spectral Doppler should demonstrate characteristic arterial or venous waveforms.
4. If unable to adequately compress vessels, apply pressure from under the extremity toward the transducer. If the artery begins to distort its shape, adequate pressure has been applied.
5. Echogenic material within the vein may be an artifact and not a DVT. Visualize any abnormality in more than one plane and optimize settings to exclude artifacts.

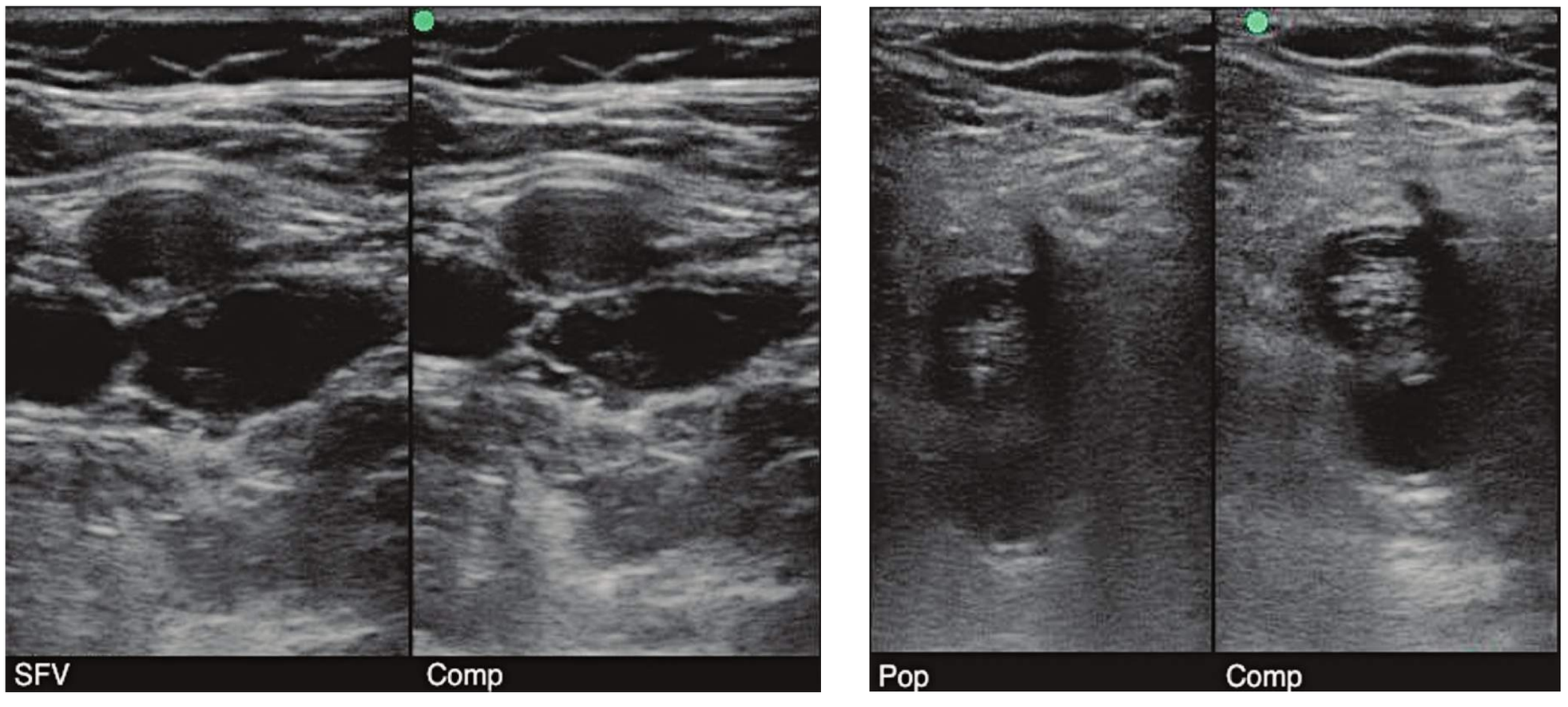


FIGURE 24.70 ■ **DVT Ultrasound.** A deep venous thrombosis is seen as a hyperechoic clot within the SFV (left) and popliteal vein (right) preventing compression. (Ultrasound contributor: Robinson M. Ferre, MD.)

Clinical Summary

As ultrasound is integrated into the bedside evaluation of many emergent symptoms and disease states, multiple protocols have emerged for the rapid assessment of critically ill patients in shock and cardiac arrest states. By combining many of the exams described previously in this chapter, one can quickly assess for the cause of a patient's hypotension or cardiac arrest, helping to sort through what is often an extensive differential diagnosis. A common approach is termed the "RUSH" protocol—Rapid Ultrasound for Shock and Hypotension. Though there are distinct protocols proposed by different authors, they combine a sonographic assessment of a patient's gross cardiac function, intravascular volume status, and identification of pathologic conditions (such as AAA or pneumothorax) that threaten a patient's hemodynamic stability. One group of authors promotes the use of the heuristic "Pump, Tank, and Pipes" as the mental framework for this evaluation, while another uses the mnemonic "HI-MAP" (heart, IVC, Morison [and other FAST views], aorta, and pulmonary/pneumothorax) to outline the protocol. Regardless of which specific protocol one uses for evaluation of the hypotensive patient, when combined with a history and physical exam, bedside ultrasound can be used to guide resuscitative efforts and tailor them to the type of shock encountered.

In addition, when confronted with a patient in cardiac arrest, bedside ultrasound can be used during CPR to help guide therapies and decision making. The protocols described previously can be used to search for the cause of a patient's arrest. Additionally, the subxiphoid and apical four-chamber windows can even be used during compressions or rapidly during pulse checks to determine the native function of the heart and distinguish between true PEA or "electromechanical dissociation" (where there is an electrical rhythm conducted but no cardiac activity) and "pseudo-PEA" where the heart's contractions are so poor that they do not produce palpable pulsations. One can sometimes even distinguish between asystole and fine ventricular fibrillation on the monitor by looking closely at the mitral valve for motion during a pulse check. Alternatively, some emergency physicians are utilizing transesophageal ultrasound probes for use in cardiac arrest—obviating the need to interrupt compressions and allowing for assessment of cardiac compressions during CPR.

Indications

- Unexplained hypotension or shock
- Cardiac arrest

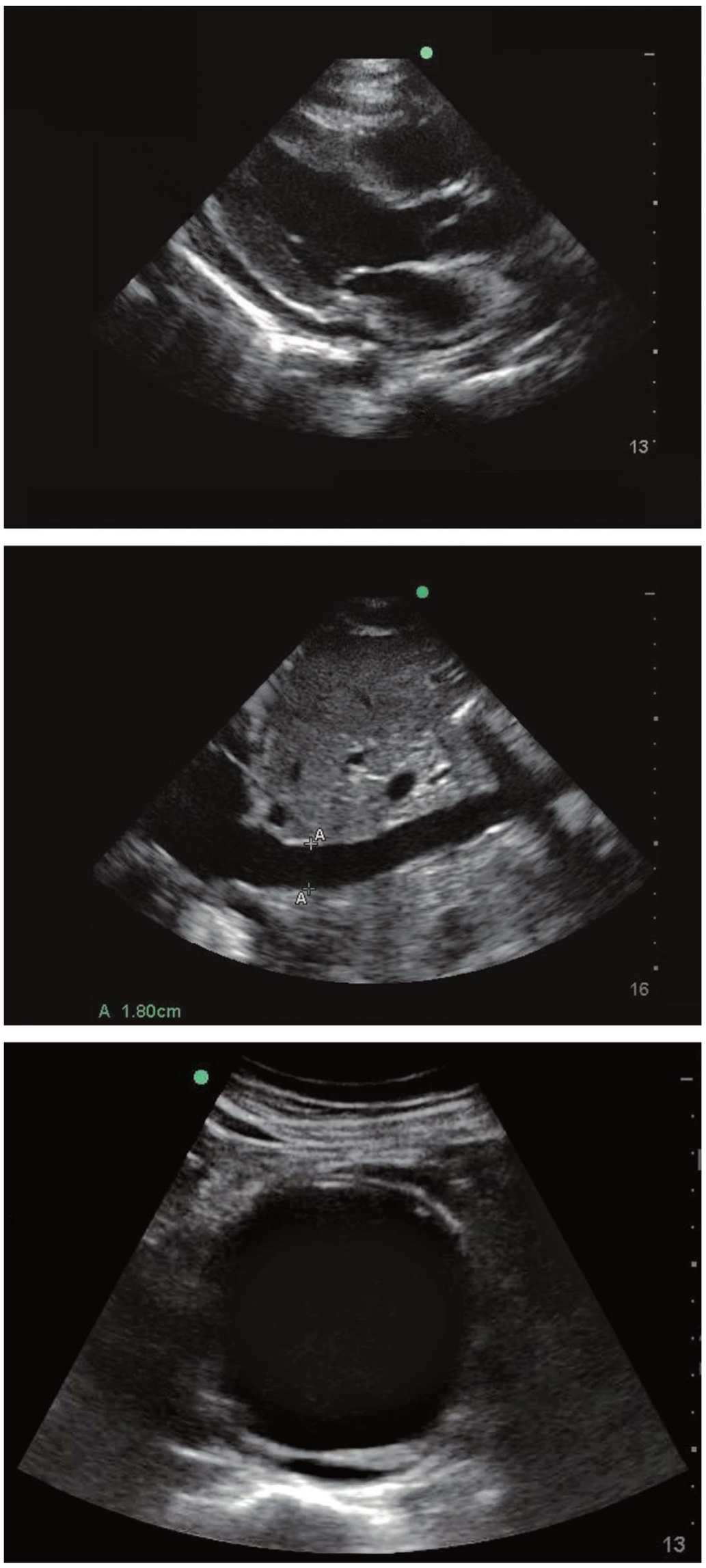


FIGURE 24.71 ■ **Ultrasound-Guided Resuscitation**. Representative images of anatomical areas assessed during ultrasound-guided resuscitation. The heart ("pump"), IVC + FAST ("tank"), and great vessels ("pipes") are assessed confluently to evaluate for the etiology of hypotension and shock. (Ultrasound contributors: Jeremy S. Boyd, MD and Lara Philips, MD.)

Once the patient has been evaluated, findings on ultrasound may be correlated with one of the types of shock identified below. The table below lists representative findings within each shock state. Note that each finding listed below is neither sufficient nor required for the diagnosis of different types of shock, and is merely suggestive. Of course, the clinical context of each patient's presentation must be taken into account in any resuscitation.

Pearls

- The subxiphoid and apical four-chamber views can be obtained during CPR without interrupting chest compressions.
- Parasternal cardiac views might be assessed during a pulse check, but should be evaluated as rapidly as possible so as to minimize chest compressions.
- When obtaining parasternal views during an arrest, have a towel ready to wipe off any gel afterward to avoid interfering with chest compressions.
- Tailor the resuscitation exam to the patient's presenting symptoms. If there is trauma, start with an E-FAST exam. If the patient presents with acute dyspnea, consider starting with cardiac or lung ultrasound first.
- In asystole, there is no spontaneous cardiac activity seen on cardiac ultrasound. Blood can often be seen swirling within the motionless ventricles when assessing after compressions.

TABLE 24.2 ■ APPROACH TO ULTRASOUND-GUIDED RESUSCITATION

	Views	Questions
"Pump"	Focused cardiac assessment: • Parasternal long • Parasternal short • Apical four-chamber • Subxiphoid	• Is there a pericardial effusion? • What is the gross left ventricular function? • Are there signs of right heart strain?
"Tank"	Volume status and integrity: • IVC • E-FAST exam	• Is the IVC collapsed or plethoric? • Is there hemoperitoneum? • Is there a hemothorax or pneumothorax? • Is there pulmonary edema?
"Pipes"	Vascular pathology: • Aorta • DVT	• Is there an AAA? • Is there an aortic dissection? • Is there a DVT?

TABLE 24.3 ■ SONOGRAPHIC FINDINGS IN VARIOUS SHOCK STATES

	Hypovolemic/ Hemorrhagic Shock	Cardiogenic Shock	Obstructive Shock	Distributive Shock
Pump	Hyperdynamic heart	Hypocontractile heart Dilated cardiomyopathy	Pericardial effusion Right heart strain McConnell sign[a] Hyperdynamic heart	Hyperdynamic heart
Tank	Collapsed IVC Hemoperitoneum Hemothorax	Distended IVC Pulmonary edema Pleural effusions	Distended IVC Absent lung sliding/lung point (pneumothorax)	Normal/collapsed IVC Pleural fluids (empyema) Focal B-lines (pneumonia)
Pipes	AAA Aortic dissection	Normal	DVT	Normal

Modified and adapted from Weingart et al. Rapid Ultrasound for Shock and Hypotension (RUSH-HIMAPP). 2009. **http://emedhome.com/**; Perera P, Mailhot T, Riley D, Mandavia D. "The RUSH exam: Rapid Ultrasound in SHock in the evaluation of the critically ill. Emerg Med Clin North Am. 2010;28(1):29–56; and Seif D, Perera P, Mailhot T, Riley D, Mandavia D. Bedside ultrasound in resuscitation and the rapid ultrasound in shock protocol. Crit Care Res Pract. 2012; 2012:503254.

[a]McConnell sign is a finding seen on focused cardiac ultrasound in the apical four-chamber view. It is characterized by RV free wall hypokinesis with preserved apical contractility. It is a sign of acute RV strain, whether due to massive pulmonary embolism or RV infarct.

Clinical Summary

Biliary ultrasound can be very rewarding. A rapid, focused exam can help determine if gallstones or gallbladder pathology is the etiology of a patient's presenting symptoms. In turn, bedside ultrasound can expedite therapy or expedite the pursuit of an alternative diagnosis. While the sonographic identification of gallstones may seem straightforward, the sonographic findings for cholecystitis are often subtle.

Indications

- Right-upper-quadrant pain
- Jaundice
- Epigastric pain

Views for Emergency Department Biliary Ultrasound

- Sagittal view of the gallbladder
- Transverse view of the gallbladder

Equipment: Recommended Transducers for Gallbladder Ultrasound

- Convex array
- Phased array

Patient Positioning

- Supine or left lateral decubitus position

Techniques

Sagittal View

- Initially, the transducer is placed in the subxiphoid region with the indicator directed toward the patient's head in a sagittal view and swept below the right costal margin to approximately the midclavicular line. The transducer is then rotated (often clockwise) to obtain a view of the gallbladder in its longest axis (Fig. 24.72).
- Scan through the gallbladder completely from medial to lateral borders including careful evaluation of the gallbladder neck. A thorough search for gallstones should be made, particularly at the neck where small symptomatic gallstones may be seen.
- Identify the liver, gallbladder, portal vein, hepatic artery, main lobar fissure, and the common hepatic duct (Figs. 24.73 and 24.75).



FIGURE 24.72 ■ **Gallbladder: Sagittal View.** Probe is placed just under the costal margin and swept across the costal margin to locate the gallbladder. The transducer is then rotated (often clockwise) to obtain a view of the gallbladder in its longest axis. (Photo contributor: Lawrence B. Stack, MD.)

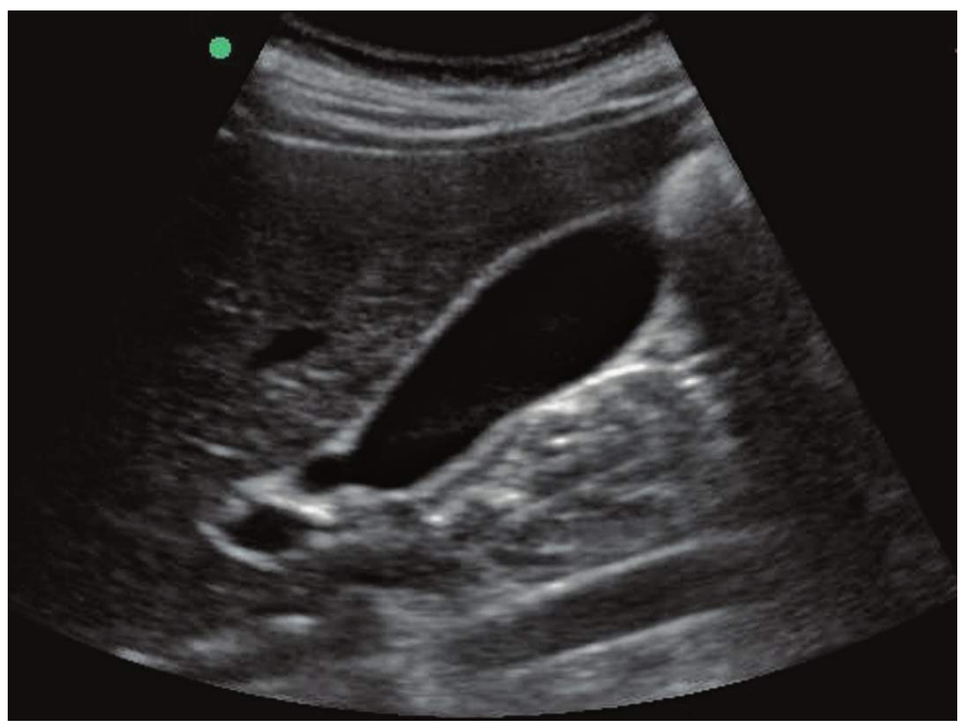


FIGURE 24.73 ■ **Gallbladder: Sagittal View.** Long-axis view of gallbladder often reveals an "exclamation mark" sign made of the gallbladder and the portal triad. (Ultrasound contributor: Robinson M. Ferre, MD.)



FIGURE 24.74 ■ **Gallbladder: Transverse.** Rotate the probe 90 degrees counterclockwise to bring the gallbladder into short axis. (Photo contributor: Lawrence B. Stack, MD.)

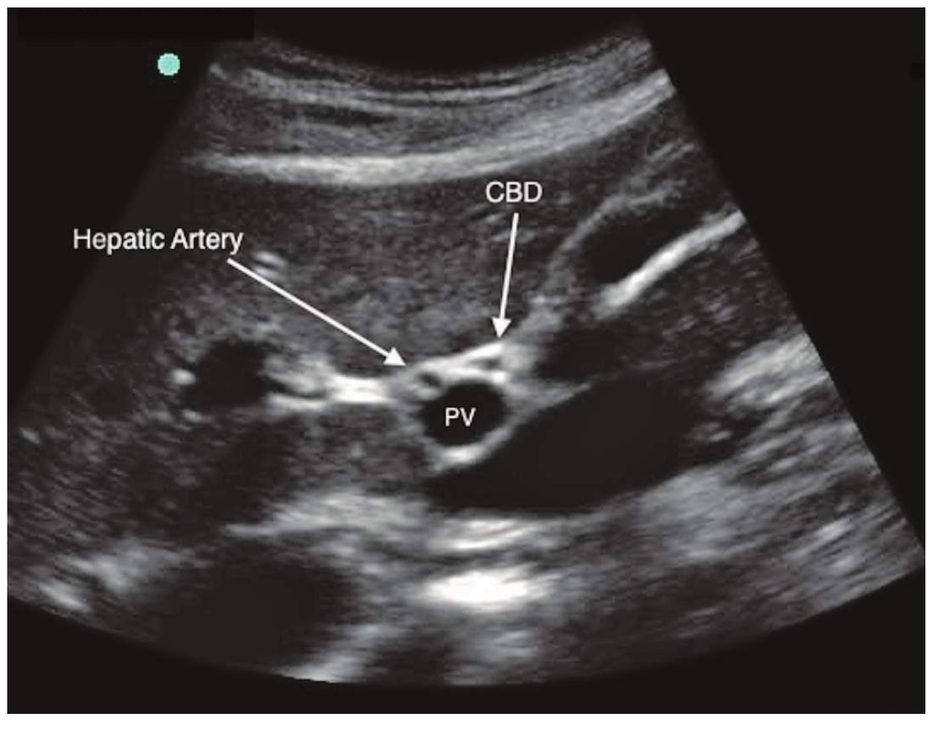


FIGURE 24.75 ■ **Gallbladder: Portal Triad.** The portal vein (PV), hepatic artery, and common bile duct (CBD) are readily seen in the transverse view. (Ultrasound contributor: Shannon Snyder, MD, RDMS.)

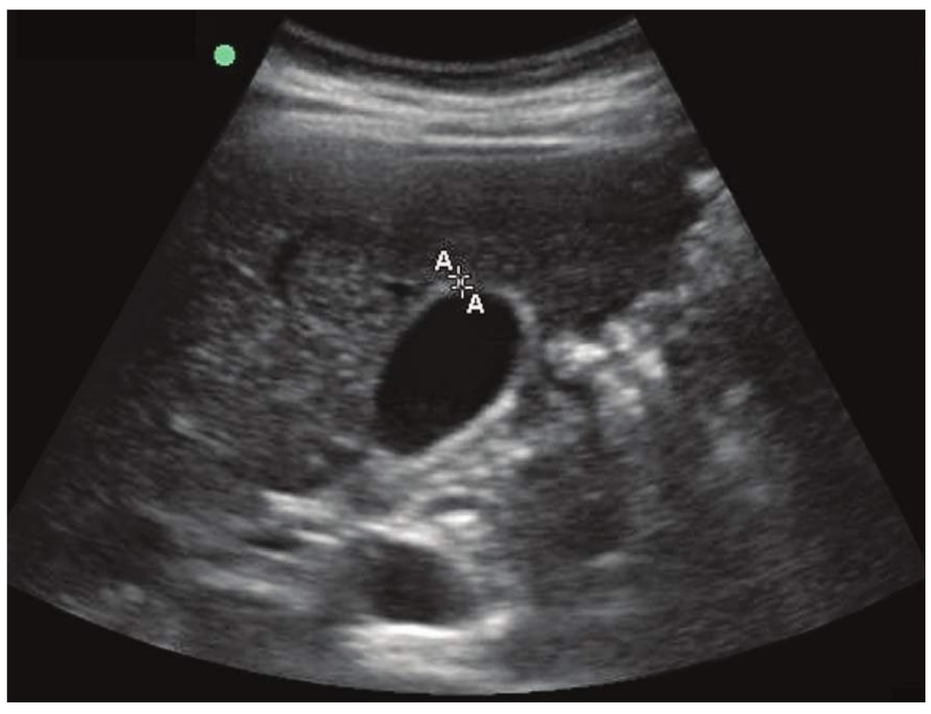


FIGURE 24.76 ■ **Gallbladder: Transverse.** Short-axis view of the gallbladder measuring the anterior wall thickness. (Ultrasound contributor: Robinson M. Ferre, MD.)

Transverse View

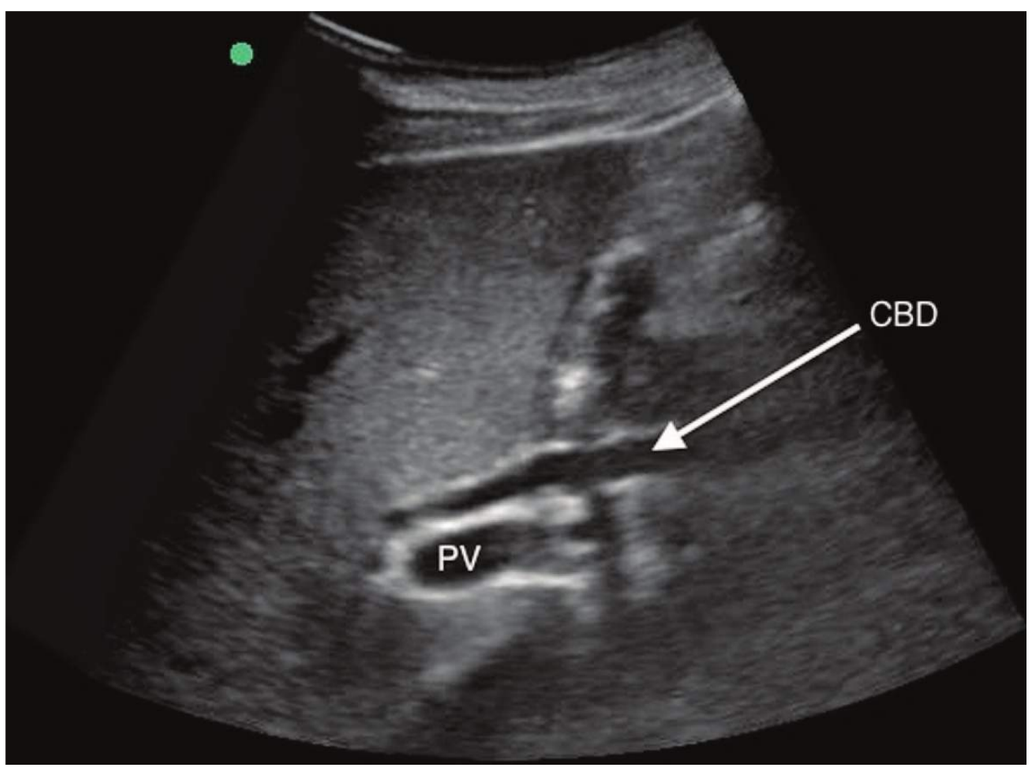
- After identifying the gallbladder in the sagittal position, rotate the transducer 90 degrees counterclockwise toward the patient's right (Fig. 24.74).
- Identify the liver, gallbladder, IVC, and common bile duct (CBD). Measure the anterior wall of the gallbladder for thickness (Fig. 24.76).

Additional Views

- If indicated, find the CBD. This can be done in two ways. First, identify the common hepatic duct at the level of the porta hepatis, and trace it down beyond the border of the liver.
- Measure the thickness of the CBD when visible from inside wall to inside wall (Fig. 24.77). Color flow Doppler will help delineate the CBD (no flow) from the hepatic artery (Fig. 24.78).

Abnormal Findings

- Gallstones: Hyperechoic (bright) oval to round structure(s) within the gallbladder, often with accoustic shadowing (Fig. 24.79A,B). Gallstones should be mobile unless impacted in the neck of the gallbladder (Fig. 24.80). Rolling the patient will allow assessment of gallstone mobility.



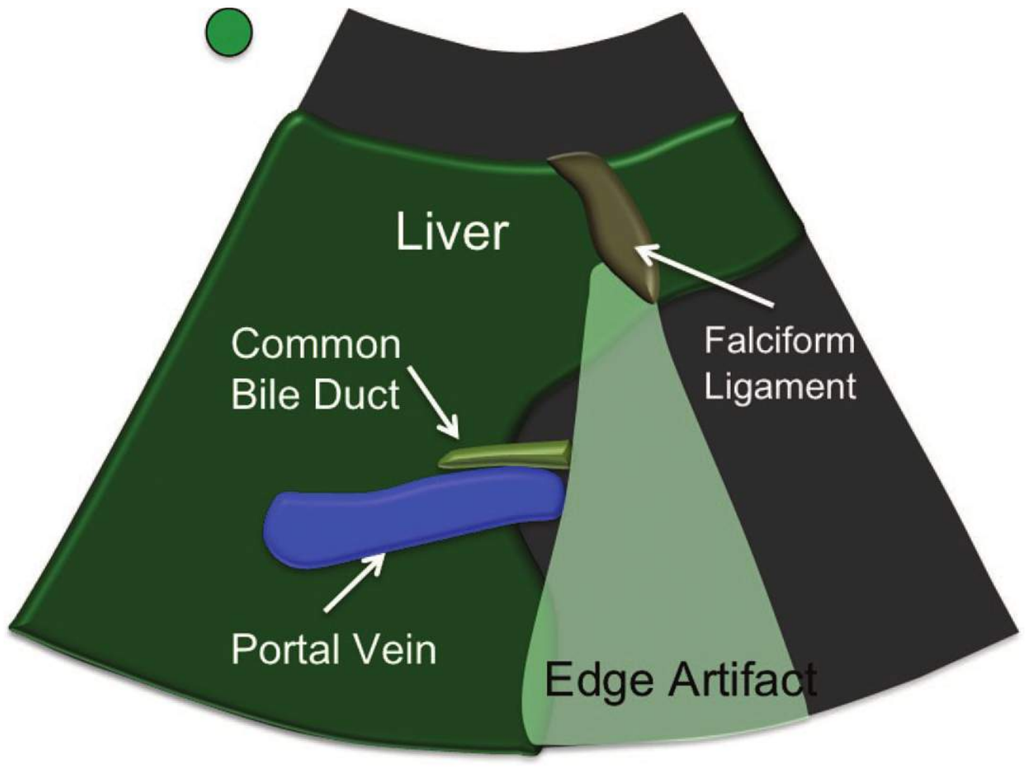


FIGURE 24.77 ■ **Gallbladder: Common Bile Duct.** Long-axis view of the portal vein (PV) and dilated common bile duct (CBD). The CBD is measured from inside wall to inside wall. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Robinson M. Ferre, MD.)

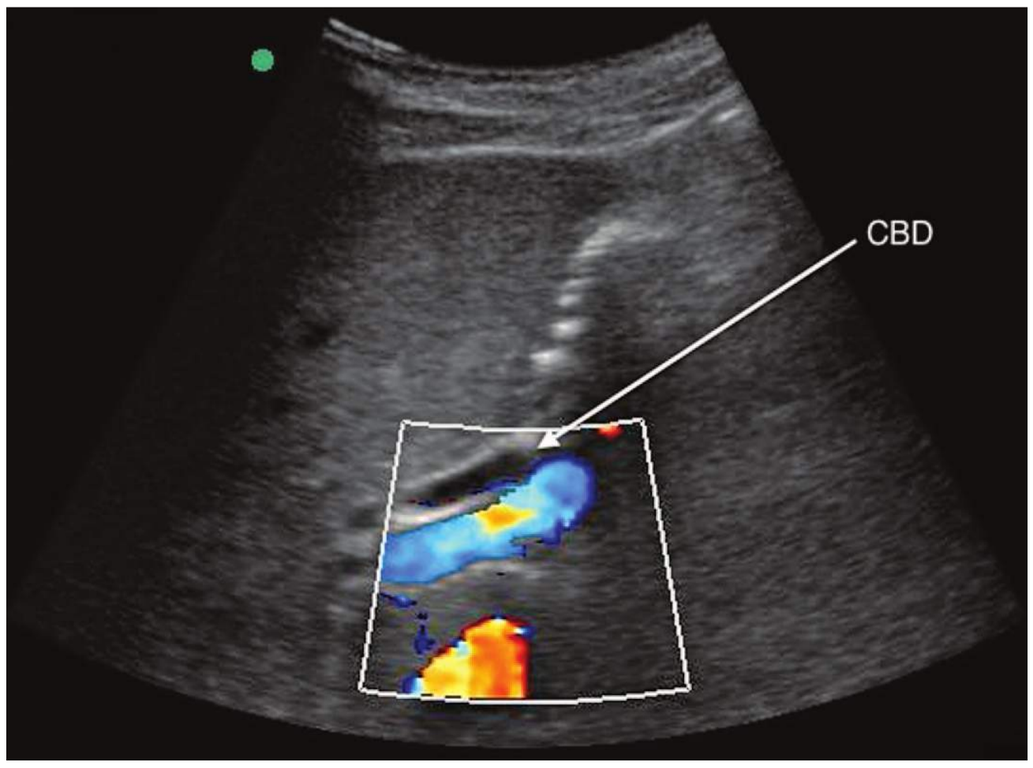
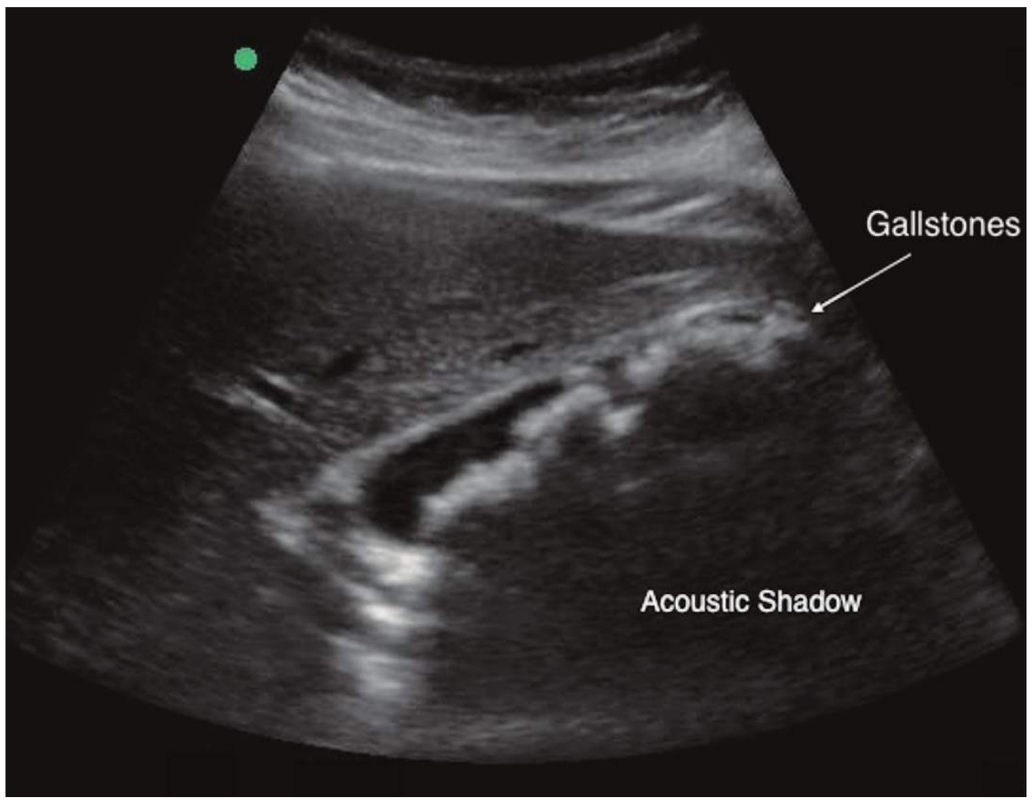


FIGURE 24.78 ■ **Gallbladdder: Common Bile Duct, Color Flow Doppler.** Long-axis view of the portal vein (color flow) and dilated common bile duct (CBD) without color flow. (Ultrasound contributor: Max Palatnik, MD.)

- Pericholecystic fluid: An anechoic (dark) stripe that borders the outer gallbladder wall and the liver. It will be visible in two views (Fig. 24.81A,B).
- Thickened gallbladder wall: A gallbladder wall that measures greater than 3 mm is considered abnormal (Fig. 24.82).
- Sonographic Murphy sign: When direct visualized compression of the gallbladder by the ultrasound probe reproduces the patient's symptoms of abdominal pain and this pain is not reproduced by compression with the ultrasound probe elsewhere in the surrounding area.
- Dilated CBD: A CBD with an internal diameter greater than 6 mm is dilated; however, documented measurements up to 8 to 9 mm can be normal in the elderly. One rule of thumb is that the CBD can measure up to 6 mm up to age 60 and thereafter an increase of 1 mm per decade of life represents the normal range.

Pearls

1. Other positions—including prone, right lateral decubitus, semi-erect, and standing—may be helpful in scanning the gallbladder and are patient specific.
2. Having the patient hold their breath after deep inspiration may help the gallbladder descend into view below the ribcage.
3. Always measure the anterior wall of the gallbladder. The thickness of the posterior wall often appears falsely thickened by posterior acoustic enhancement.
4. Small gallstones (1-3 mm) may not be large enough to cause acoustic shadowing. Increasing the frequency and ensuring that your focal zone is on the area of interest will increase the likelihood that shadowing will be seen.
5. The CBD lies just anterior to the portal vein, a structure with markedly hyperechoic walls compared to the hepatic veins. Placing the patient in the left lateral decubitus position may help visualize the CBD.



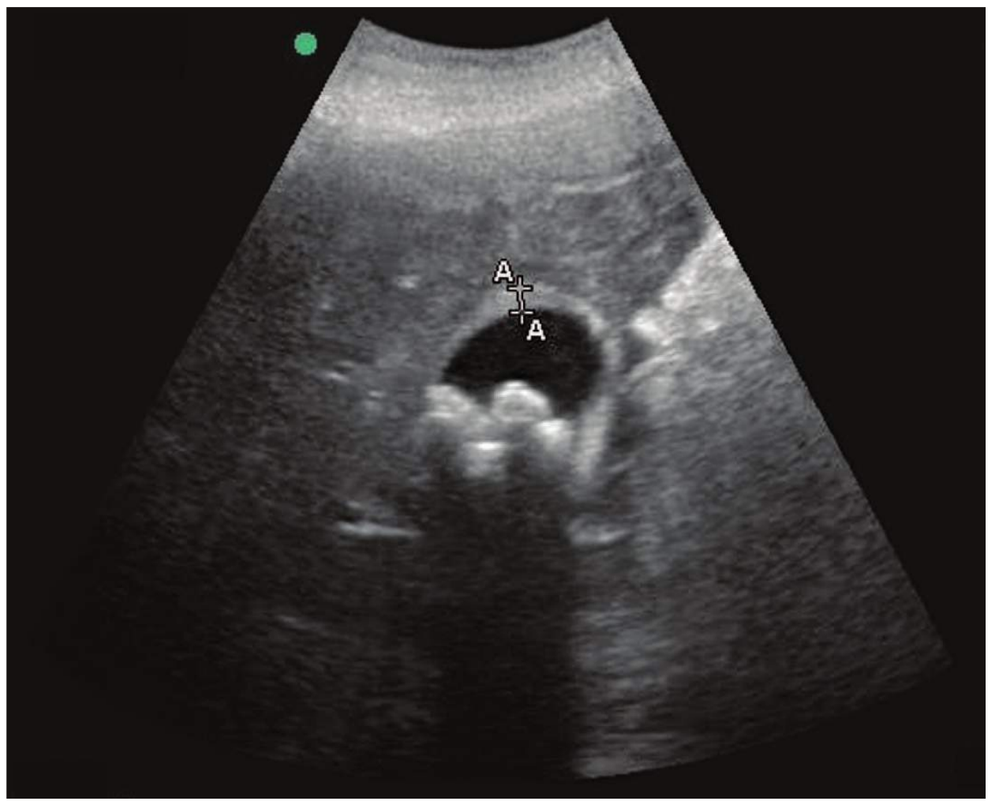


FIGURE 24.79A, B ■ **Gallbladder: Gallstones.** Long- and short-axis views of the gallbladder with a large number of gallstones casting an acoustic shadow posterior to the calcified stones. (Ultrasound contributor: Shannon Snyder, MD, RDMS.)

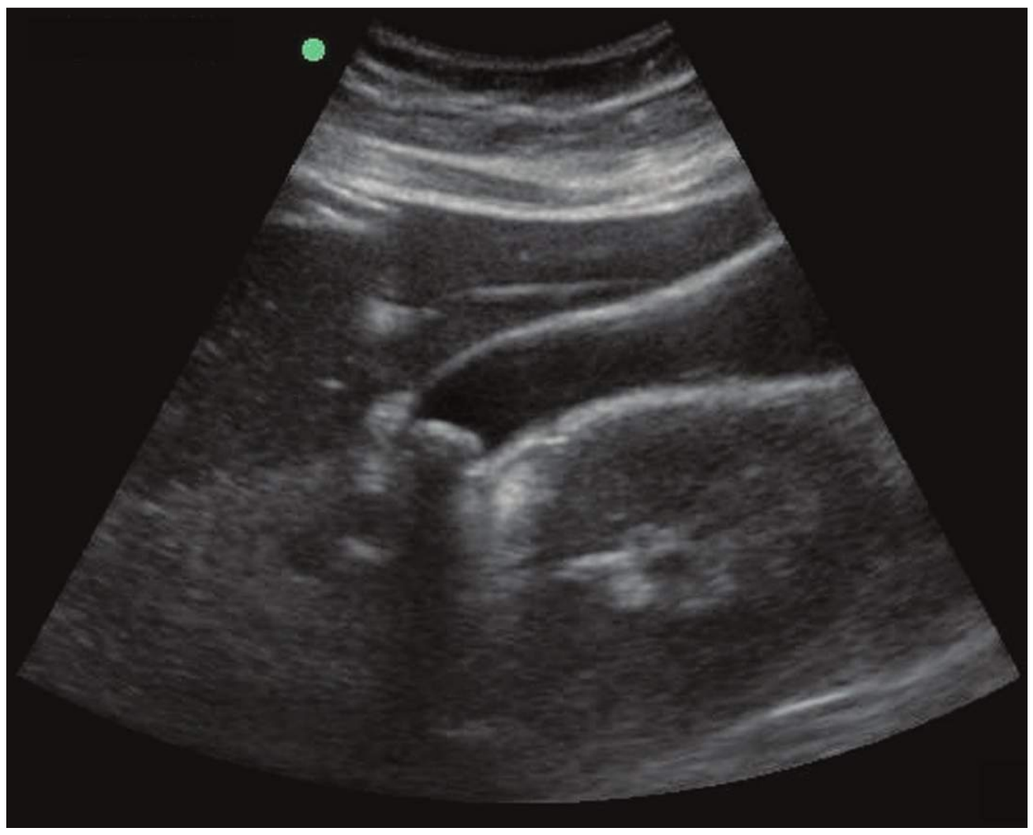


FIGURE 24.80 ■ **Gallbladder: Sagittal View, Gallstone.** Long-axis view of the gallbladder with a single gallstone lodged in the gallbladder neck. (Ultrasound contributor: Robinson M. Ferre, MD.)

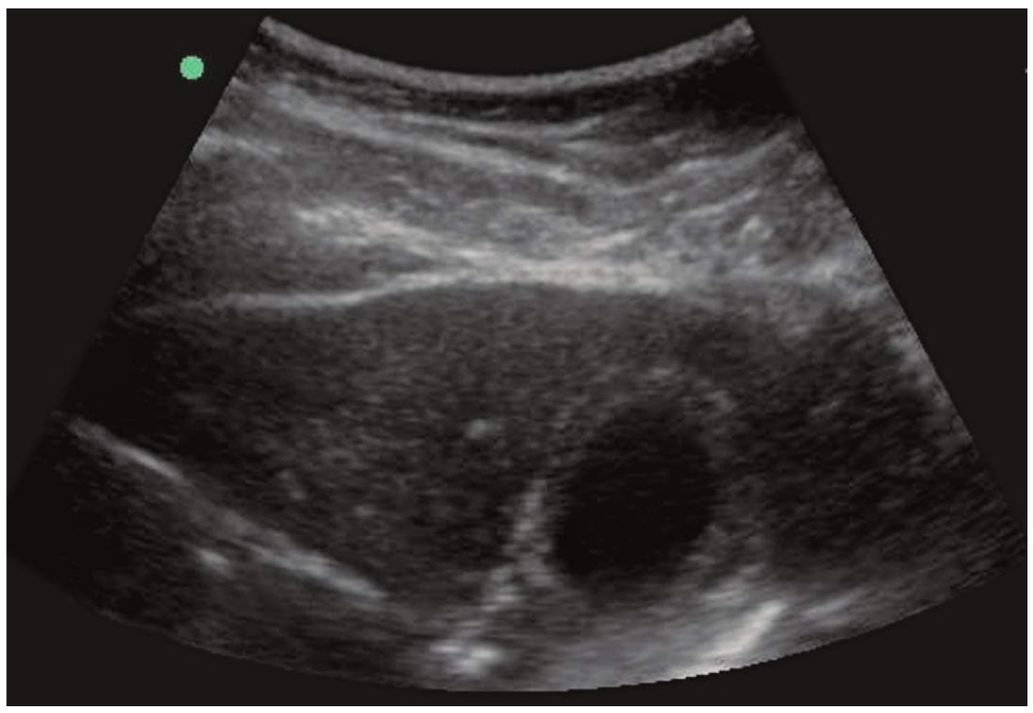
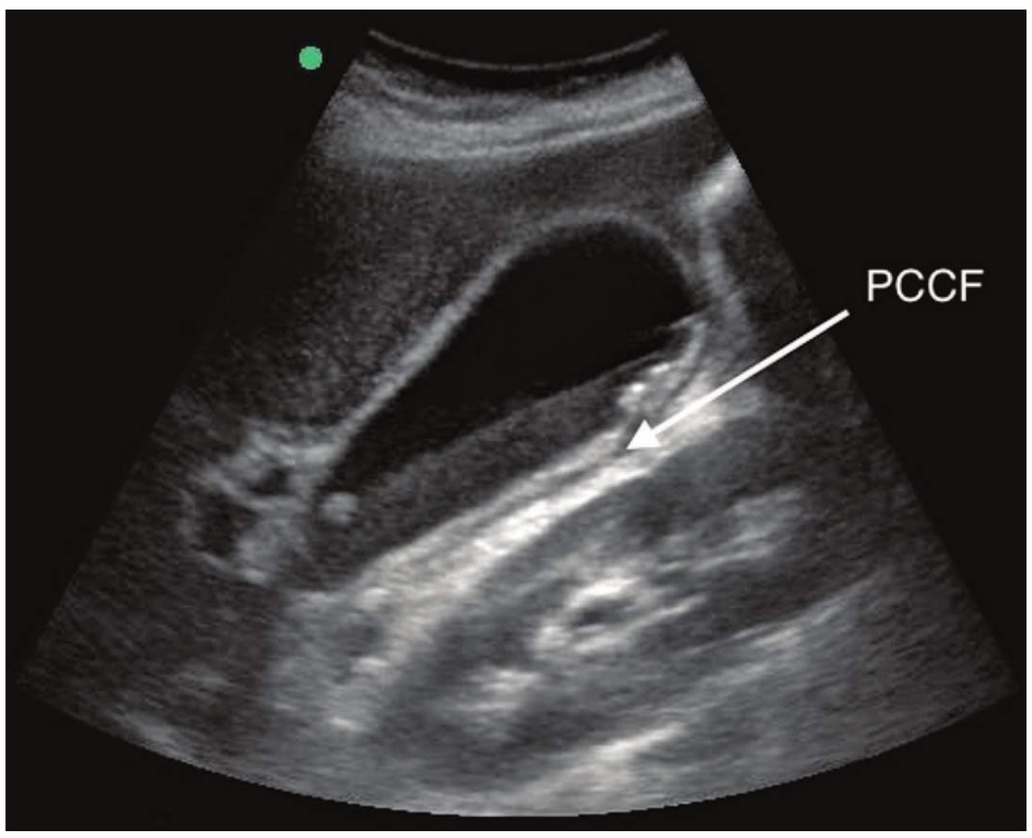


FIGURE 24.82 ■ **Gallbladder: Transverse View, Cholecystitis.** Short-axis view of the gallbladder with thickening of the wall. (Ultrasound contributor: Jordan Rupp, MD.)



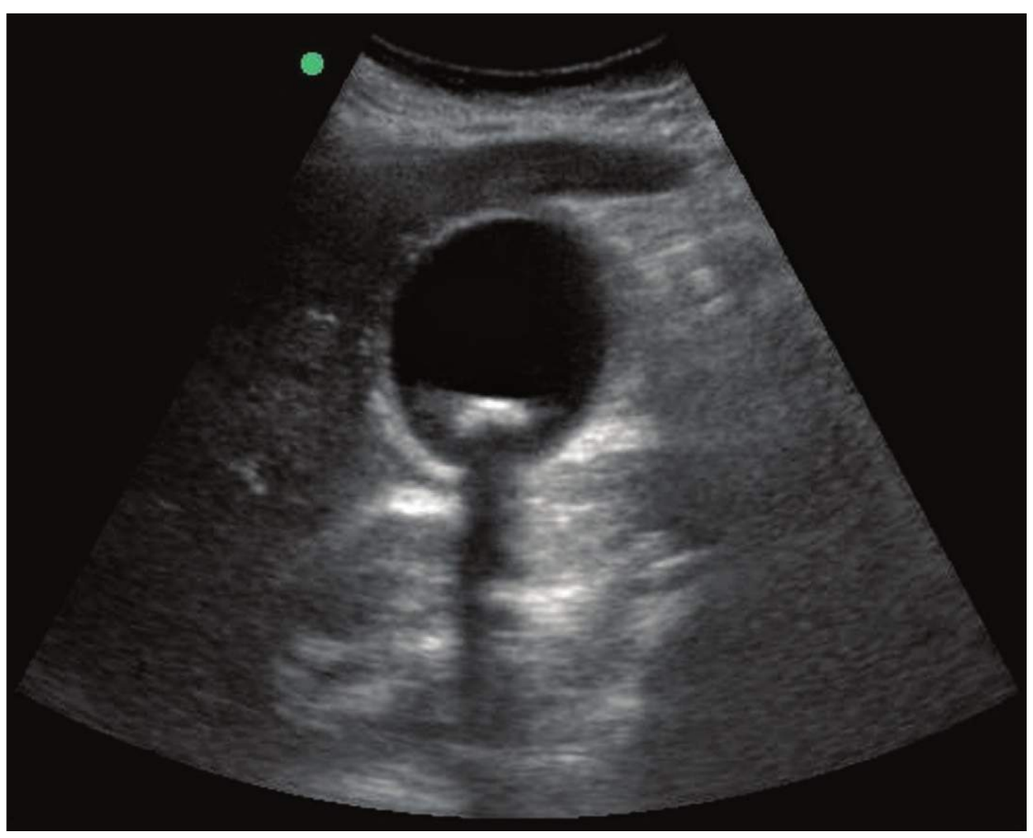


FIGURE 24.81A, B ■ **Gallbladder.** Gallbladder sludge, gallstone, and pericholecystic fluid (PCCF). Long- and short-axis view of the gallbladder showing a gallstone and biliary sludge. There is also a thin stripe of PCCF. (Ultrasound contributor: Jeremy Boyd, MD.)

Clinical Summary

Ultrasound of the kidneys and bladder can yield helpful diagnostic information for patients presenting with symptoms of urinary retention, decreased urinary output, or abdominal/flank pain consistent with renal colic. Obstructive uropathy due to kidney stones is the principal pathology identified with emergency physician–performed renal ultrasound. Bladder volume is easily calculated by viewing and measuring the bladder in two planes. In focused renal ultrasound, the goal is not to identify renal or ureteral calculi, rather the kidneys are evaluated for hydronephrosis. The presence of hydronephrosis in the patient with renal colic is presumed to be a direct result of ureteral obstruction. There are no accurate means of determining the degree of obstruction by the presence of hydronephrosis.

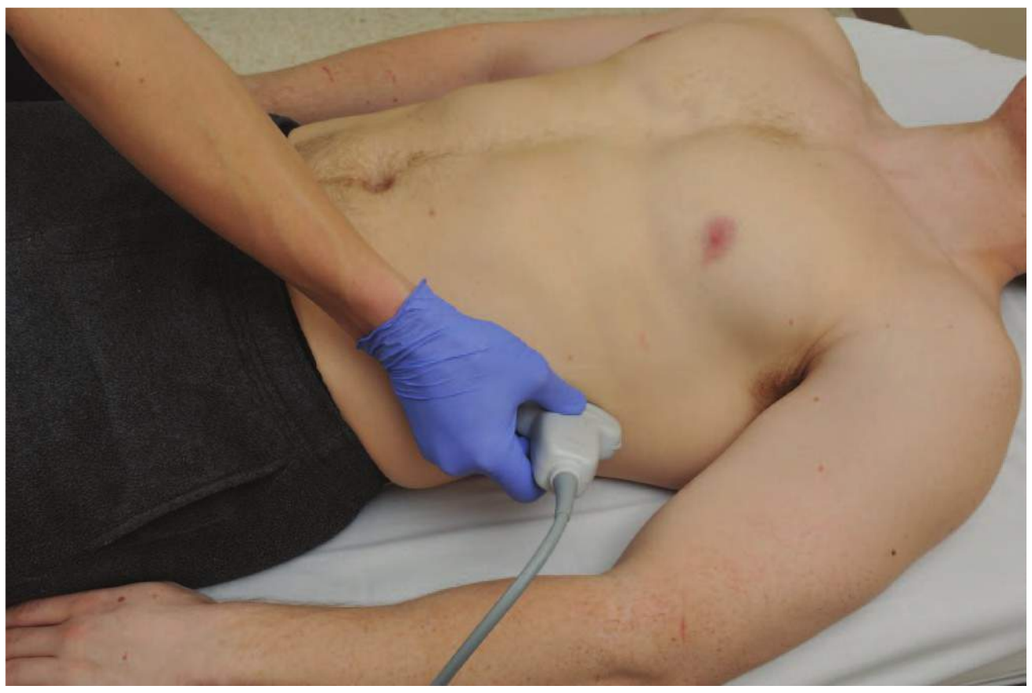


FIGURE 24.83 ■ **Renal Ultrasound.** The transducer is directed as a coronal section through the body in the mid to posterior axillary lines. (Photo contributor: Lawrence B. Stack, MD.)

Indications

- Flank/abdominal pain
- Renal colic
- Hematuria
- Urinary retention
- Decreased urinary output

The recommended sonographic approach to the kidney is similar to that for the RUQ and LUQ windows in the E-FAST examination previously discussed, with the probe angled toward the retroperitoneum for better views of the kidneys. The coronal view allows the sonographer to visualize the right or left kidney from the superior to inferior poles. Transverse or axial views of the entire kidney should be obtained as well. Renal EUS is most easily interpreted when comparative images are obtained between the right and left kidneys. The bladder view is also nearly identical to that of the E-FAST exam described earlier. However, rather than focusing on the potential spaces of the peritoneum, attention is focused on the bladder and its contents.

Views for Emergency Department Renal and Bladder Ultrasound

1. Coronal or sagittal kidney view
2. Transverse kidney view
3. Sagittal bladder view
4. Transverse bladder view

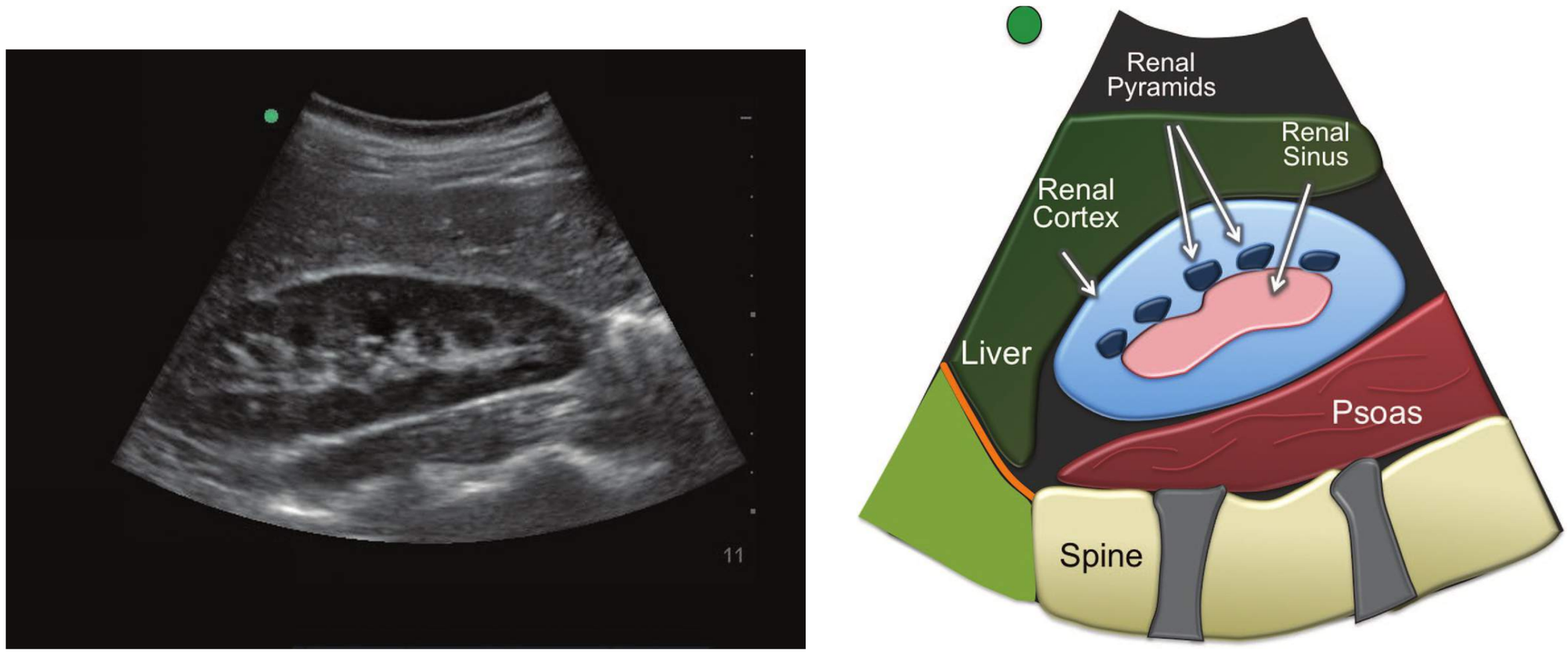


FIGURE 24.84 ■ **Renal Ultrasound, Right Coronal View.** The liver, right kidney, renal cortex (with pyramids), and central renal sinus are seen. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Shannon Snyder, MD, RDMS.)

Equipment: Recommended Transducers for Renal Ultrasound

- Convex array
- Phased array

Patient Position

- The patient is supine.
- Alternatively, the patient may be placed in a prone position with the probe placed in the midscapular line and the transducer directed toward the abdomen.

Technique

Kidney: Right and Left Coronal Views

- The transducer indicator is oriented toward the patient's head.
- The transducer is directed as a coronal section through the body in the mid to posterior axillary lines (Fig. 24.83). Begin scanning between the 9th and 11th ribs on the right and the 8th and 11th ribs on the left.
- Identify the liver, right kidney, renal cortex (with pyramids), and central renal sinus (Fig. 24.84).
- Identify the spleen, left kidney, renal cortex (with pyramids), and central renal sinus.

Kidney: Right and Left Transverse Views

- Obtain the coronal view as directed above, but rotate the transducer 90 degrees counterclockwise to visualize the renal parenchyma and central renal sinus in a cross-sectional view (Fig. 24.85).

Bladder: Sagittal View

- With the indicator oriented toward the patient's head, the transducer is placed just above the symphysis pubis and is directed into the pelvis (see Fig. 24.17).
- Identify the bladder (triangular in this view when fully distended), uterus (pear-shaped if present), prostate, seminal vesicles, and rectum (see Figs. 24.18 and 24.19).

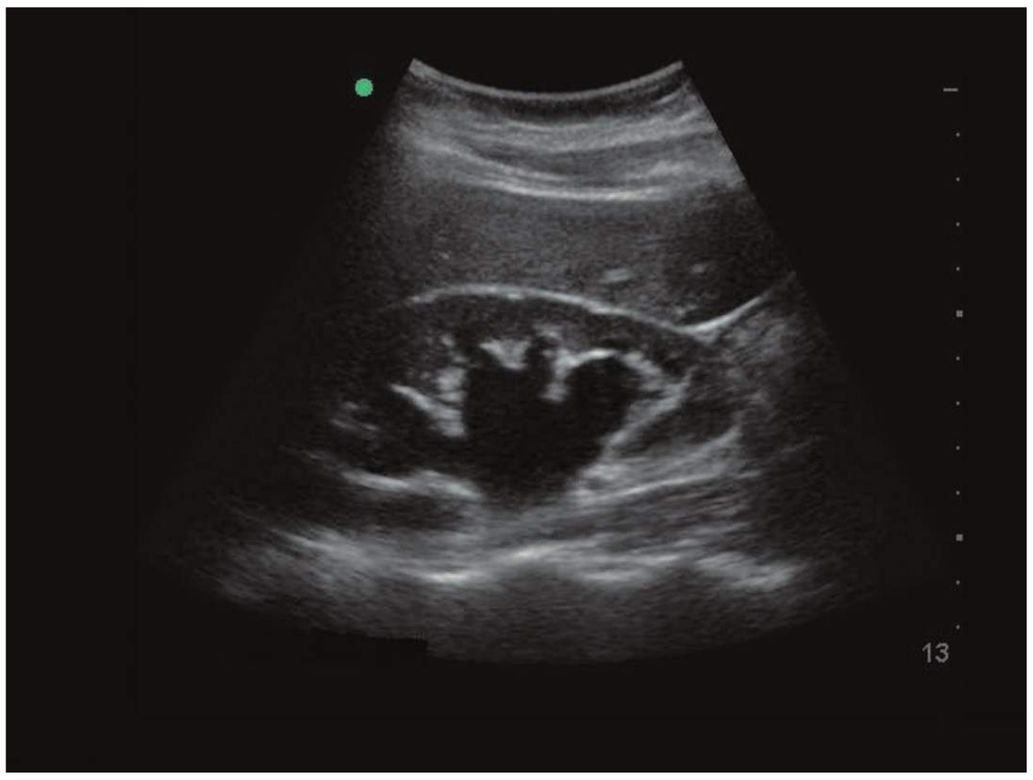
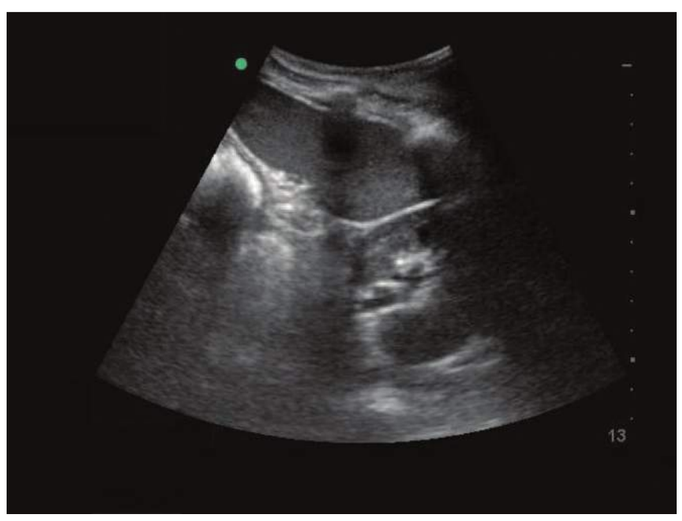


FIGURE 24.86 ■ **Renal Ultrasound: Hydronephrosis.** Moderate hydronephrosis is seen as dilatation of the bright renal sinus and secondary calyces by anechoic (black) fluid. (Ultrasound contributor: Shannon Snyder, MD, RDMS.)





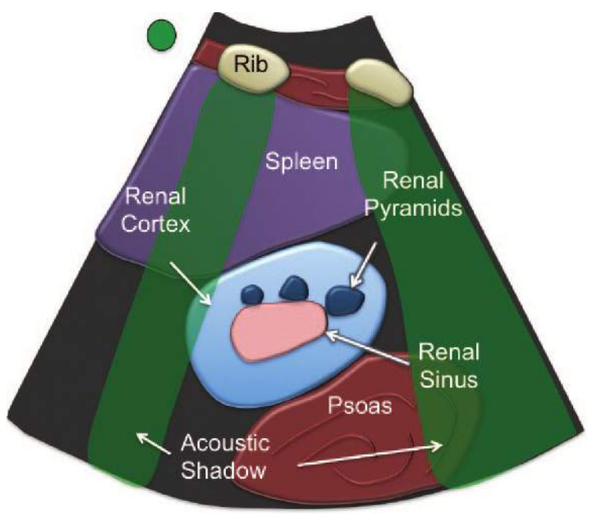


FIGURE 24.85 ■ **Renal Ultrasound, Left Transverse View.** Rotate the transducer 90 degrees counterclockwise to visualize the renal parenchyma and central renal sinus in a cross-sectional view. (Illustration courtesy of Robinson M. Ferre, MD; photo contributor: Lawrence B. Stack, MD; ultrasound contributor: Shannon Snyder, MD, RDMS.)

Bladder: Transverse View

- With the indicator oriented toward the patient's right, the transducer is placed about 1 to 2 cm above the symphysis pubis, with the transducer angled into the peritoneum (see Fig. 24.20).
- Identify the bladder (rectangular in this view when fully distended), uterus (oval hyperechoic structure if present), prostate, seminal vesicles, and rectum (see Fig. 24.21).

Findings

- Hydronephrosis: Dilatation of the bright renal sinus with black, anechoic fluid (Fig. 24.86). Hydronephrosis is graded based on its extension into the secondary calyces and whether there is accompanying cortical thinning (Fig. 24.87).
- Renal calculi: Bright hyperechoic oval/round structures within the cortex or renal sinus (acoustic shadowing is often present). With color flow Doppler, uroliths will often demonstrate twinkling artifact deep to their reflective echogenic surface (Fig. 24.88).
- Urinary retention: By calculating the volume of the bladder, one may determine the presence of urinary retention. Greater than 50 to 100 mL of retained postvoid urine is diagnostic.

Pearls

1. Rib shadows will be evident with the coronal view and transverse views. Have the patient hold his or her breath in inspiration and move the transducer a rib space higher or lower to visualize the kidney from the superior to the inferior pole.
2. The presence of a ureteral jet confirms the passage of urine into the bladder from a specific side. Its absence is sensitive, but not specific for complete ureteral obstruction (Fig. 24.89).
3. When evaluating for renal colic, stones may frequently be seen on the bladder views when they are located at the ureterovesicular junction (UVJ). (Fig. 24.90).

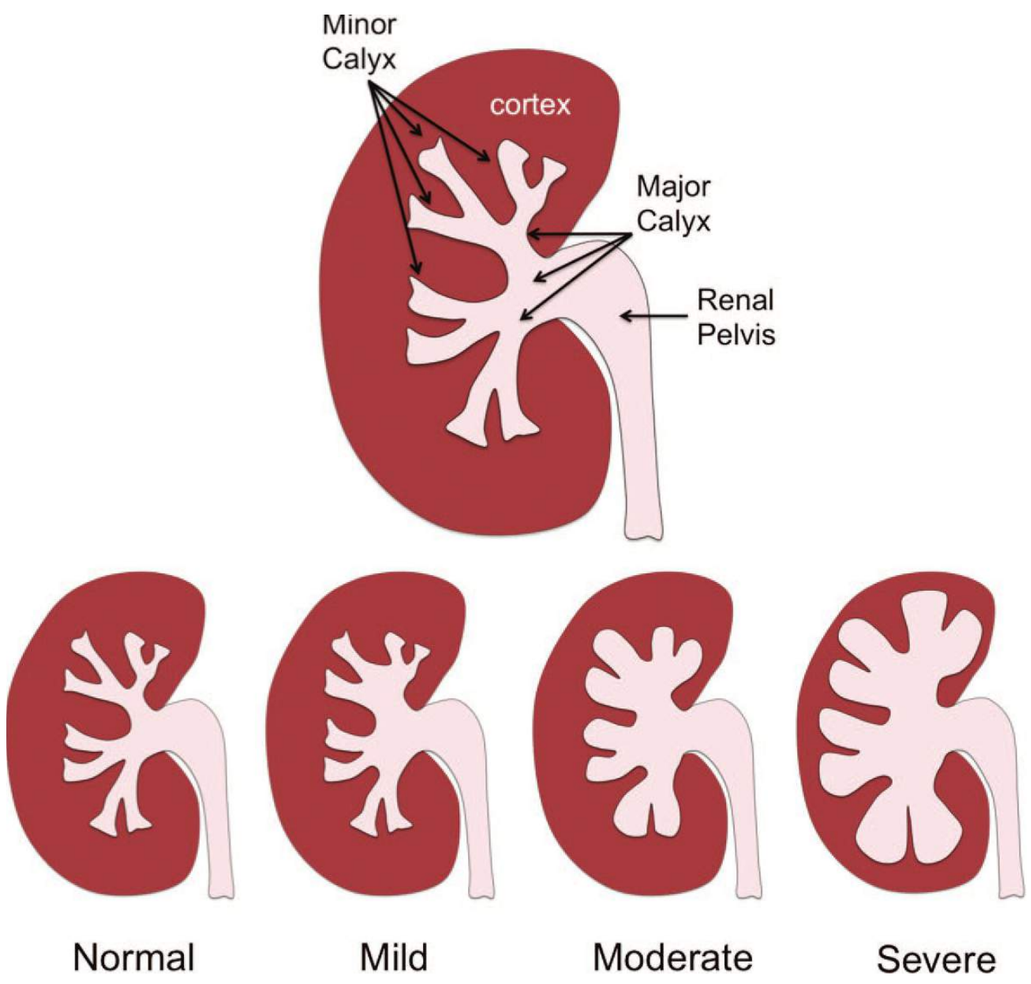


FIGURE 24.87 ■ **Hydronephrosis Grading.** Hydronephrosis is graded based on its extension into the secondary calyces and whether there is accompanying cortical thinning. Mild hydronephrosis does not involve the secondary calyces, moderate does, and severe hydronephrosis is accompanied by cortical thinning. (Illustration courtesy of Robinson M. Ferre, MD.)

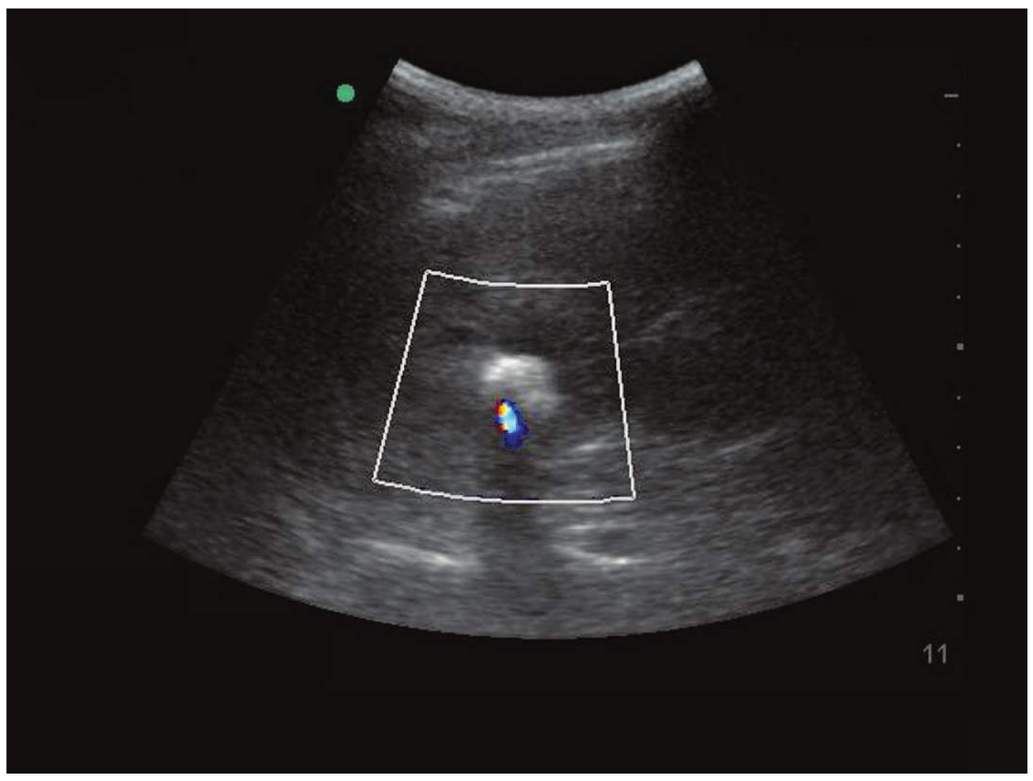


FIGURE 24.88 ■ **Renal Calculi, Twinkle Artifact.** With color flow doppler, uroliths will often demonstrate twinkling artifact deep to their reflective echogenic surface. (Ultrasound contributor: Jeremy S. Boyd, MD.)

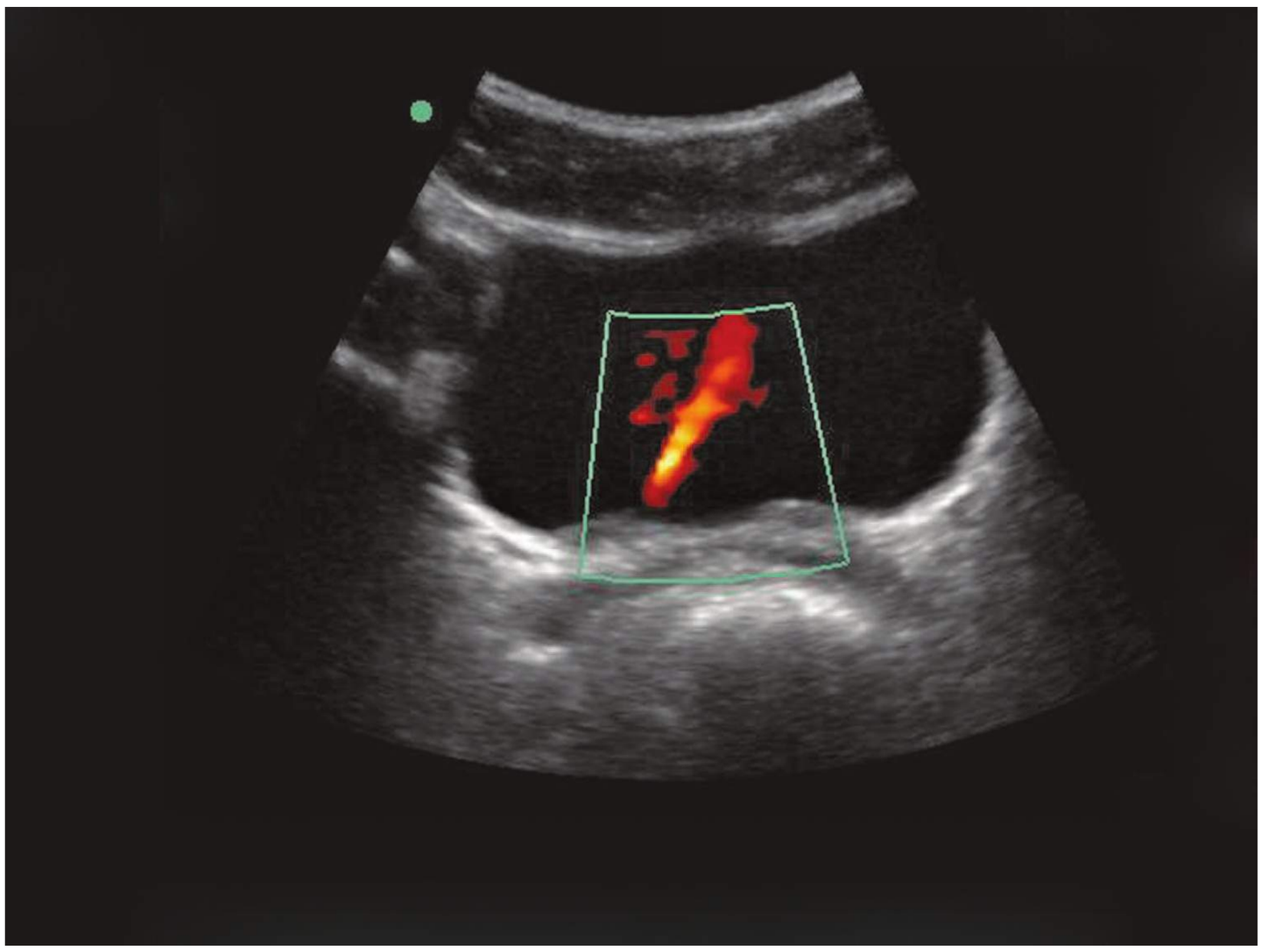


FIGURE 24.89 ■ **Ureteral Jet.** The presence of a ureteral jet confirms the passage of urine into the bladder from a specific ureter. (Ultrasound contributor: Jeremy S. Boyd, MD.)

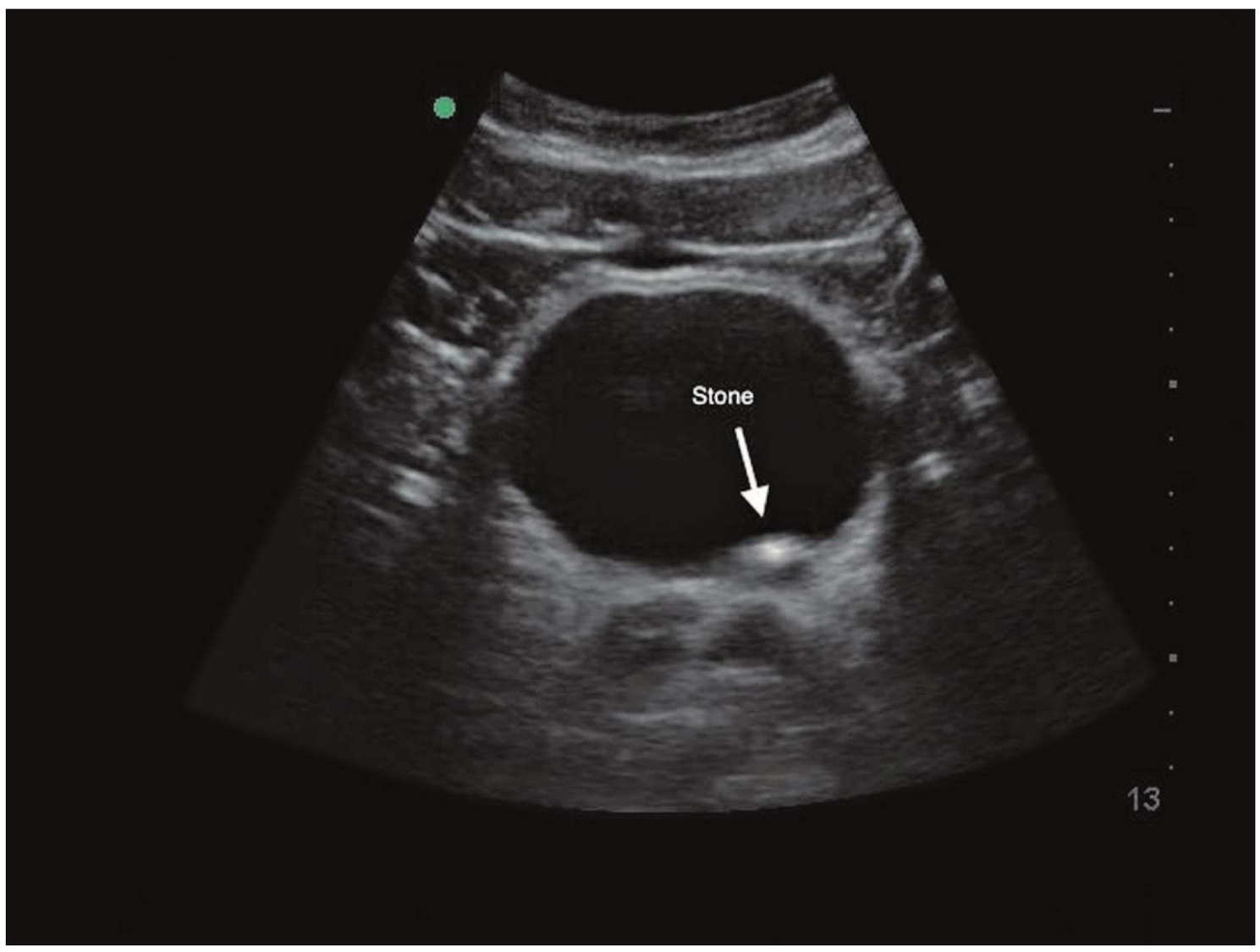


FIGURE 24.90 ■ **Uterovesicular Junction (UVJ) Stone.** Stones may frequently be seen on the bladder views when they are located at the UVJ. (Ultrasound contributor: Robinson M. Ferre, MD.)

Clinical Summary

Ultrasound is frequently used to evaluate the patient presenting with pelvic pain and/or vaginal bleeding, and may aid in the diagnosis of numerous forms of pathology such as urinary retention, molar pregnancies, and ovarian torsion. Pelvic EUS is most commonly used in the evaluation of pregnant patients who present with signs concerning for ectopic pregnancy or miscarriage. The primary goal of pelvic EUS is to evaluate for the presence of an intrauterine pregnancy (IUP) and assess for risk factors that are concerning for an ectopic pregnancy. Ultrasound of pregnant patients is accomplished with two different scanning techniques: transabdominal and transvaginal.

Pregnant patients presenting with abdominal pain or vaginal bleeding during the first trimester must be evaluated for the presence of an ectopic pregnancy. This is commonly accomplished in the ED setting by identifying an IUP in a patient who is at low risk for a heterotopic pregnancy. Patients not receiving infertility treatments with evidence of an IUP can be safely discharged from the ED.

Indications

- Pelvic/abdominal pain
- Vaginal bleeding
- Suspected pregnancy

Required Views for the Emergency Department Pelvic Ultrasound

- Sagittal view
- Transverse view

Equipment: Recommended Transducers for Pelvic Sonography

- Convex array (transabdominal)
- Endocavitary probe (transvaginal)

Patient Positioning

- The patient is supine for transabdominal views.
- The patient is supine and preferably in the lithotomy position for transvaginal views.

Techniques

Transabdominal Sonography: Sagittal View

- With the indicator pointed toward the patient's head, place transducer superior to the symphysis pubis (Fig. 24.91).
- Identify the bladder, uterus, rectum, ovaries, and the rectouterine pouches (pouch of Douglas). Identify an IUP if present (Fig. 24.92).

Transabdominal Sonography: Transverse View

- From the sagittal view, rotate the transducer 90 degrees counterclockwise so the transducer indicator points to the patient's right. The transducer should be superior to symphysis pubis (Fig. 24.93).



FIGURE 24.91 ■ **Transabdominal Sagittal View.** With the indicator pointed toward the patient's head, place the transducer superior to the symphysis pubis. (Photo contributor: Lawrence B. Stack, MD.)

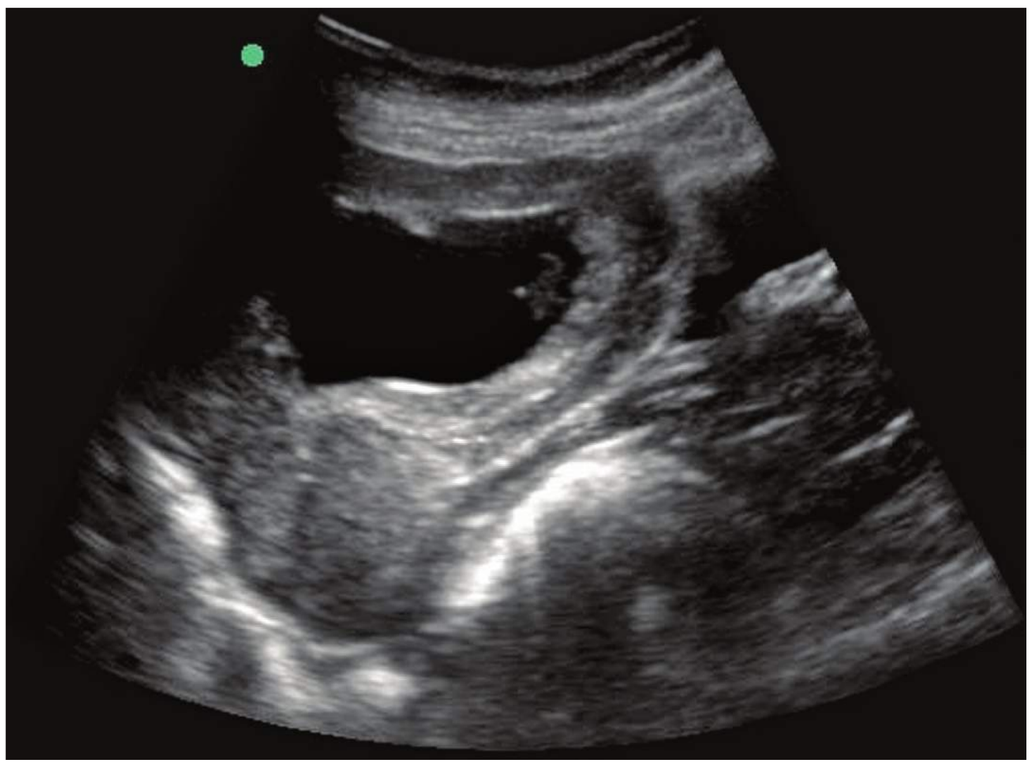


FIGURE 24.92 ■ **Transabdominal Sagittal View.** Gravid uterus showing cervix and vagina posterior to the bladder. (Ultrasound contributor: Robinson M. Ferre, MD.)



FIGURE 24.93 ■ **Transabdominal Transverse View.** From the sagittal view, rotate the transducer 90 degrees counterclockwise so the transducer indicator points to the patient's right. (Photo contributor: Lawrence B. Stack, MD.)

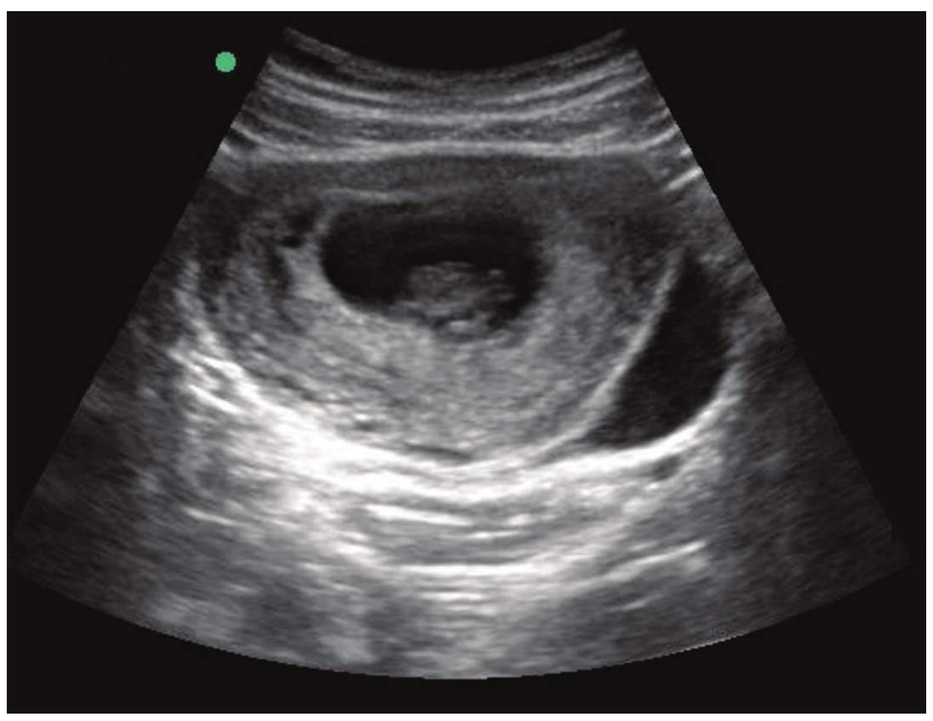


FIGURE 24.94 ■ **Transabdominal Transverse View: Uterus.** First trimester pregnancy showing uterus with gestational sac and fetal pole. Some of the urinary bladder is seen deep and lateral (right on the image) to the uterus. (Ultrasound contributor: Robinson M. Ferre, MD.)

- Identify the bladder, uterus, rectum, ovaries, and the vesicouterine and rectouterine (pouch of Douglas) areas (Fig. 24.94).

Transvaginal Sonography: Sagittal View

- With a condom or commercially available endocavitary probe cover covering the transducer, insert the transducer with the marker directed toward the ceiling. The transducer should be inserted until it rests in the anterior fornix of the vagina (Fig. 24.95).
- Identify the uterus, rectum, ovaries, and rectouterine pouch (Fig. 24.96,). Identify an IUP if present.

Coronal View

- From the sagittal view, rotate the transducer counterclockwise 90 degrees so that the indicator is directed toward the patient's right (Fig. 24.97).
- Identify the uterus, rectum, ovaries, and rectouterine pouch (Fig. 24.98).

Abnormal Findings

- Free intraperitoneal fluid: Anechoic (black) bands of fluid located in the rectouterine pouch (pouch of Douglas).
- Ectopic pregnancy: Most commonly, an ectopic pregnancy is identified as an adnexal mass with an associated empty uterus (Figs. 10.39 and 24.99). At times, an extrauterine gestational sac with or without a fetal pole may be seen. Rarely, a pseudogestational sac (an anechoic fluid

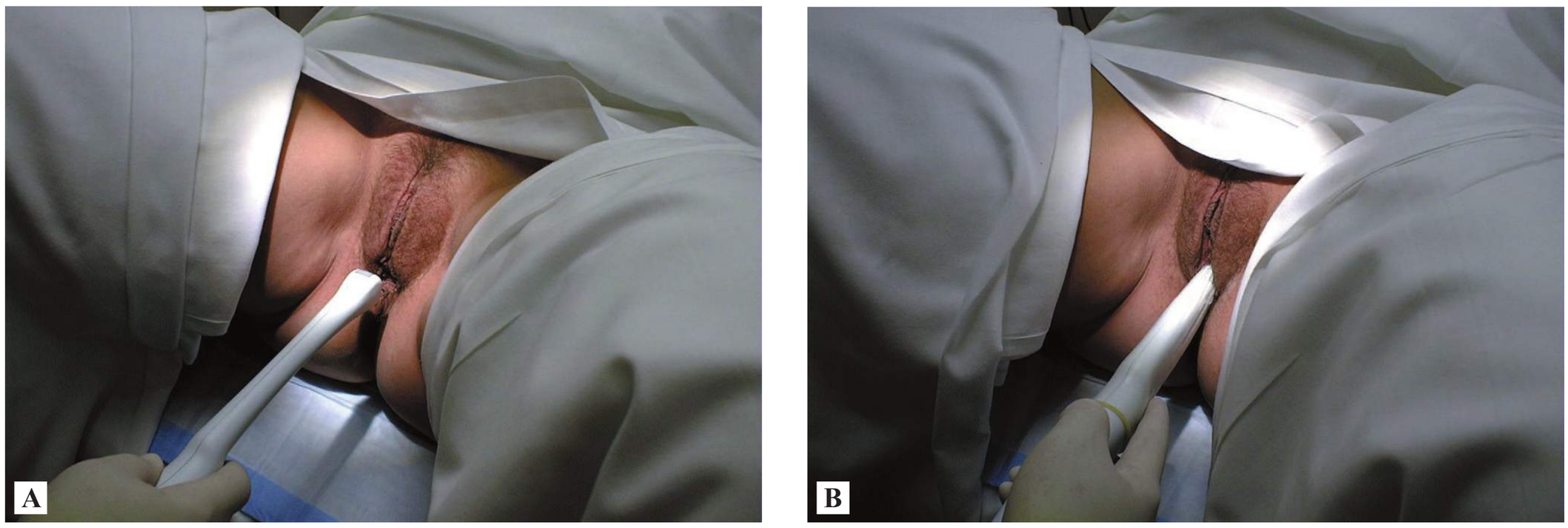


FIGURE 24.95 ■ **Transvaginal Sagittal View.** The transducer is directed toward the anterior fornix in a line through the umbilicus (**A**); it is placed into the vagina (**B**). The probe is advanced gradually. (Photo contributor: Michael J. Lambert, MD.)

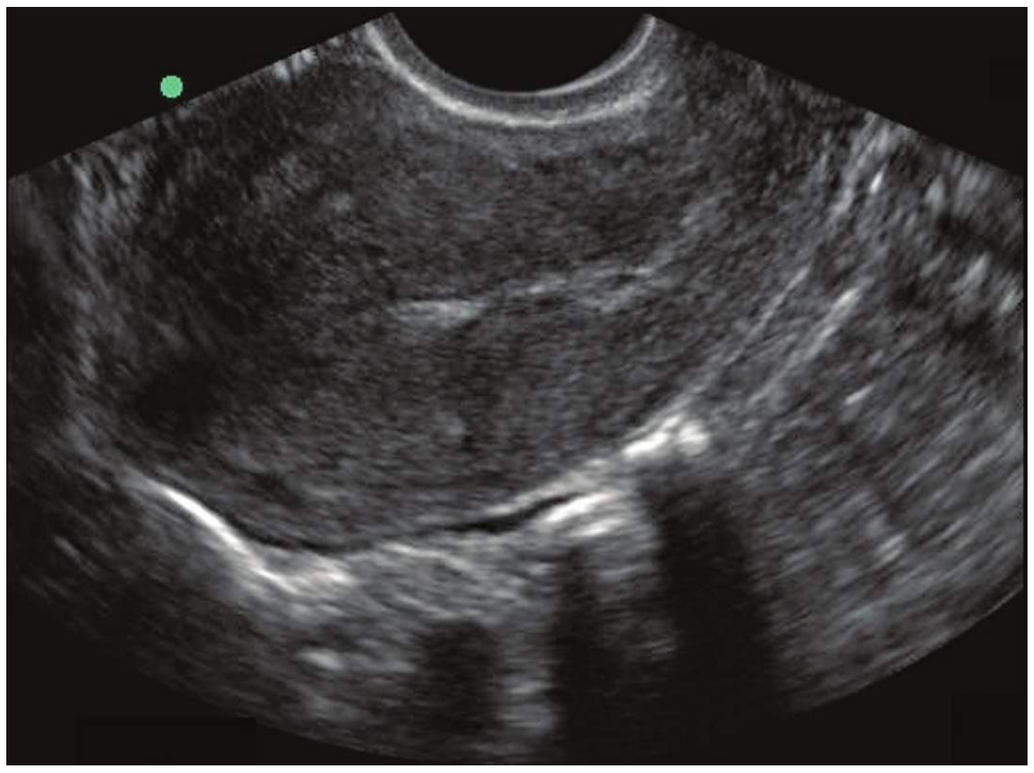


FIGURE 24.96 ■ **Transvaginal: Sagittal View.** The uterus with endometrial stripe. (Ultrasound contributor: Robinson M. Ferre, MD.)

collection less than 1 cm in diameter within the uterus) may mimic an early gestational sac.

- IUP: The first evidence of an IUP is a gestational sac with two thick, concentric echogenic ring within the body or fundus of the uterus. Because this may be confused with a pseudogastional sac, for EUS, the first definitive evidence of an IUP is a gestational sac with a visualized yolk sac and/or fetal pole. A live IUP has both a fetal pole and cardiac activity. (See Figs. 10.37 and 10.38.)
- Abnormal IUP: A fetal pole greater than 7 mm without cardiac activity is diagnostic of a fetal demise. A gestational sac greater than 16 mm without a fetal pole and an enlarged yolk sac greater than 7 mm are concerning for failed IUP, but are not diagnostic. A mean gestational sac diameter of 25 mm or more without a fetal pole is diagnostic of failed IUP. (See Figs. 10.45 and 10.46.)
- Interstitial or cervical ectopic pregnancy: An IUP must be within the body or fundus of the uterus. A gestational sac that is within the cornua or cervix of the uterus, although technically within the uterus, is still considered an ectopic pregnancy. A gestational sac within 5 to 7 mm of the border of the myometrium is concerning for interstitial ectopic pregnancy (see Figs. 10.41 and 10.42).

Pearls

1. A full bladder improves visualization of structures posterior to the bladder in the transabdominal approach. An empty bladder is necessary for transvaginal ultrasound.
2. A small amount of free fluid found in the pouch of Douglas may be physiologic.
3. The bladder is a good landmark to find initially for orientation to the anatomy when scanning transabdominally.
4. When scanning transvaginally in the sagittal view, identify the endometrial stripe from the fundus of the uterus to the cervix. This is accomplished by tilting the probe (anteriorly to posteriorly) while maintaining a sagittal plane of the uterus.

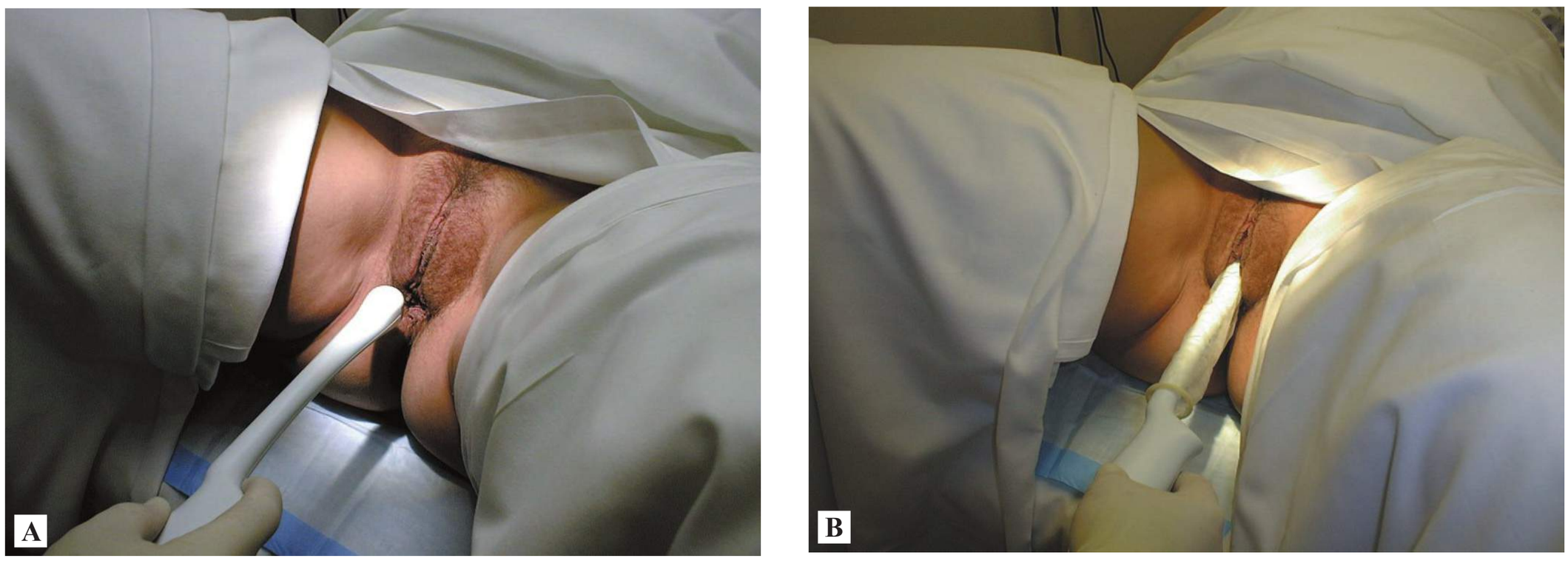


FIGURE 24.97 ■ **Transvaginal: Coronal View.** From the sagittal view, the transducer is rotated counterclockwise 90 degrees (**A**) and directed toward the posterior fornix in a line through the umbilicus (**B**). (Photo contributor: Michael J. Lambert, MD.)

5. Return to the fundus of the uterus in a sagittal view and slowly evaluate the right and then left borders of the uterus in the longitudinal axis.
6. Turn the transducer counterclockwise 90 degrees to enter the coronal plane. The uterus usually appears as an oval. Scan posteriorly to the cervix and then superiorly to the fundus of the uterus to exclude the presence of a bicornuate uterus.
7. If a pregnancy or intrauterine sac is identified with fetal cardiac activity, M-mode, not Doppler, should be used for documentation of fetal heart rate.
8. Evaluate both adnexa/ovaries individually by scanning each side in both coronal and sagittal planes. The ovaries ideally will be located anteromedially to the external iliac vessels. This is often only a guide to their location, and a methodical approach is often required to visualize both ovaries.
9. If the uterus is difficult to identify on TVS, withdraw the transducer slightly. A common error is insertion of the transducer too far, thus bypassing the uterus and imaging only bowel.

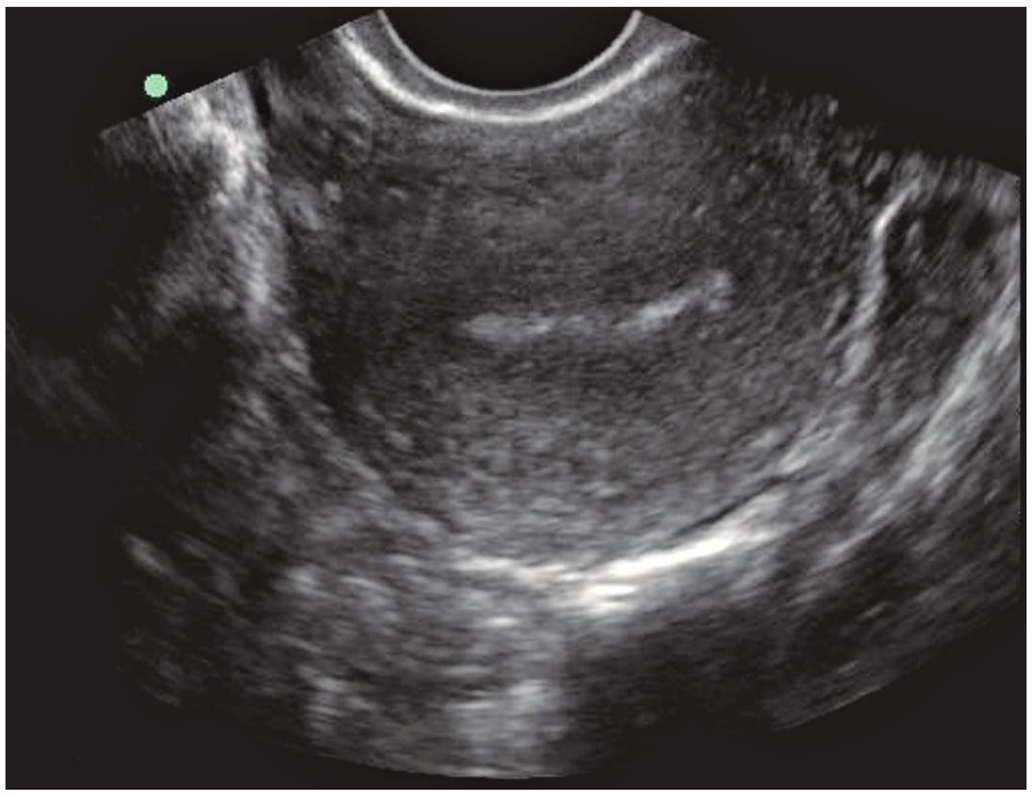


FIGURE 24.98 ■ **Transvaginal: Coronal.** The uterus with endometrial stripe. Left ovary visualized adjacent to the uterus on the right of the image. (Ultrasound contributor: Robinson M. Ferre, MD.)

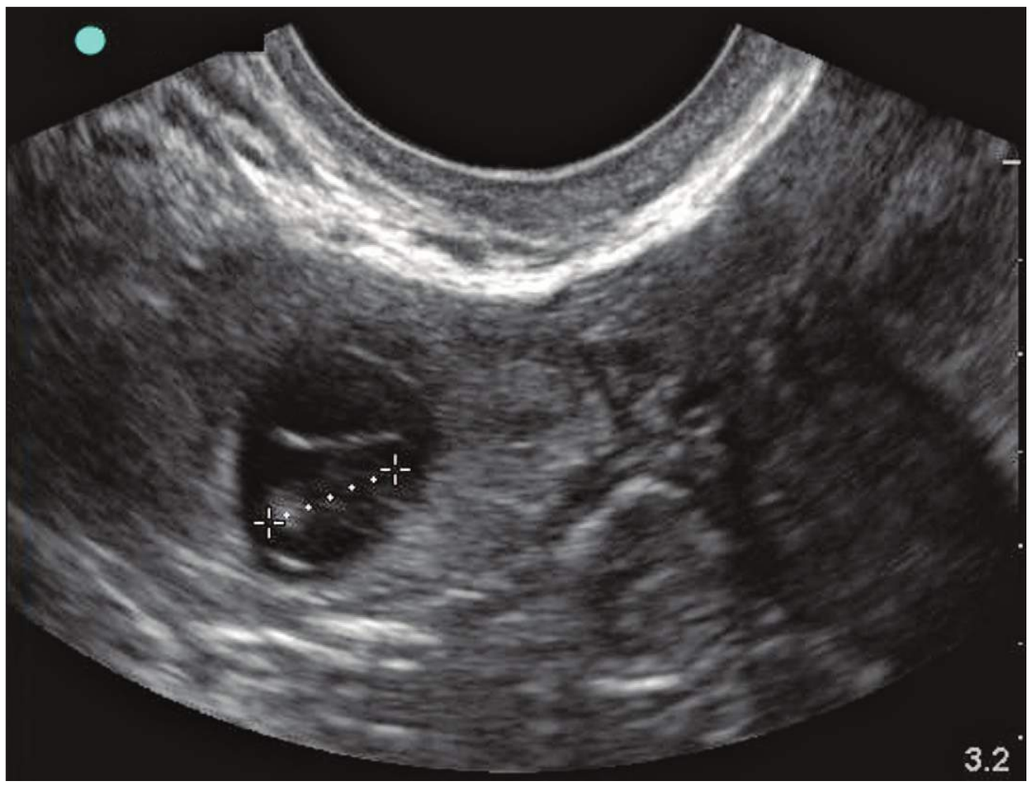


FIGURE 24.99 ■ **Transvaginal: Right Adnexa.** Ectopic pregnancy located in the right adnexa. (Ultrasound contributor: Robinson M. Ferre, MD.)

Clinical Summary

Vascular access is a necessity for evaluation and treatment of many patients in the ED. Traditionally, knowledge of common venous anatomy in experienced hands was the standard method of obtaining venous access. Factors such as obesity, prior access, and volume depletion can increase the difficulty of the conventional approach. This increased difficulty not only leads to failure to obtain access, but also increases the risk of complications.

Ultrasound-guided vascular access reduces the variables associated with the traditional landmark-based approach for central venous access and has become the standard of care. Additionally, ultrasound is useful for both difficult peripheral venous and arterial access.

Indications

- Need for venous access
- Obscured landmarks (obesity, trauma, etc)
- Cardiac arrest

Equipment

- Linear
- Convex

Patient preparation

- Position and prepare the patient as per convention for access and use a sterile sheath for the transducer.
- For central venous access, the patient is supine with the access site in a dependent position.

Technique

There are two main approaches to all venous access.

Short-Axis Approach

- In a transverse view, identify the structures of interest:
  - Vein
  - Corresponding artery
  - Others (nerve, trachea, etc)
- Confirm the vein as the collapsible vascular structure, and trace the vessel to ensure there are no anatomic variants that may disrupt your procedure.
- In the short-axis approach, maintain your transverse view and position the vein in the center of the image.
- Introduce the needle immediately distal to the transducer's edge at approximately 45-degree angle to the patient's skin and perpendicular to the long axis of the probe (Fig. 24.100).
- Maintaining the probe's orientation to the vessel, slide the probe away from the puncture site to maintain visualization of the tip of your needle by observing:
  - The needle tip
  - Tissue movement
  - Ring-down artifact from the needle
- Follow the tip of the needle until it contacts the anterior surface of the vein.
- Continue to advance until a venous flash is seen and the needle has reached the intended depth. If no flash is seen at the appropriate depth in the correct path, slowly withdraw the needle and observe for venous return.
- Continue catheter placement as per common practice.

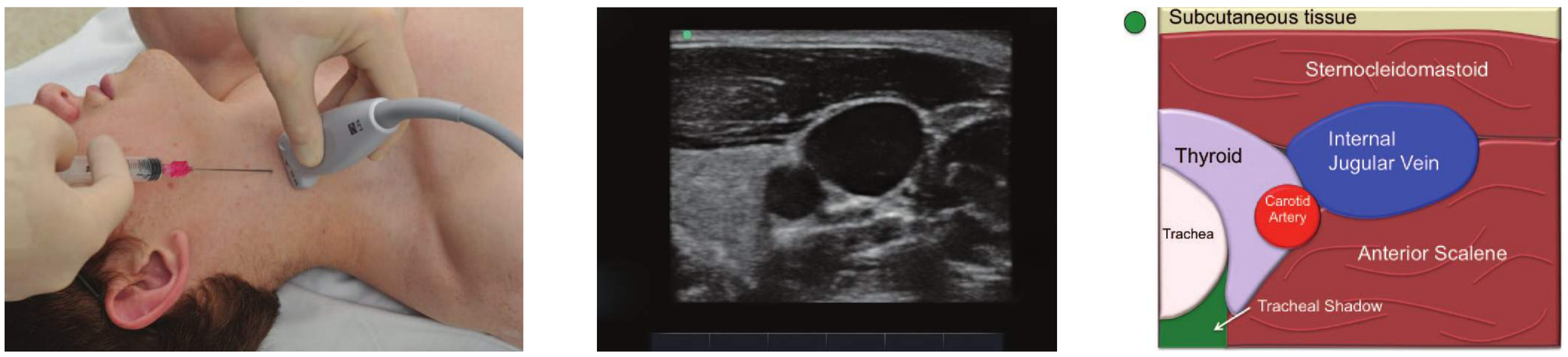


FIGURE 24.100 ■ **Internal Jugular Vein Cannulation, Short-Axis Approach.** The internal jugular vein and carotid artery are identified in cross-section. Introduce the needle immediately distal to the transducer's edge at approximately a 45-degree angle to the patient's skin and perpendicular to the long axis of the probe. (Photo contributor: Lawrence B. Stack, MD; ultrasound contributor: Jeremy S. Boyd, MD; illustration courtesy of Robinson M. Ferre, MD.)

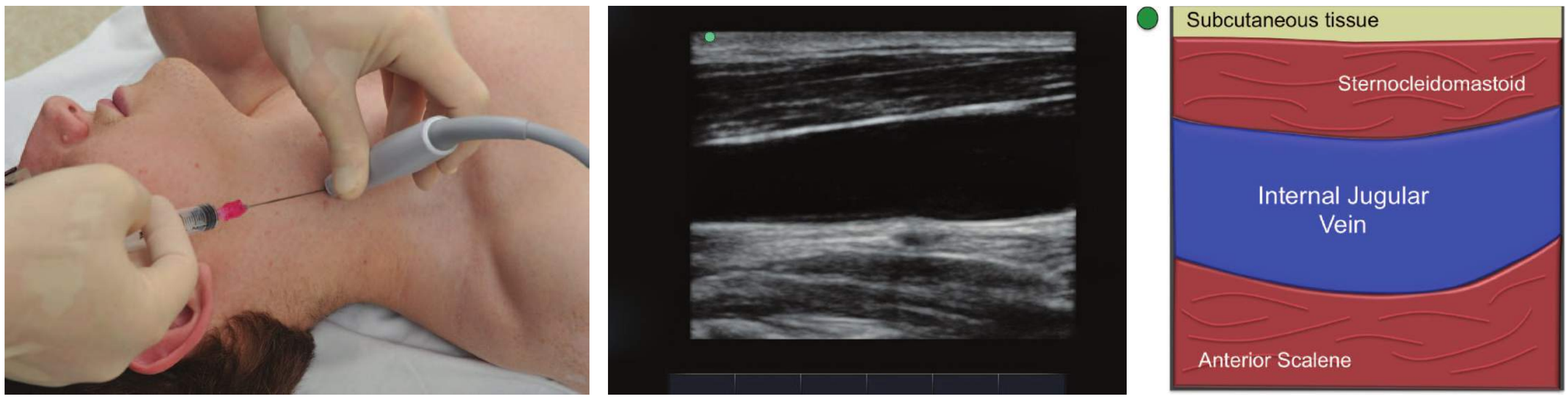


FIGURE 24.101 ■ **Internal Jugular Vein Cannulation, Long-Axis Approach.** The internal jugular vein is identified in its long axis. Introduce the needle immediately distal to the transducer's edge at approximately a 45-degree angle to the patient's skin and parallel to the long axis of the probe. (Photo contributor: Lawrence B. Stack, MD; ultrasound contributor: Jeremy S. Boyd, MD; illustration courtesy of Robinson M. Ferre, MD.)

Long-Axis Approach

- In a transverse view, identify the structures of interest and confirm the vein as the collapsible vascular structure as above.
- Once the vein of interest is identified and centered on the screen, rotate the probe so that the transducer is in the same axis as the vein.
- Introduce the needle at the distal edge of the transducer.
- Direct the needle along the long axis of the probe careful to keep the needle completely in-line with the probe and ultrasound beam. (Fig. 24.101)
- Identify the path of the advancing needle as indicated by observing:
  - The needle tip
  - Tissue movement
  - Ring-down artifact from the needle
- Follow the tip of the needle until it contacts the anterior surface of the vein.
- Continue to advance until a venous flash is seen or the needle has reached the intended depth.
- If no flash is seen at the appropriate depth in the correct path, slowly withdraw the needle and observe for venous return.
- Continue catheter placement as per common practice.

Central Venous Access

Special considerations for central access:

- Typical sites include the internal jugular vein (Figs. 24.100 and 24.101) and the femoral vein, but the subclavian/axillary vein can also be accessed with ultrasound guidance (Fig. 24.102).
- Be careful to maintain sterile precautions including a sterile transducer cover.

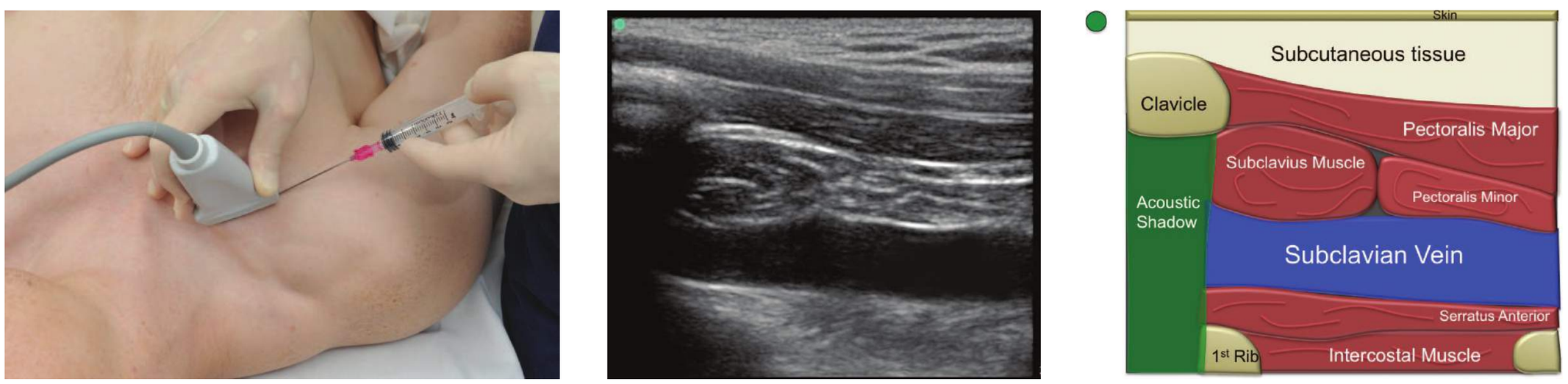


FIGURE 24.102 ■ **Subclavian Vein Cannulation, Long-Axis Approach.** The subclavian vein is identified in its long axis with the proximal edge of the transducer overlying the clavicle. Introduce the needle immediately distal to the transducer's edge at approximately a 30- to 45-degree angle to the patient's skin and parallel to the long axis of the probe. The needle should be visualized throughout its entire course. (Illustration courtesy of Robinson M. Ferre, MD; photo contributor: Lawrence B. Stack, MD.)

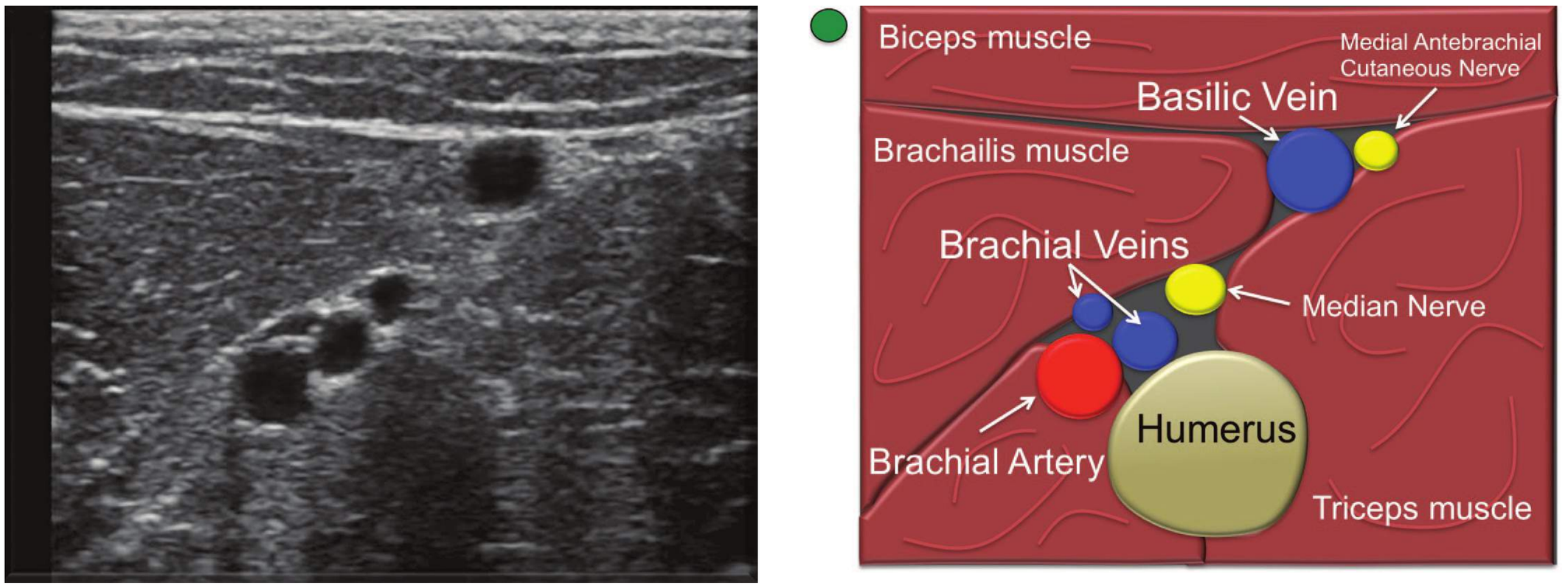


FIGURE 24.103 ■ **Deep Brachial Veins.** Transverse anatomy of the deep veins of the upper arm. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Steven Downie, MD.)

Peripheral Venous Access

Special considerations for peripheral access:

- Apply a tourniquet if needed.
- Ensure that both the needle tip and the plastic catheter are within the vein before advancing the catheter overlying the needle. This often requires advancing the needle just beyond the depth where an initial flash is obtained.
- The veins of the upper arm (basilic, deep brachial, and cephalic) are good targets for ultrasound-guided peripheral venous access (Figs. 24.103 and 24.104).

Pearls

1. Ultrasound identifies the relevant anatomy, but care must be used to follow the typical procedures for venous access.
2. When using a sterile sheath (or glove), use conductive media both inside and outside the sheath. The gel outside the sheath should also be sterile.
3. Venous access may be performed by a single provider or with an assistant.
4. Having the patient cough or perform the Valsalva maneuver may distend the internal jugular vein to facilitate venous access.



FIGURE 24.104 ■ **Peripheral IV Insertion.** Approaches to ultrasound-guided peripheral venous access of the upper extremity: short-axis (**A**), long-axis (**B**), and "backscratcher" approach (**C**). The backscratcher approach facilitates easy access to the more medial basilic vein while allowing improved patient comfort. It is easily performed from the head of the bed. (Photo contributor: Lawrence B. Stack, MD.)

PROCEDURAL GUIDANCE: THORACENTESIS/PARACENTESIS

Clinical Summary

Ultrasound is a source of valuable information that can improve the safety of a wide variety of procedures. The same principles of needle localization and guidance apply. Thoracentesis and paracentesis may be performed using these dynamic "real-time" needle imaging techniques. Alternatively, a static approach may be employed by identifying the location of pathologic fluid and a site for needle insertion. The procedure is then performed in the usual fashion without use of the transducer.

Indications

- Need for thoracentesis or paracentesis.
- Pleural effusion or ascites

Equipment

- Convex
- Linear

Patient Preparation

- For paracentesis, supine position or supine with the head of bed slightly elevated.
- For thoracentesis, the seated upright position is preferred. Lateral decubitus positioning or recumbent position with the ipsilateral arm raised behind the head may also be used.

Technique

Paracentesis

- After positioning patient, ultrasound the abdomen to identify the largest and safest pocket of ascites (Fig. 24.105). The patient may be repositioned to further improve the target.
- After optimizing the image and the patient's positioning, the proceduralist may ultrasound throughout the paracentesis (dynamic technique) or mark the ideal target on the patient and then move forward with the procedure in the usual fashion (static technique).
- The same sterile techniques should be applied with use of ultrasound as per common practice including a sterile probe cover and sterile gel if dynamic imaging is used.
- Continue the procedure as per common practice.

Thoracentesis

- After positioning patient, ultrasound the pleural space posteriorly to identify the largest and safest pocket of pleural fluid (Fig. 24.106). The transducer should be held in the long or sagittal plane with the indicator aimed superiorly. The patient may be repositioned to further improve access.

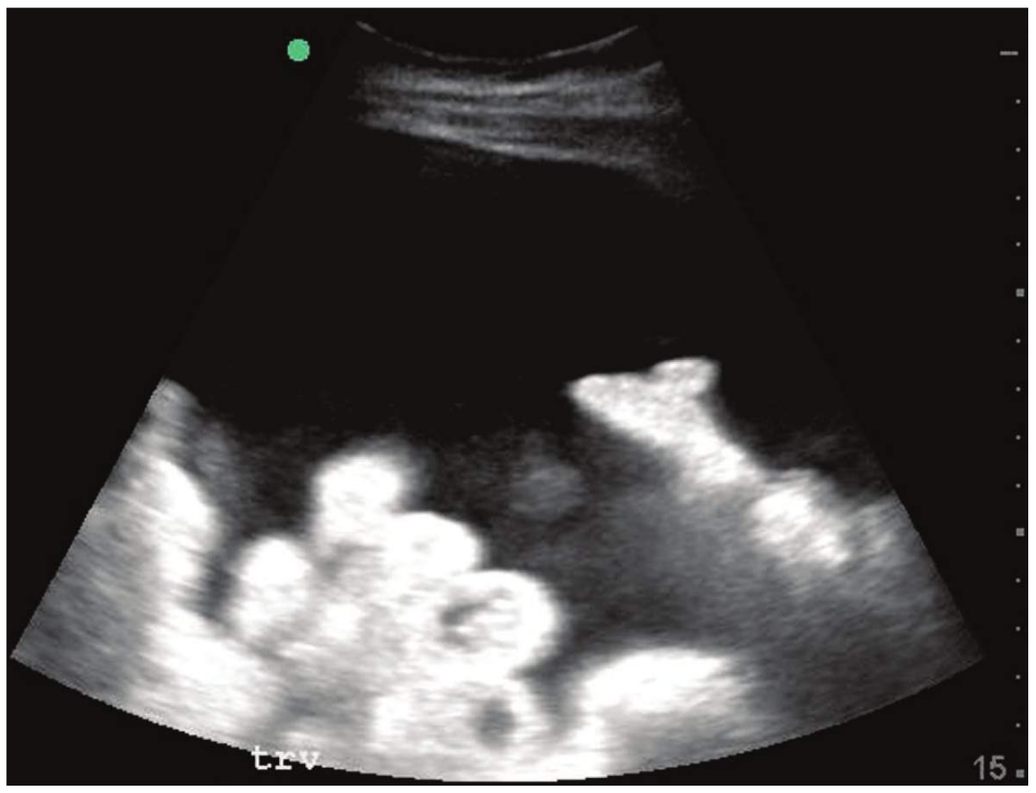


FIGURE 24.105 ■ **Paracentesis: Abdomen, Transverse.** Inferior abdomen with significant ascites. (Ultrasound contributor: Eric Wu, MD.)

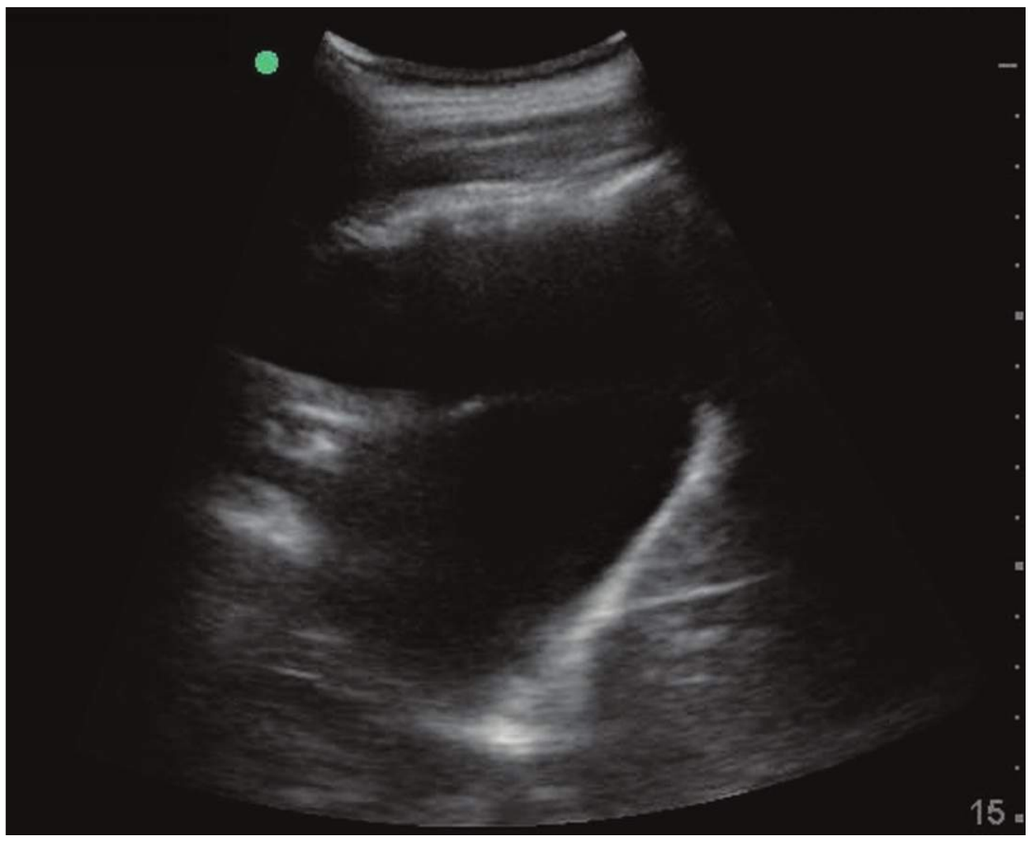


FIGURE 24.106 ■ **Thoracentesis: Right Thorax, Sagittal View.** Large pleural effusion identified prior to being drained via thoracentesis. (Ultrasound contributor: Craig Sheedy, MD.)

- After optimizing the image and the patient's positioning, the proceduralist may ultrasound throughout the paracentesis (dynamic technique) or mark the ideal target on the patient and then move forward with the procedure in the usual fashion (static technique).
- When choosing a procedure site using a static technique, locate a site that would be safe throughout the respiratory cycle and avoid diaphragmatic injury. The diaphragm and subdiaphragmatic contents can be easily visualized by ultrasound during normal respiration.
- The same sterile techniques should be applied with use of ultrasound as per common practice including a sterile probe cover and sterile gel if dynamic imaging is used.
- Continue the procedure as per common practice.

Pearls

1. When performing procedures with dynamic ultrasound guidance, keeping the needle tip in view is crucial to procedure safety.
2. Ultrasound identifies the relevant anatomy, but care must be used to follow the typical procedures for paracentesis/thoracentesis (eg, inserting needle immediately superior to the rib to avoid the intercostal neurovascular bundle).
3. Color flow Doppler imaging should be used to identify abdominal or thoracic wall vessels and prevent vascular injury.

OCULAR ULTRASOUND

Clinical Summary

Emergency physician–performed ocular ultrasound is a rapid and reliable way to evaluate the anterior and posterior chambers of the eye for a variety of emergent conditions, including lens dislocation, intraocular foreign body, and retinal and posterior vitreous detachment. It is particularly useful in the setting of ocular trauma when a direct fundoscopic examination is needed, but difficult or impossible to obtain.

Indications

- Visual loss
- Ocular trauma

Equipment: Recommended Transducers for Ocular Ultrasound

- High-frequency linear array
- It is important to select the "ophthalmic exam setting" or confirm and then adjust the energy output levels of your ultrasound system that are approved for ocular ultrasound.

Patient Positioning

- With the patient lying supine, place a liberal amount of gel on top of the patient's closed eyelid. The orientation marker should be directed toward the patient's right for transverse images and cephalad for sagittal images.
- The probe should exert little to no pressure on the patient's eye. This is accomplished by stabilizing your fingers on the bridge of the nose (when imaging the right eye) or the zygomatic arch (for the left eye) (Fig. 24.107A,B).

Techniques

Transverse and Sagittal Views

- With the indicator pointing toward the patient's right, identify the anterior chamber, lens, posterior chamber, retina, optic nerve, and retrobulbar space (Fig. 24.108).
- The probe should be fanned back and forth to ensure that the entire retina is visualized.
- The overall gain should be increased to assess for evidence of hemorrhage or vitreous detachment in the posterior chamber.
- If assessing for elevated intracranial pressure, the optic nerve sheath diameter (ONSD) should be measured 3 mm from the retina (Fig. 24.109). If measuring the ONSD, it is essential to ensure that the plane of the ultrasound beam is not tangential to the optic nerve.
- The eye should be imaged in both the sagittal and transverse planes to increase the accuracy of detecting pathology, particularly retinal detachment.

Abnormal Findings

- Retinal detachment: A retinal detachment is the separation of the neurosensory retina from the retinal pigment epithelium. The neurosensory retina is a dense neurovascular structure that is highly reflective of ultrasound and will appear as a bright echogenic line on top of the remaining choroid and retinal pigment epithelium (Fig. 24.110). An acute retinal detachment will appear to "wave" if the patient is asked to move their eyes side to side. An older retinal detachment is more rigid and will often lack this mobility.



FIGURE 24.107A, B ■ **Ocular Ultrasound.** Place a liberal amount of gel on top of the patient's closed eyelid. The orientation marker should be directed toward the patient's right for transverse images and cephalad for sagittal images. The probe should exert little to no pressure on the patient's eye. (Photo contributor: Lawrence B. Stack, MD.)

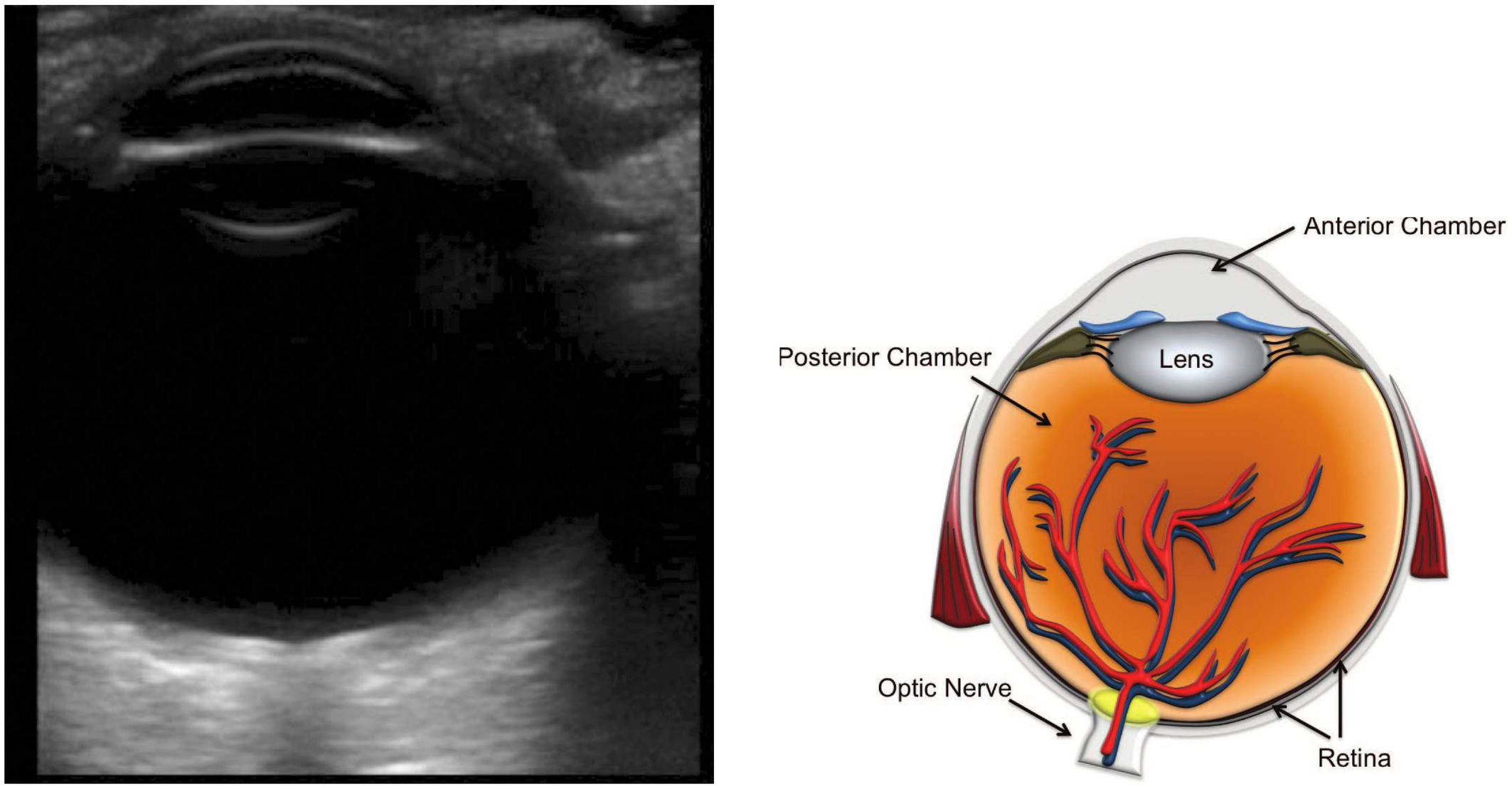


FIGURE 24.108 ■ **Ocular Ultrasound.** Normal eye anatomy. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Robinson M. Ferre, MD.)

- Vitreous detachment: The normally anechoic vitreous will demonstrate various echoes that are usually only seen with increasing the gain. These small, filament-like threads of the detached vitreous body will "swirl" around with eye movements (Fig. 24.111).
- Vitreous hemorrhage: Hyperechoic areas in the vitreous or anterior chamber may represent hemorrhage. It is usually necessary to increase the gain to see posterior vitreous hemorrhage (Fig. 24.112).
- Dilated optic nerve sheath: As intracranial pressure increases, this pressure is transmitted to spinal fluid and along the optic nerve sheath. An ONSD greater than 4.5 mm in children and 5 mm in adults is a sensitive, but not specific, finding for elevated intracranial pressure. The average of three repeated measurements increases accuracy.
- Dislocated lens: The lens is normally positioned in a central location, immediately under the iris. The anterior convex portion of the normal lens is not seen with

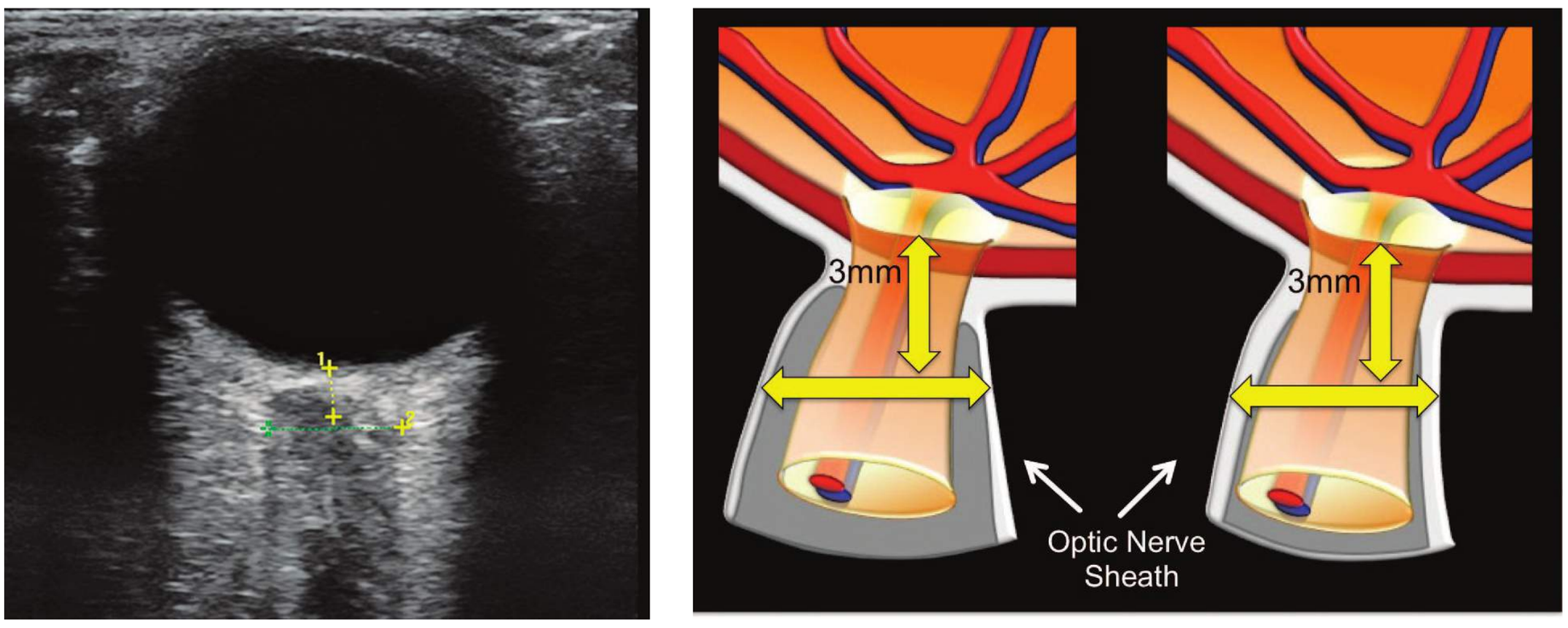


FIGURE 24.109 ■ **Ocular Ultrasound.** The ONSD is measured 3 mm from the retina. The diagram shows representations of a dilated ONSD (left) next to a normal one (right). (Illustration courtesy of Robinson M. Ferre, MD.)

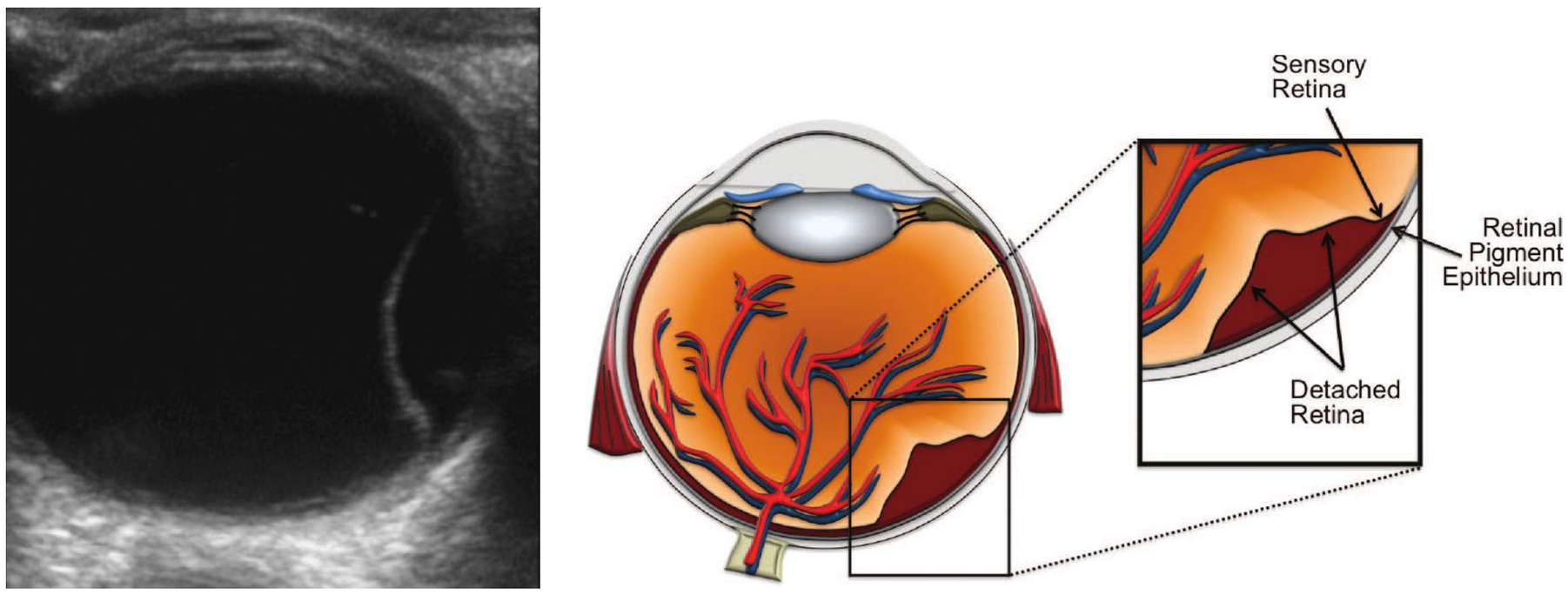


FIGURE 24.110 ■ **Ocular Ultrasound: Retinal Detachment.** A retinal detachment is the separation of the neurosensory retina from the retinal pigment epithelium. (Ultrasound contributor: Robinson M. Ferre, MD; illustration courtesy of Robinson M. Ferre, MD.)

ultrasound, but the posterior concave portion is easily seen. A dislocated lens will be absent from its normal position and a diligent search will reveal its location somewhere in the posterior chamber (Fig. 24.113). A lens subluxation is much more subtle and is visualized as instability of the lens with eye movement.

Pearls

1. Confirmation of an abnormality can often be made by using the unaffected eye as the control. Using multiple planes to image a structure will improve accuracy.
2. Ocular ultrasound often necessitates increasing gain settings to visualize pathologic findings, particularly in the posterior chamber. Overgain the image, then gradually decrease the gain. This will allow small areas of vitreous hemorrhage or the lacy appearance of a vitreous detachment to be readily identified.
3. Both retinal and vitreous detachments are tethered to their origin and can be seen moving when the eye is moved by the patient medially or laterally. Because the retinal is continuous with the optic nerve, a retinal detachment will never appear to cross over the optic nerve head.
4. If it is important to keep the eye free of ultrasound gel, a small Tegaderm™ can be placed over the closed eyelid and the gel can then be placed on top of the Tegaderm™.

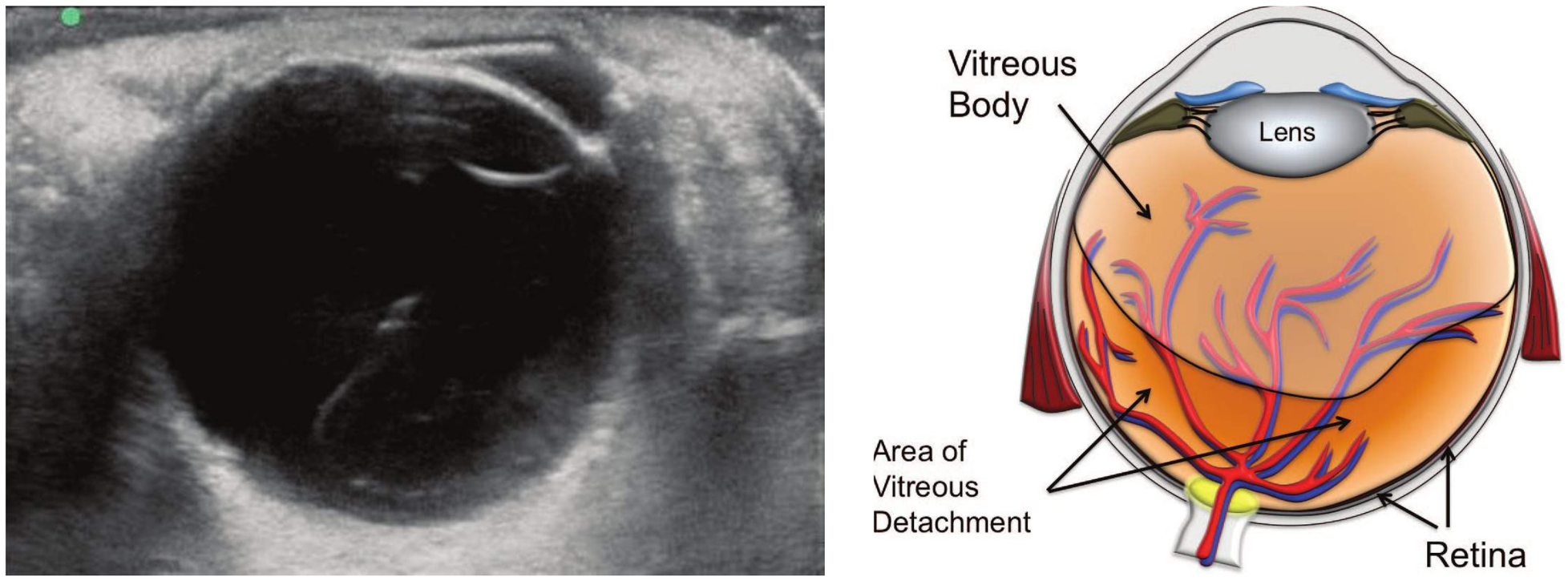
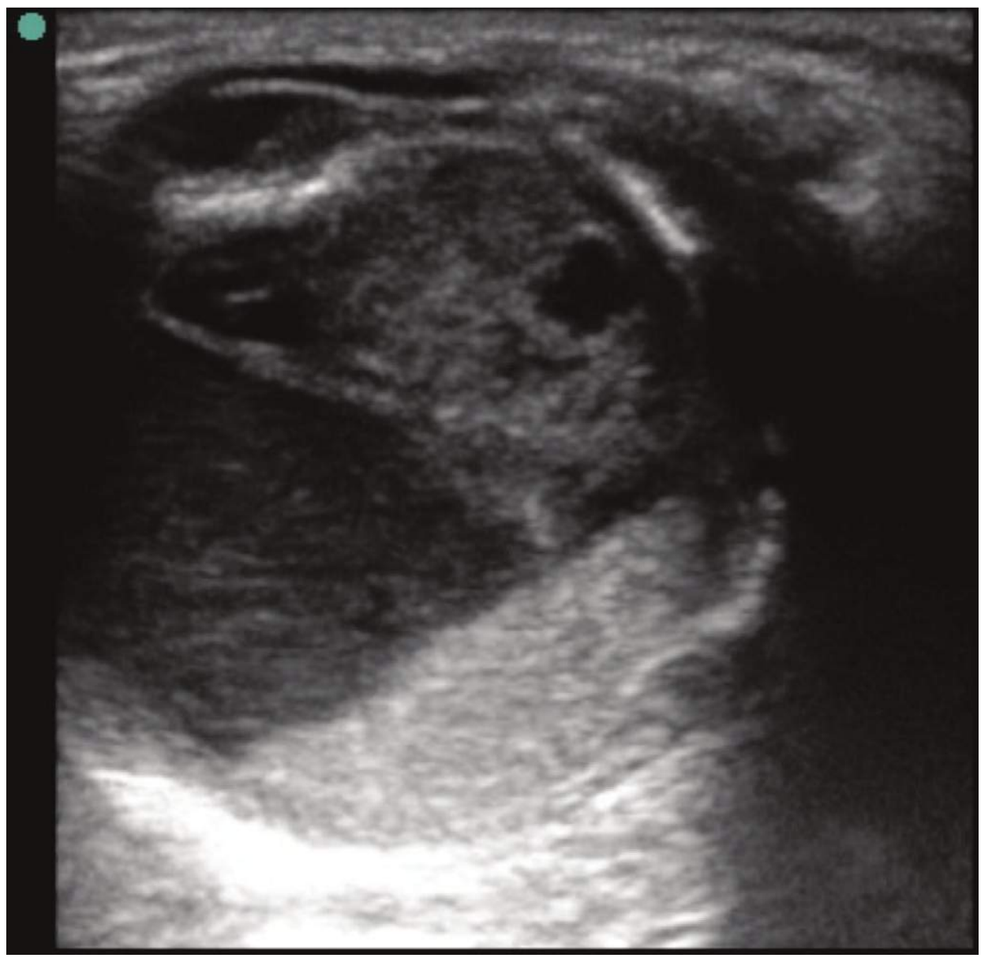


FIGURE 24.111 ■ **Ocular Ultrasound: Vitreous Detachment.** The normally anechoic vitreous will demonstrate various echoes that are usually only seen with increasing the gain. (Ultrasound contributor: Robinson M. Ferre, MD; illustration courtesy of Robinson M. Ferre, MD.)



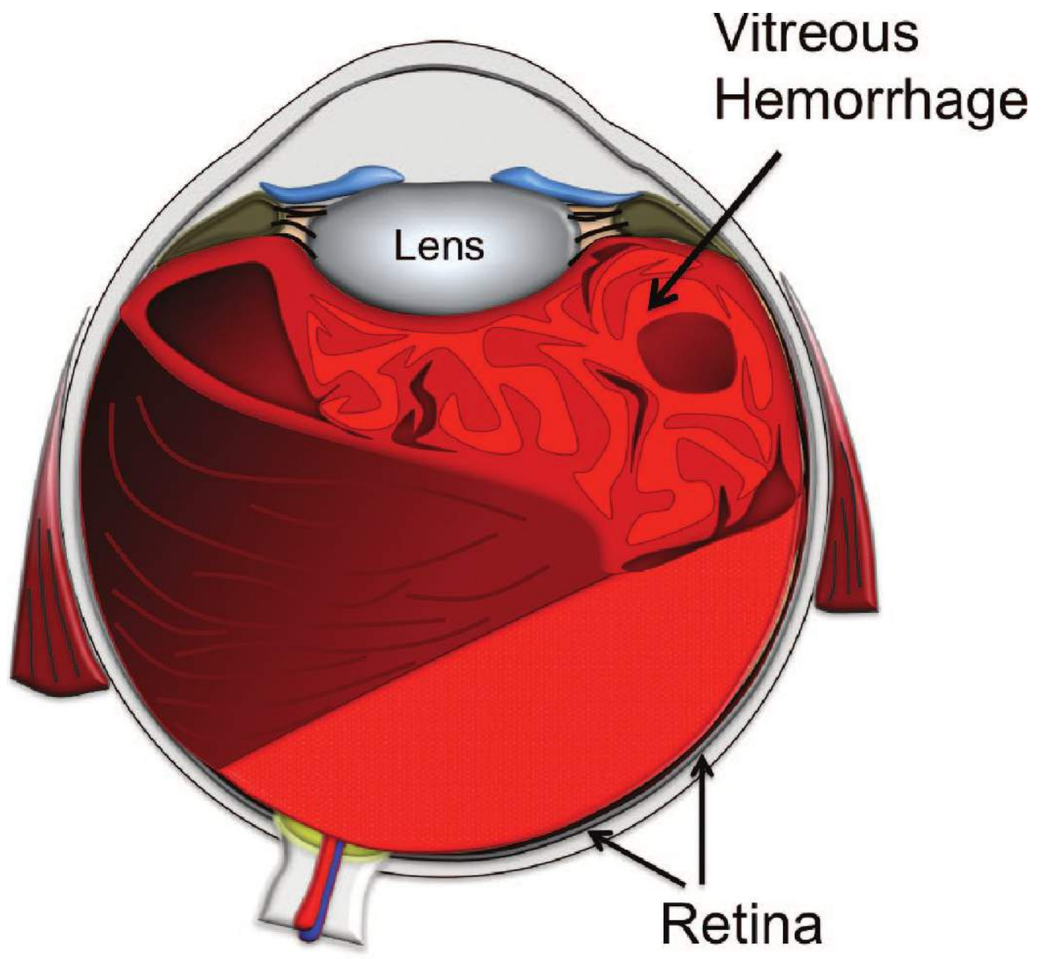
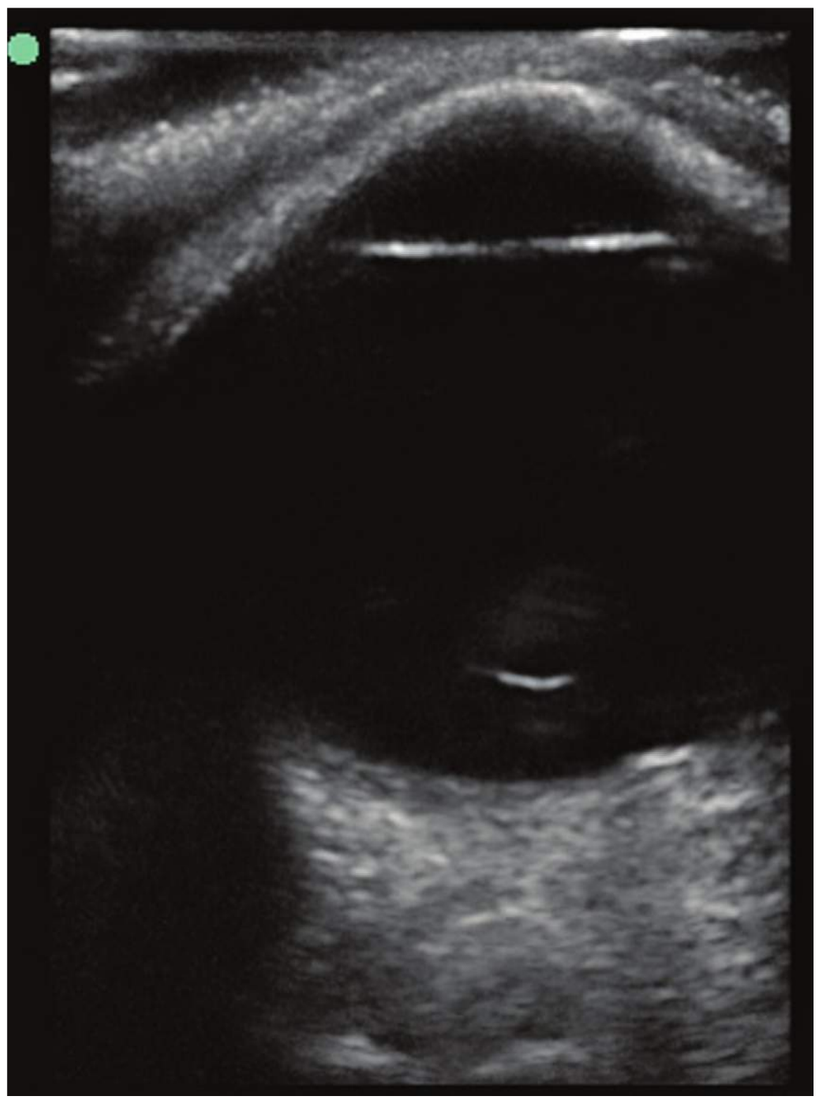


FIGURE 24.112 ■ **Ocular Ultrasound: Vitreous Hemorrhage.** Hyperechoic areas in the vitreous or anterior chamber may represent hemorrhage. It is usually necessary to increase the gain to see posterior vitreous hemorrhage. (Ultrasound contributor: Robinson M. Ferre, MD; illustration courtesy of Robinson M. Ferre, MD.)



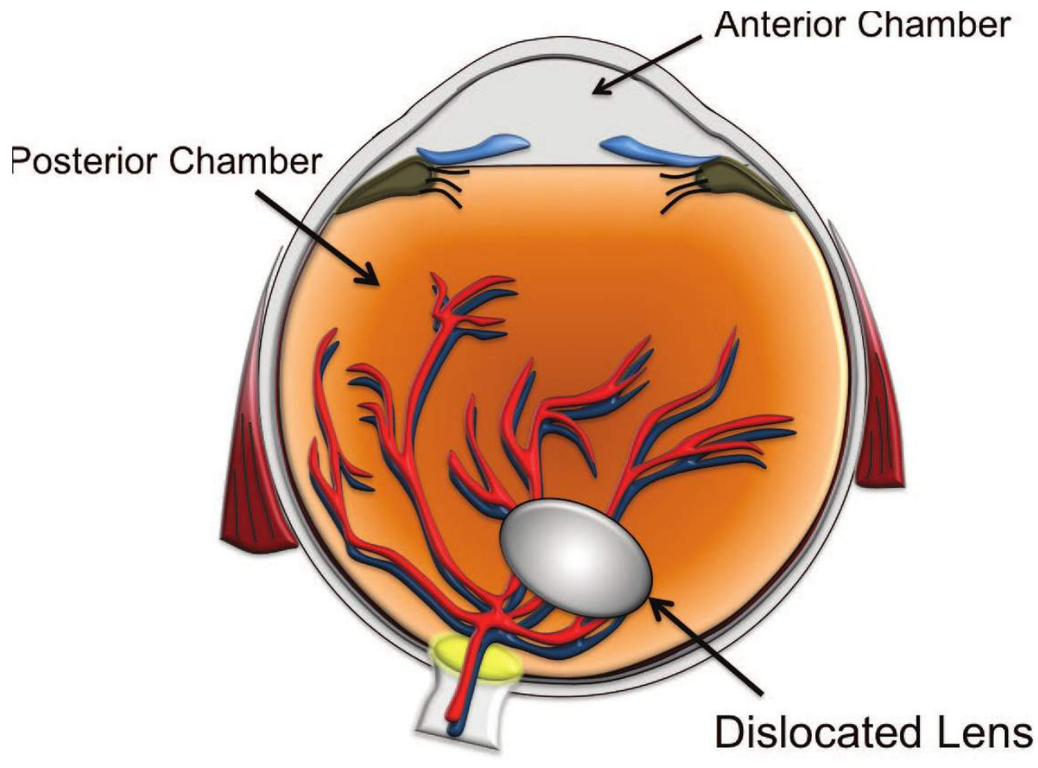


FIGURE 24.113 ■ **Ocular Ultrasound: Lens Dislocation.** A dislocated lens will be absent from its normal position and a diligent search will reveal its location somewhere in the posterior chamber. (Ultrasound contributor: Robinson M. Ferre, MD; illustration courtesy of Robinson M. Ferre, MD.)

SOFT TISSUE ULTRASOUND

Clinical Summary

Soft tissue ultrasound has been shown to increase diagnostic accuracy, speed disposition, and reduce complications of procedures.

Indications

- Suspected fluid collection (abscess, hematoma, etc)
- Localization of subcutaneous foreign body
- Guidance for procedures

Equipment

- High-frequency linear array

Patient Positioning

- Place the patient in a position of comfort that allows maximum exposure of the area of interest.

Technique

- Image the area of interest by gently sweeping the probe across the skin. This should be done in two perpendicular planes. If possible, orient the planes along conventional axes of the body (ie, transverse and longitudinal).
- Identify the dermis, subcutaneous tissue, tendon, muscle, ligament, bone, and relevant neurovascular structures.
- Waterbath evaluation technique (WET): Distal extremities can be placed in a tub of water covering the area of interest and the water can be used as a conductive media for the ultrasound (Fig. 24.114). Ultrasound waves travel well through water, permitting a standoff approach where the probe need not actually touch the skin. This is better tolerated by the patient as no contact with the painful area is necessary and this technique facilitates imaging of extremely superficial structures.
- Apply superficial marking to overlying skin if necessary.

Abnormal Findings

- Edema/cellulitis: Increased interstitial fluid appears similar on ultrasound regardless of the cause (ie, cellulitis, lower extremity–dependent edema, etc). Initially, increased echogenicity and loss of definition of the tissue is noted in the subcutaneous fat. This obscures deeper structures such as muscle and bone. As the condition progresses, fluid is noted to accumulate diffusely in a reticular pattern, creating a cobblestone appearance (Fig. 24.115).
- Abscess: An abscess appears as a focal fluid accumulation, often with a significant amount of increased echogenicity in the surrounding tissues. The fluid may appear anechoic or have mixed complex echoes (Fig. 24.116). If drainage will be attempted, the depth and location of the abscess, any associated tracts, and other important and relevant adjacent structures should be noted and marked if appropriate (vessels, nerves, etc).



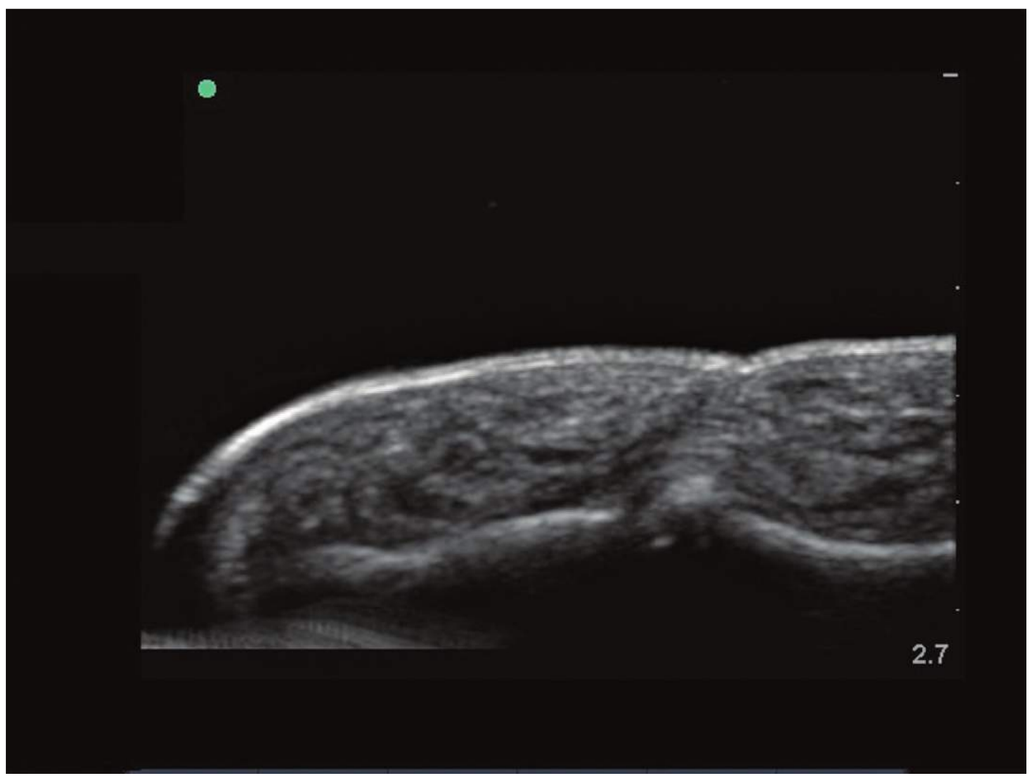


FIGURE 24.114 ■ **Waterbath Evaluation of the Extremity.** Distal extremities can be placed in a tub of water covering the area of interest and the water can be used as a conductive media for the ultrasound. The ultrasound image shows the volar aspect of the distal phalanx of the middle finger obtained using the waterbath technique. (Photo contributor: Lawrence B. Stack, MD; ultrasound contributor: Jordan Rupp, MD.)

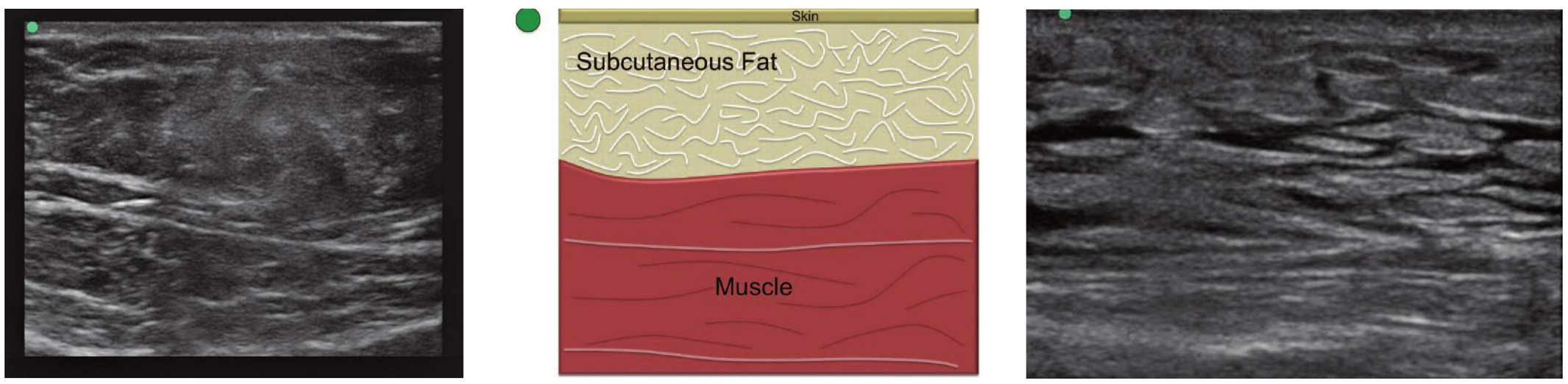


FIGURE 24.115 ■ **Soft Tissue Ultrasound: Cellulitis.** Initially, increased echogenicity and loss of definition of the tissue are noted in the subcutaneous fat (left). As the condition progresses, fluid is noted to accumulate diffusely in a reticular pattern creating a cobblestone appearance (right). (Ultrasound contributor: Robinson M. Ferre, MD; illustration courtesy of Robinson M. Ferre, MD.)

- Joint effusion: Ultrasound can readily identify fluid accumulation within joints. A distended joint capsule will be visualized with anechoic or heterogeneous echogenic fluid (Fig. 24.117). Thickened synovial lining may be noted as well. Drainage can be assisted either statically, locating and marking area of maximal effusion, or dynamically, using ultrasound to guide the needle throughout the entire procedure.
- Subcutaneous foreign body: Most foreign bodies are visible using ultrasound. The foreign body's appearance depends on the shape, size, and composition of the object. Careful, slow passes should be made over the likely location, noting any abnormal object or defect in tissue (Fig. 24.118). EUS can assist in removal by either a static approach, marking the skin overlying the location, or dynamic technique guiding removal during real-time ultrasound. Care should be taken to identify any structures damaged by the foreign body or that may complicate removal.

Pearls

1. Always scan the area of interest in two planes. This will help to identify the exact location of the abnormality and exclude artifacts.
2. If needed, use the surrounding, unaffected tissue or other extremity as a control.
3. To optimize the image, reduce the depth to the appropriate level and increase the frequency of the probe. The dermis and other extremely superficial structures (<0.5 cm) may not be well visualized due to near-field artifact. Consider the use of a standoff pad or water bath if feasible.
4. For procedures requiring sterile precautions, a sterile sheath should be used to cover the transducer for dynamically guided procedures. Many sterile ultrasound sheaths are commercially available, although, less preferably, a sterile glove or transparent adherent dressing (eg, Tegaderm™) can be used. Sterile ultrasound gel or another sterile liquid (eg, Betadine or chlorhexidine) should be used as a conductive medium.

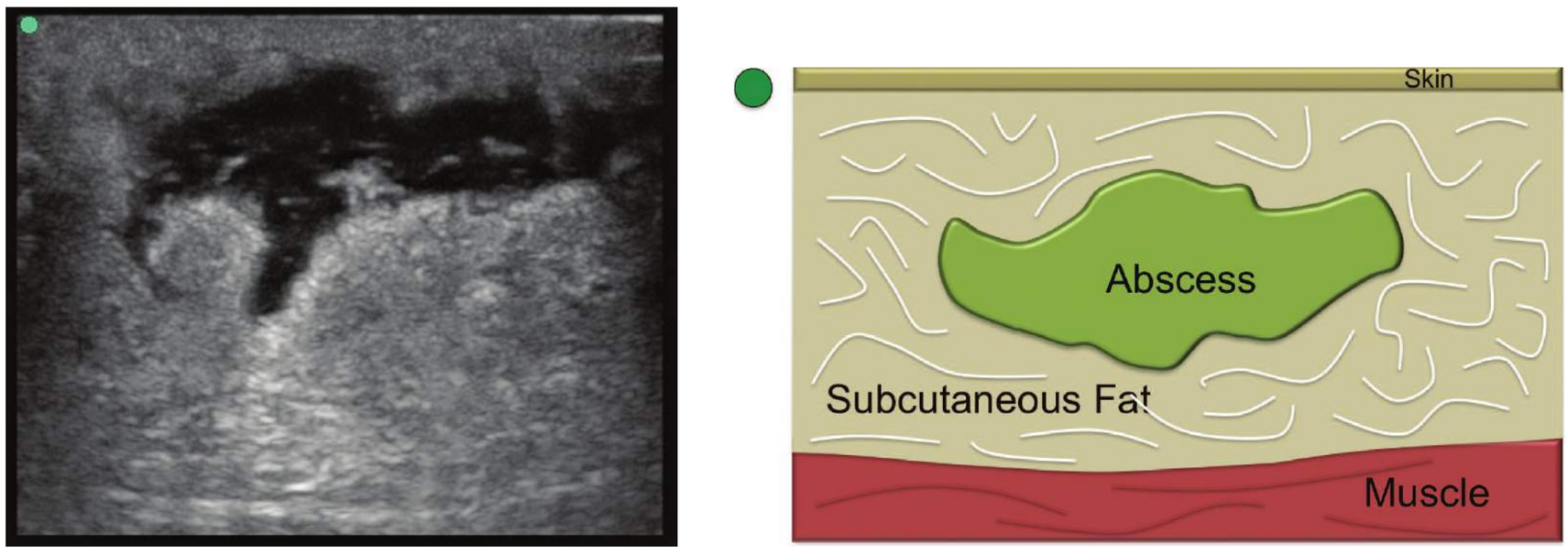


FIGURE 24.116 ■ **Soft Tissue Ultrasound: Abscess.** An abscess appears as a focal fluid accumulation, often with a significant amount of increased echogenicity of the surrounding tissues. The fluid may appear anechoic or have mixed complex echoes. (Ultrasound contributor: Robinson M. Ferre, MD; illustration courtesy of Robinson M. Ferre, MD.)

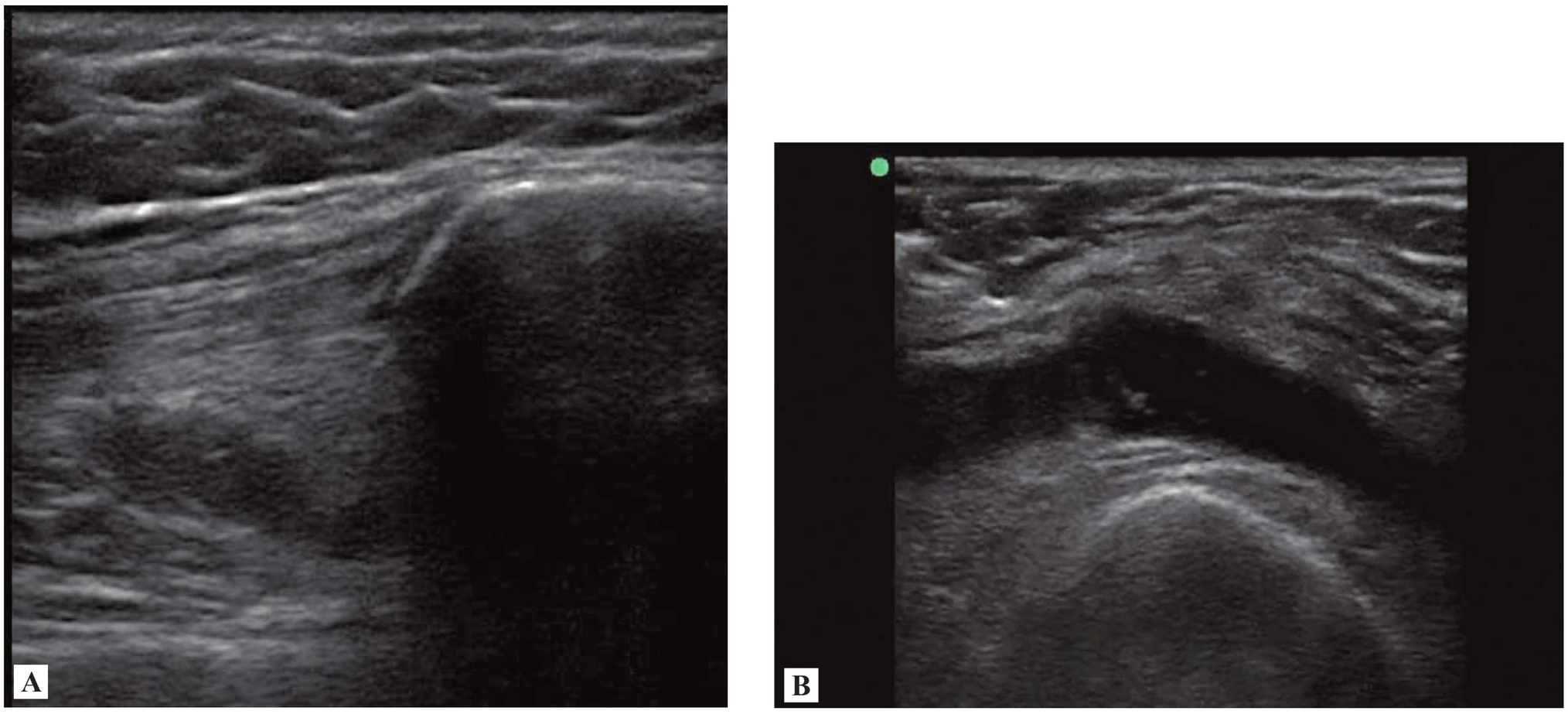


FIGURE 24.117 ■ **Soft Tissue Ultrasound: Joint Effusion.** A knee effusion is seen in sagittal (**A**) and transverse (**B**) views. (Ultrasound contributor: Robinson M. Ferre, MD.)

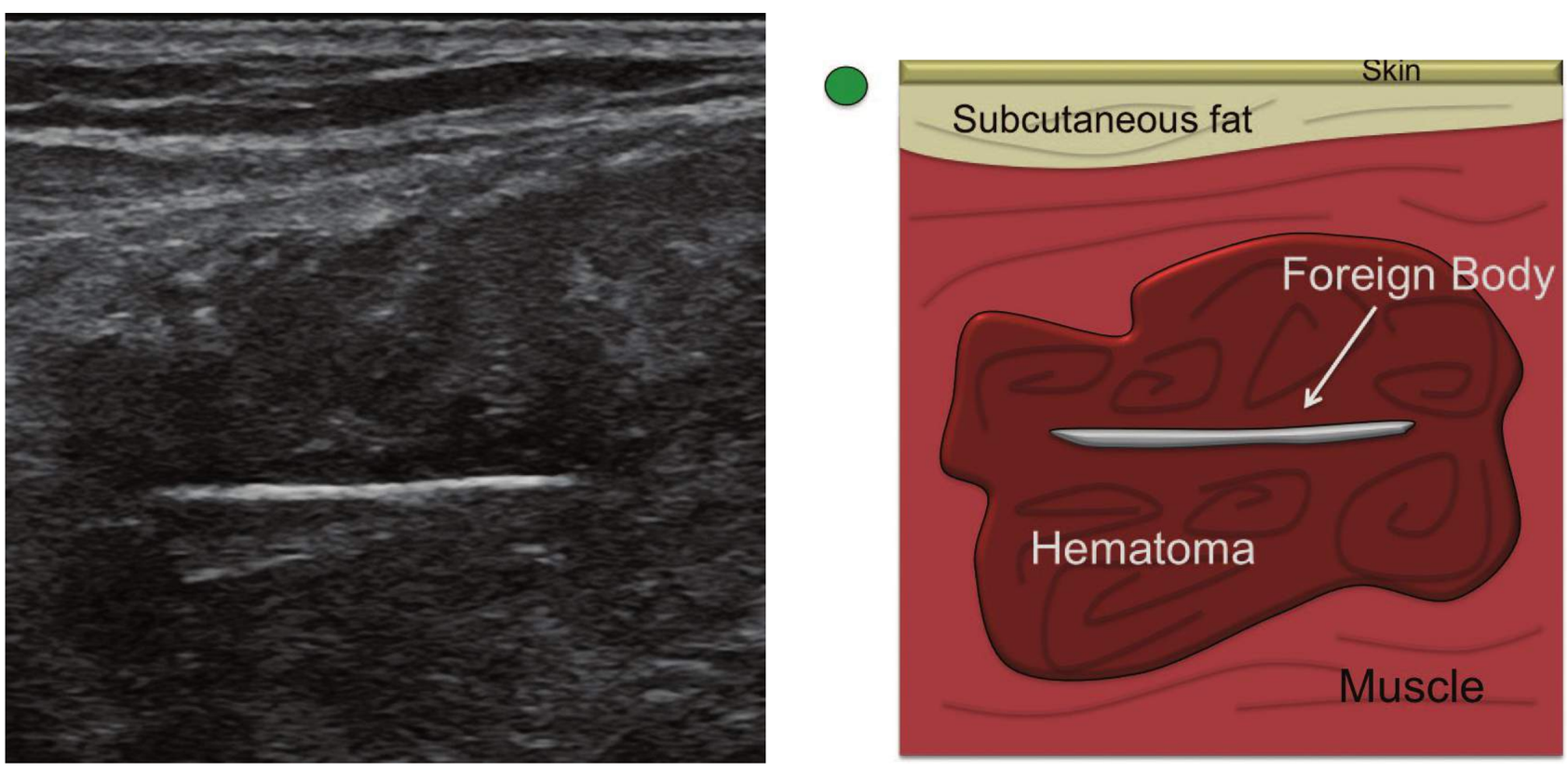


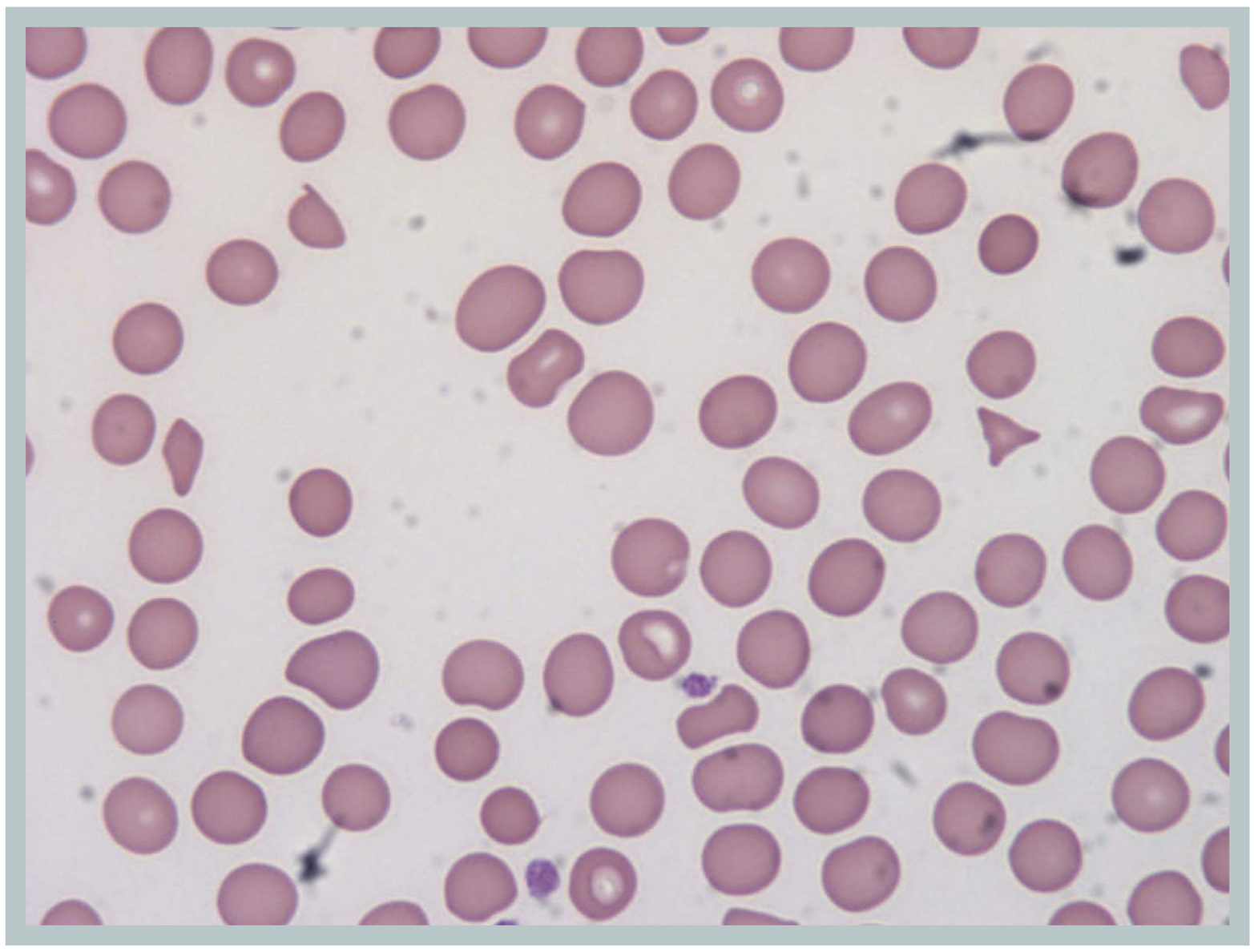
FIGURE 24.118 ■ **Soft Tissue Ultrasound: Foreign Body.** An echogenic, linear foreign body is visualized in the superficial soft tissues with surrounding hematoma. (Illustration courtesy of Robinson M. Ferre, MD; ultrasound contributor: Robinson M. Ferre, MD.)

Chapter 25

MICROSCOPIC FINDINGS

Brett Bechtel

Scan QR code or go to www.ematlas.com for video related to chapter



Schistocytes. Several fragmented red blood cells are seen in this smear in a patient with microangiopathic hemolytic anemia. (Photo contributor: James P. Elrod, MD, PhD.)

The author wishes to thank Diane M. Birnbaumer for her contributions to prior editions.

Uses

To evaluate for the presence of cells, casts, and crystals.

Materials

Freshly collected urine specimen, centrifuge, graduated centrifuge tubes, glass microscope slide, and coverslip.

Method

1. Pour 10 mL of freshly collected urine into a graduated centrifuge tube.
2. Centrifuge at ×400 to ×450 gravity for 5 minutes.
3. Decant 9 mL of supernatant, leaving 1 mL in the tube.
4. Resuspend the centrifuged pellet in the remaining 1 mL of urine by stirring with a pipet.
5. Place one drop of resuspended urine on a glass microscope slide.
6. Overlay with a coverslip.
7. Examine initially using scanning ×10 power, emphasizing the periphery of the coverslip, since urinary elements tend to gather at the edges.
8. Switch to ×40 power to focus on specific urinary elements such as cells, casts, and crystals. Use ×100 power as needed for specific identification.

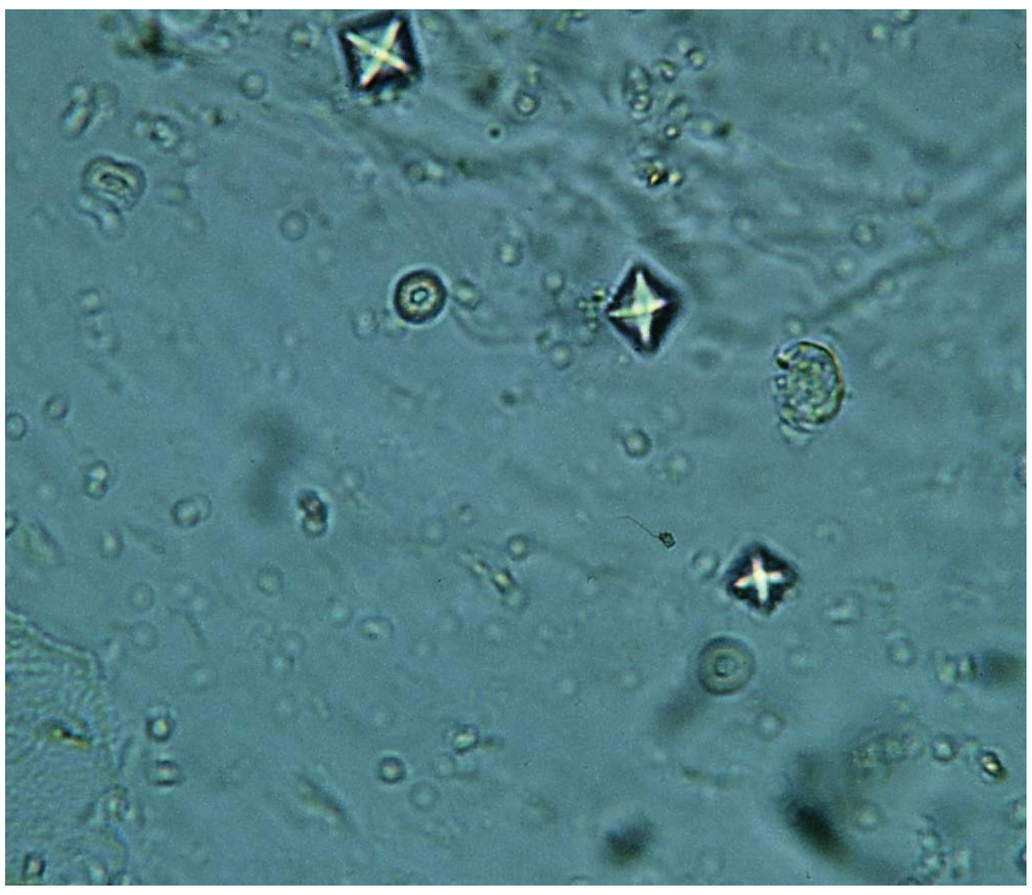


FIGURE 25.1 ■ **Calcium Oxalate Crystals.** Calcium oxalate crystals come in two shapes. The classically described octahedral, or envelope-shaped, crystals are made of calcium oxalate dihydrate. Calcium oxalate monohydrate crystals are needle-shaped. They are seen in acid or neutral urine. They may be found in the urine of patients with ethylene glycol ingestion. In addition, the urine of patients with ethylene glycol ingestion may also fluoresce under a Wood lamp. (Reproduced with permission from Strasinger SK, Di Lorenzo MS. Urinalysis and Body Fluids. 4th ed. Philadelphia: F.A. Davis Company; 2001, p. 97.)

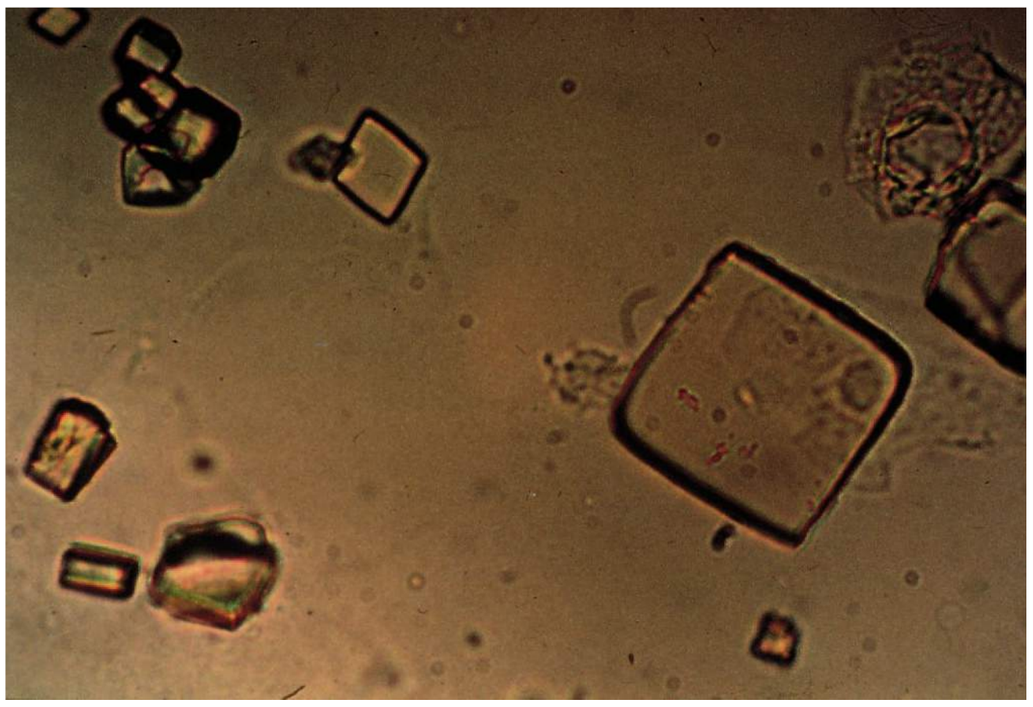


FIGURE 25.2 ■ **Uric Acid Crystals.** Uric acid crystals often have a yellow hue and a variety of sizes and shapes. They are found in acidic urine. (Reproduced with permission from Strasinger SK, Di Lorenzo MS. Urinalysis and Body Fluids. 5th ed. Philadelphia: F.A. Davis Company; 2008, p. 114.)

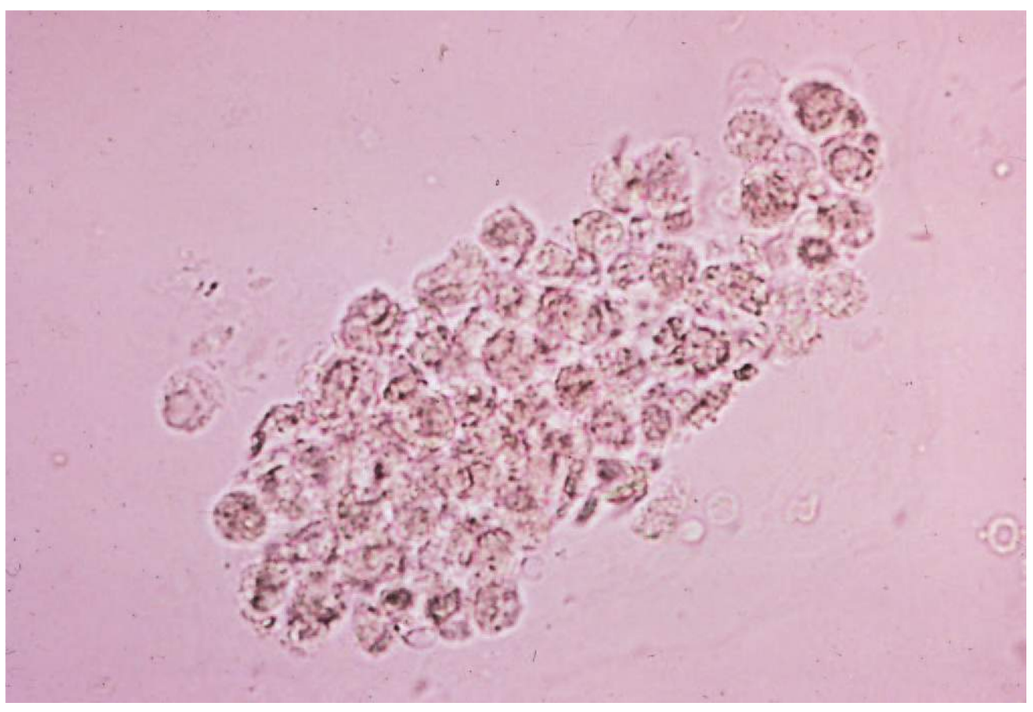


FIGURE 25.3 ■ **White Blood Cell Casts.** Usually two to three cells in width, white blood cell casts are indicative of upper urinary tract infection such as pyelonephritis. (Photo contributor: The American Society of Clinical Pathologists. © American Society of Clinical Pathologists.)

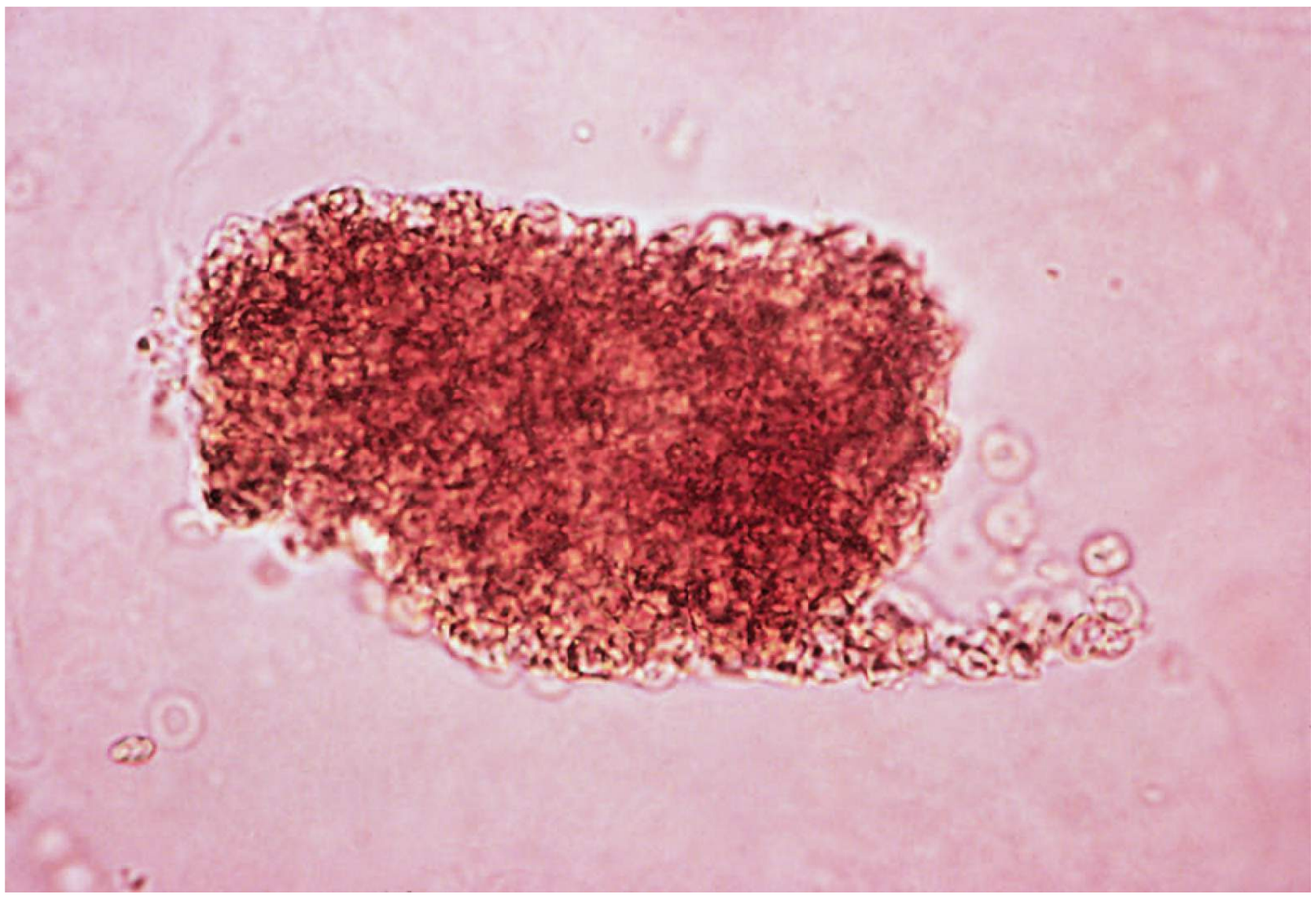


FIGURE 25.4 ■ **Red Blood Cell Casts.** Red blood cell casts range from 3 to 10 cells in width and are seen in glomerulonephritis. (Photo contributor: The American Society of Clinical Pathologists. © American Society of Clinical Pathologists.)

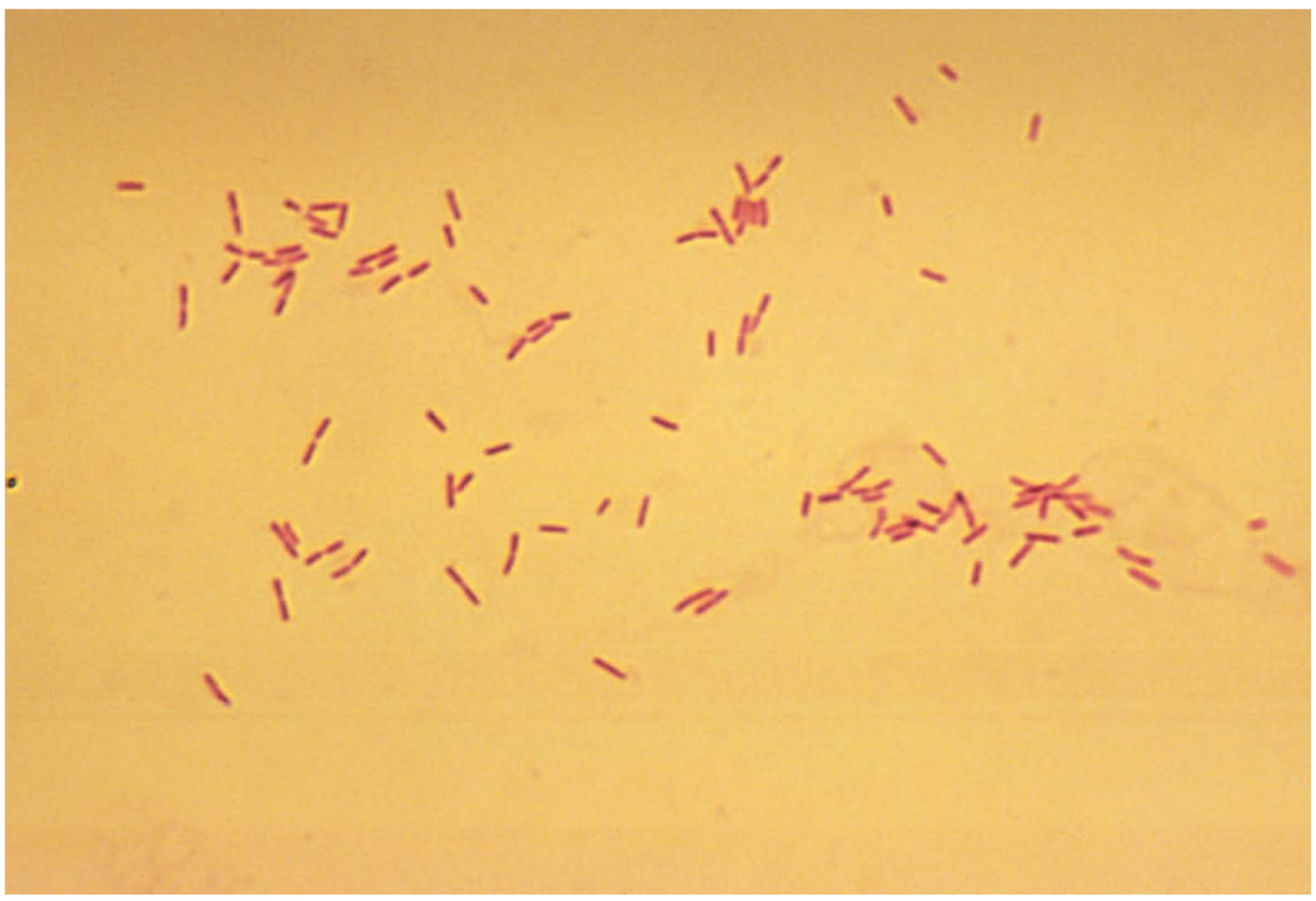


FIGURE 25.5 ■ **Bacteria.** Bacteria are often seen in urine specimens and either can be consistent with infection or may result from local contamination from surrounding skin during specimen collection. (Photo contributor: Roche Laboratories, Division of Hoffman-LaRoche Inc., Nutley, NJ.)

SYNOVIAL FLUID ANALYSIS FOR CRYSTALS

Uses

To determine the presence of uric acid crystals (in patients with gout) or calcium pyrophosphate crystals (in patients with pseudogout) in joint fluid.

Materials

Freshly collected joint fluid, glass microscope slide, coverslip, and polarizer.

Method

1. To prevent interference from polarizing artifacts, clean the slide and coverslip with alcohol prior to using them.
2. Using freshly collected unspun joint fluid, place a drop of joint fluid on the glass microscope slide.
3. Overlay coverslip.
4. View the slide using the polarizer.
5. Scan at ×10 power; ×100 power is needed to see intracellular crystals.



FIGURE 25.6B ■ **Compensated Polarized Uric Acid Crystals (×500).** Once crystals are found with a direct polarizing light, identification is made by using a compensated polarized light. The yellow crystal is aligned parallel to the slow vibration component of the compensator (negatively birefringent). The blue crystal is perpendicular (crossed urate blue). (Reproduced with permission from Strasinger SK, Di Lorenzo MS. Urinalysis and Body Fluids. 5th ed. Philadelphia: F.A. Davis Company; 2008, p. 217.)

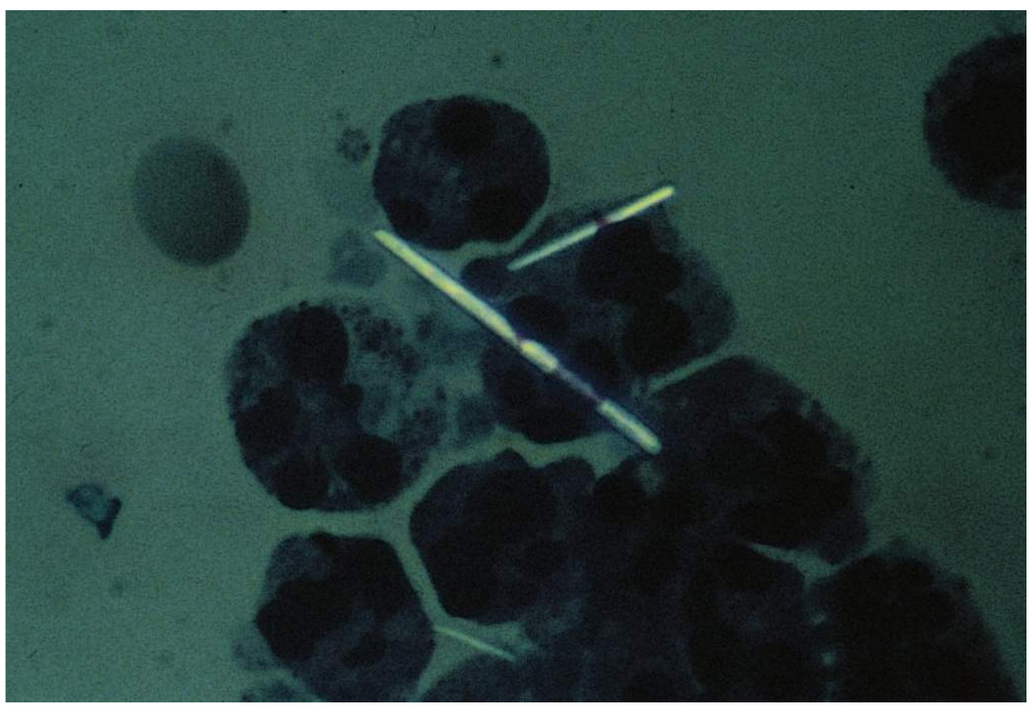


FIGURE 25.6A ■ **Polarized Uric Acid Crystals (×500).** Intracellular needle-like uric acid crystals are seen within the polymorphonuclear cells from the joint fluid in a patient with gout, using a direct polarizing light. (Reproduced with permission from Strasinger SK, Di Lorenzo MS. Urinalysis and Body Fluids. 5th ed. Philadelphia: F.A. Davis Company; 2008, p. 216.)

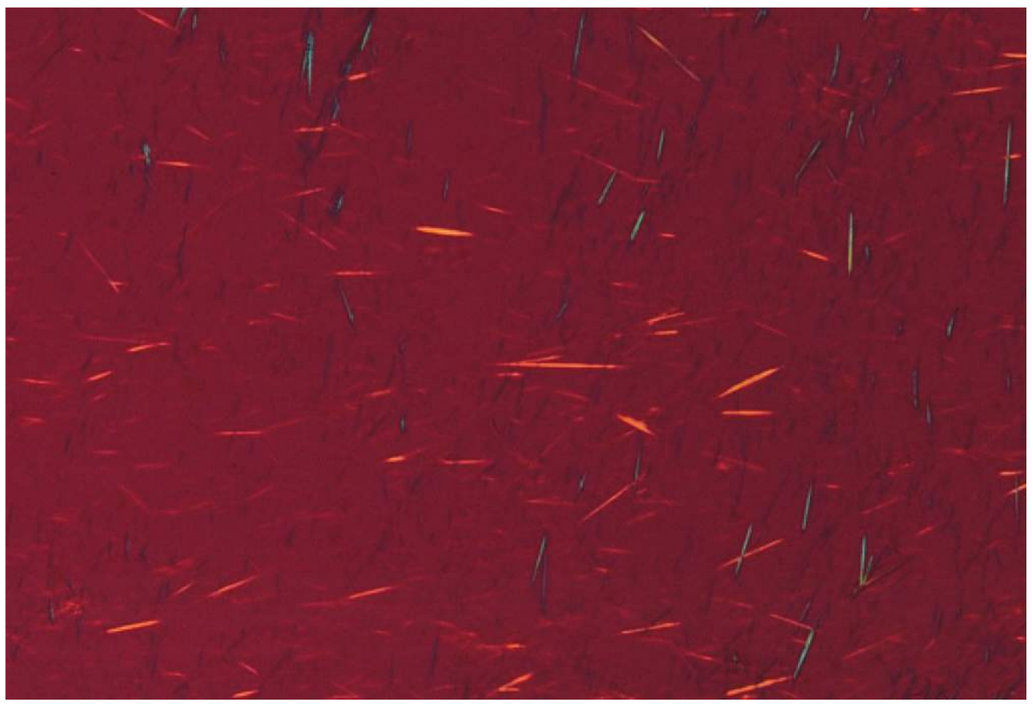


FIGURE 25.6C ■ **Extracellular Uric Acid Crystals (×100).** Extracellular uric acid crystals are seen under compensated polarized light. Notice the change of color with crystal alignment. (Reproduced with permission from Strasinger SK, Di Lorenzo MS. Urinalysis and Body Fluids. 5th ed. Philadelphia: F.A. Davis Company; 2008, p. 216.)

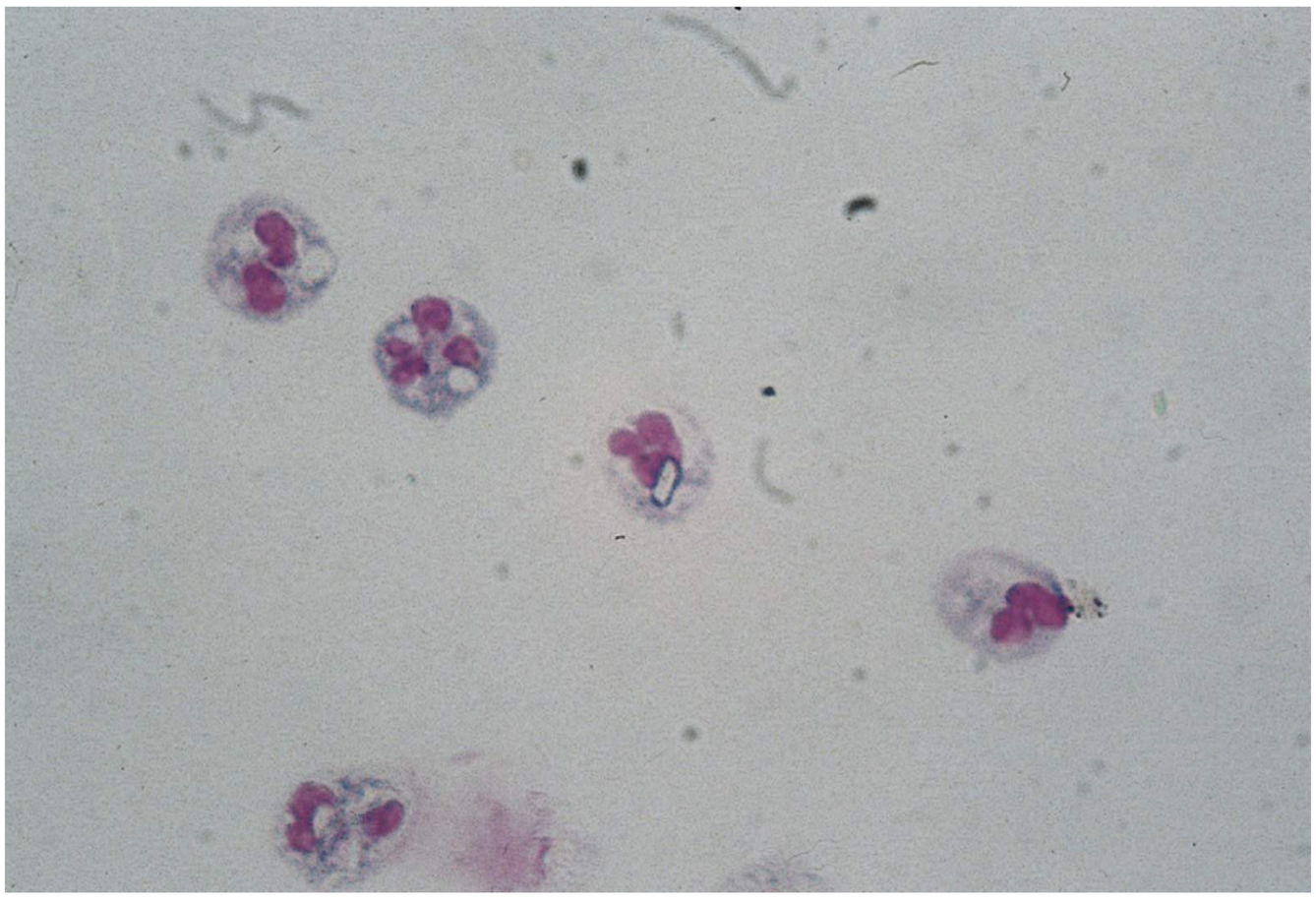


FIGURE 25.7A ■ **Polarized Calcium Pyrophosphate Crystals (×1000).** Intracellular rhomboid crystals in the joint of a patient with pseudogout. They may also appear as rods. (Reproduced with permission from Strasinger SK, Di Lorenzo MS. Urinalysis and Body Fluids. 5th ed. Philadelphia: F.A. Davis Company; 2008, p. 216.)

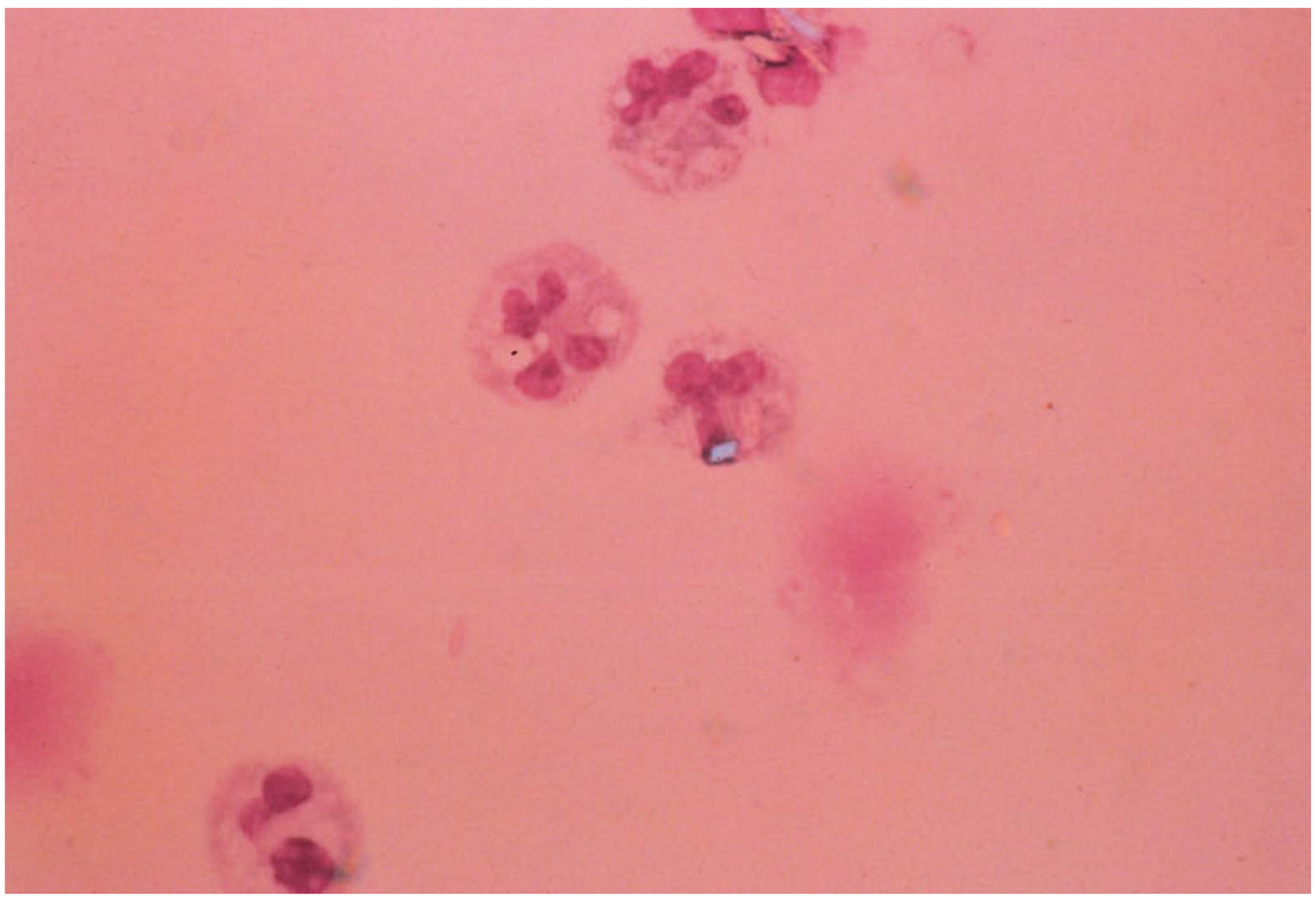


FIGURE 25.7B ■ **Compensated Polarized Calcium Pyrophosphate Crystals (×1000).** The blue calcium pyrophosphate crystal is aligned parallel to the slow vibration component of the compensator (positively birefringent). (Reproduced with permission from Strasinger SK, Di Lorenzo MS. Urinalysis and Body Fluids. 5th ed. Philadelphia: F.A. Davis Company; 2008, p. 217.)

GRAM STAIN

Uses

The first step in the identification of a predominant bacterial organism in a specimen. Classifies an organism by its cell wall's ability to retain crystal violet dye during solvent treatment. Morphology of the organism is also identified.

Materials

Freshly collected specimen to be examined, glass microscope slide, crystal violet, Gram iodine, acetone-alcohol (acetone, 30 mL, and 95% alcohol, 70 mL), safranin, and Bunsen burner.

Method

1. Put specimen on dry, clean glass microscope slide and allow to air dry.
2. Heat-fix specimen by gently passing over flame.
3. Cover specimen with crystal violet for 1 minute.
4. Rinse off completely with water; do not blot.
5. Cover specimen with Gram iodine for 1 minute.
6. Rinse off completely with water; do not blot.
7. Decolorize for 30 seconds with gentle agitation in acetone-alcohol.
8. Rinse off completely with water; do not blot.
9. Cover with safranin for 10 to 20 seconds.
10. Rinse off completely with water and let air dry.

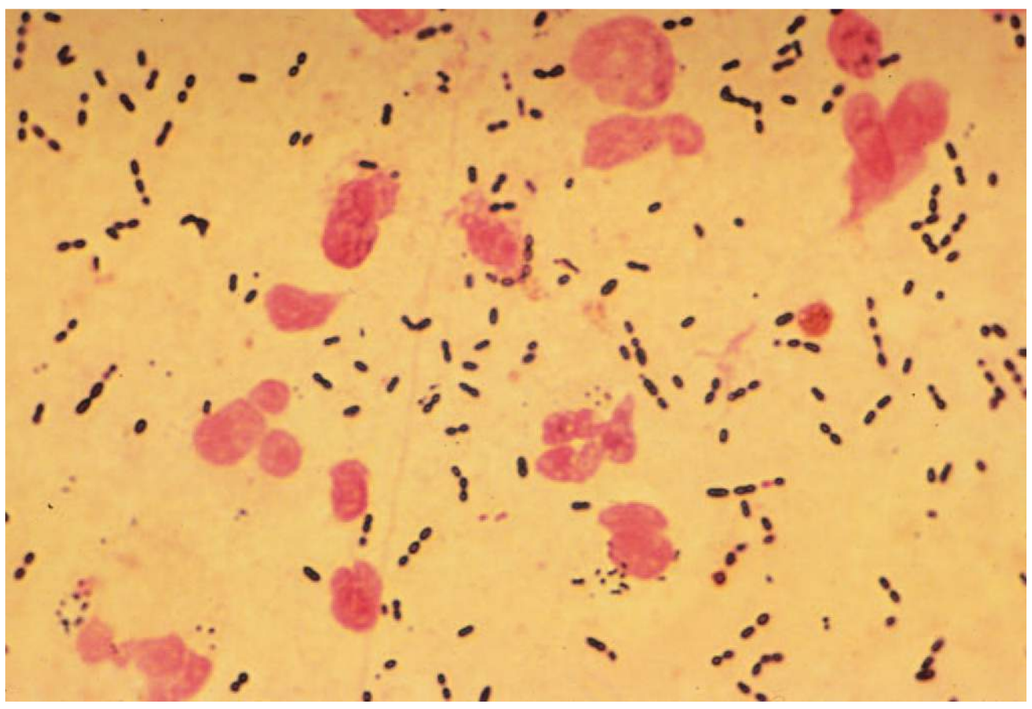


FIGURE 25.8 ■ **Gram Stain**—Streptococcus pneumoniae. Gram-positive, kidney-shaped diplococci of S pneumoniae. (Photo contributor: Roche Laboratories, Division of Hoffman-LaRoche Inc. Nutley, NJ.)

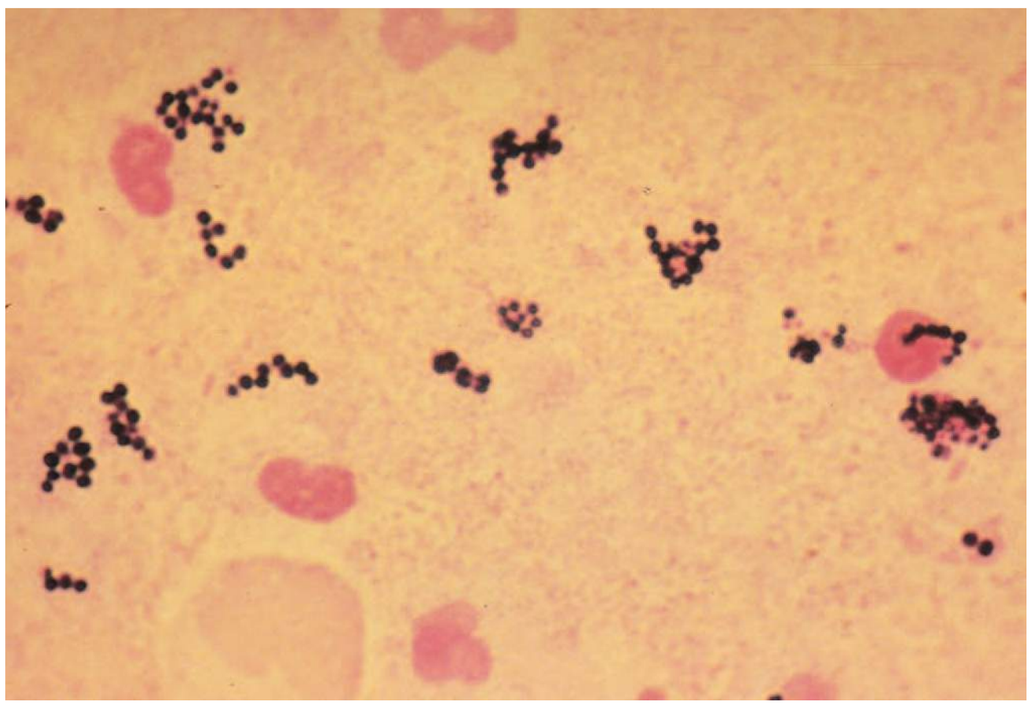


FIGURE 25.9 ■ **Gram Stain**—Staphylococcus aureus. Small clusters of gram-positive cocci seen in S aureus infection. (Photo contributor: Roche Laboratories, Division of Hoffman-LaRoche Inc. Nutley, NJ.)

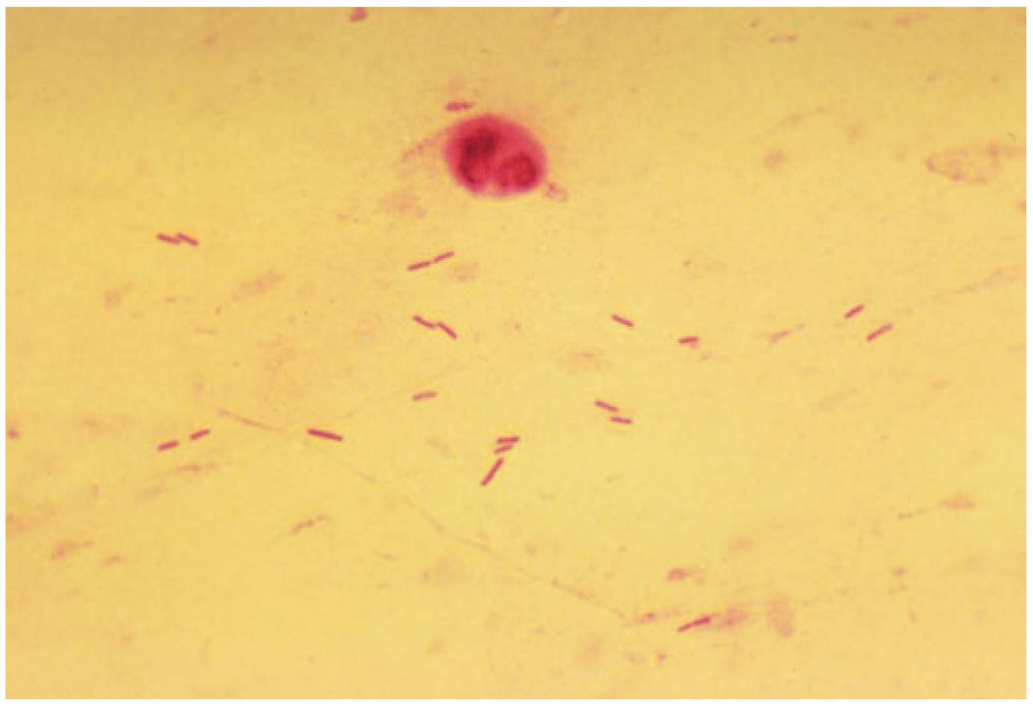


FIGURE 25.10 ■ **Gram Stain—Gram-Negative Rods.** Gram-negative rods of Pseudomonas aeruginosa. (Photo contributor: Roche Laboratories, Division of Hoffman-LaRoche Inc. Nutley, NJ.)

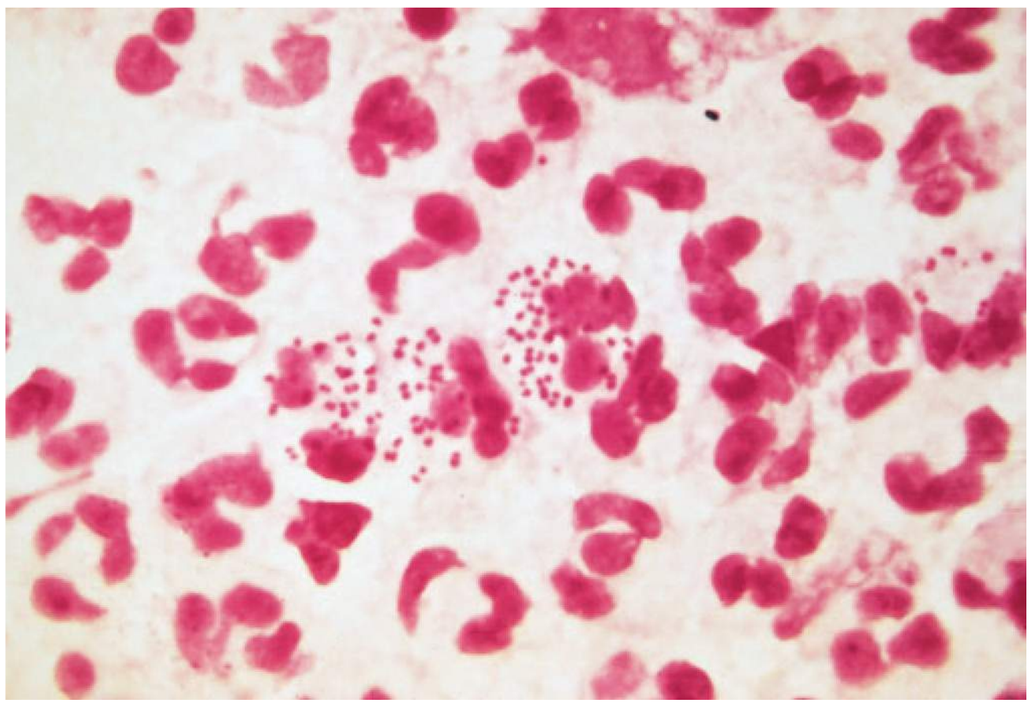


FIGURE 25.11 ■ **Gram Stain**—Neisseria gonorrhoeae. Multiple gram-negative, intracellular diplococci from a patient with N gonorrhoeae. (Reproduced with permission from Morse SA, Holmes KK, Ballard RC, Moreland AA (eds). Atlas of Sexually Transmitted Diseases and AIDS. 4th ed. Saunders Elsevier; 2010. Copyright © 2010 Elsevier, Ltd.)

Uses

To evaluate a lesion (chancre, mucous patche, condyloma lata, skin rash) for the presence of Treponema pallidum.

Materials

Compound microscope with dark-field condenser (dark-field microscope), glass microscope slide, coverslip, and physiologic saline.

Method for Obtaining and Viewing the Specimen

1. From chancre or condyloma lata:
   a. Gently abrade the lesion with a dry gauze.
   b. Dab away any bleeding.
   c. Touch slide to exudative fluid in base of lesion.
   d. Overlay coverslip and view immediately under dark-field microscope using ×40 and ×100 objectives.
2. From mucous patch:
   a. Touch slide to mucous patch.
   b. Overlay coverslip and view immediately under dark-field microscope using ×40 and ×100 objectives.
3. From skin lesion:
   a. Gently scrape surface of skin lesion with edge of a number 15 scalpel blade.
   b. Dab away any bleeding.
   c. Touch slide to exudative fluid rising from skin lesion.
   d. Overlay coverslip and view immediately under dark-field microscope using ×40 and ×100 objectives.

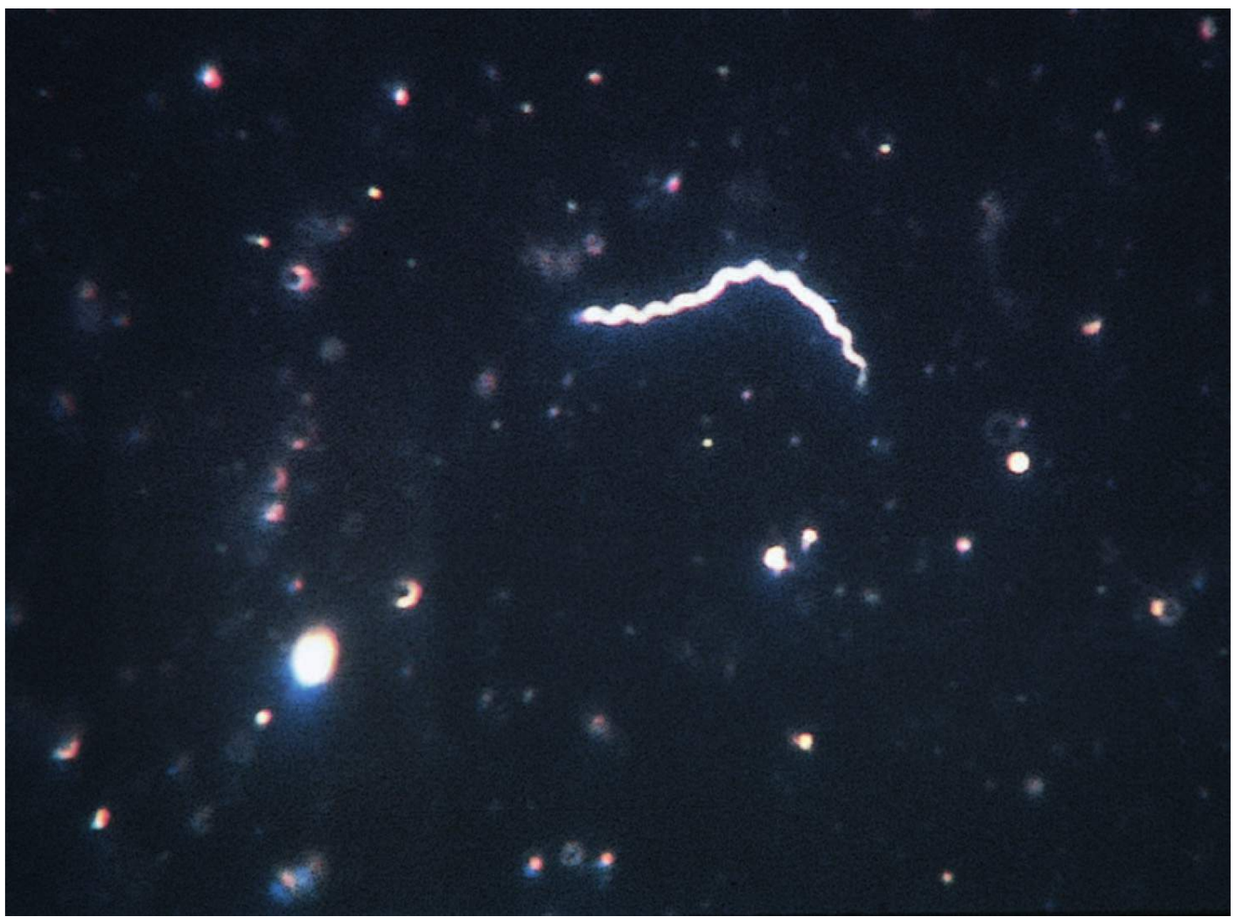


FIGURE 25.12 ■ **Dark-Field Microscopy.** Examined under a dark-field microscope at ×40 or ×100 power, spirochetes appear as motile, bright corkscrews against a black background. (Reproduced with permission from Morse SA, Holmes KK, Ballard RC, Moreland AA (eds). Atlas of Sexually Transmitted Diseases and AIDS. 4th ed. Saunders Elsevier; 2010. Copyright © 2010 Elsevier, Ltd.)

VAGINAL FLUID WET MOUNT

Uses

To examine for clue cells, Trichomonas, and sperm.

Materials

Aqueous sodium chloride, glass microscope slide, and coverslip.

Method

1. Place a drop of saline onto the middle of the glass slide. (Alternative method: Place several drops of saline in a small glass test tube and place the swab in the tube. The swab can then be wiped onto a slide at a later time.)
2. Mix a small amount of vaginal fluid to be examined into the saline drop.
3. Overlay a coverslip.
4. Examine directly through microscope at ×40 and ×100 (oil immersion).

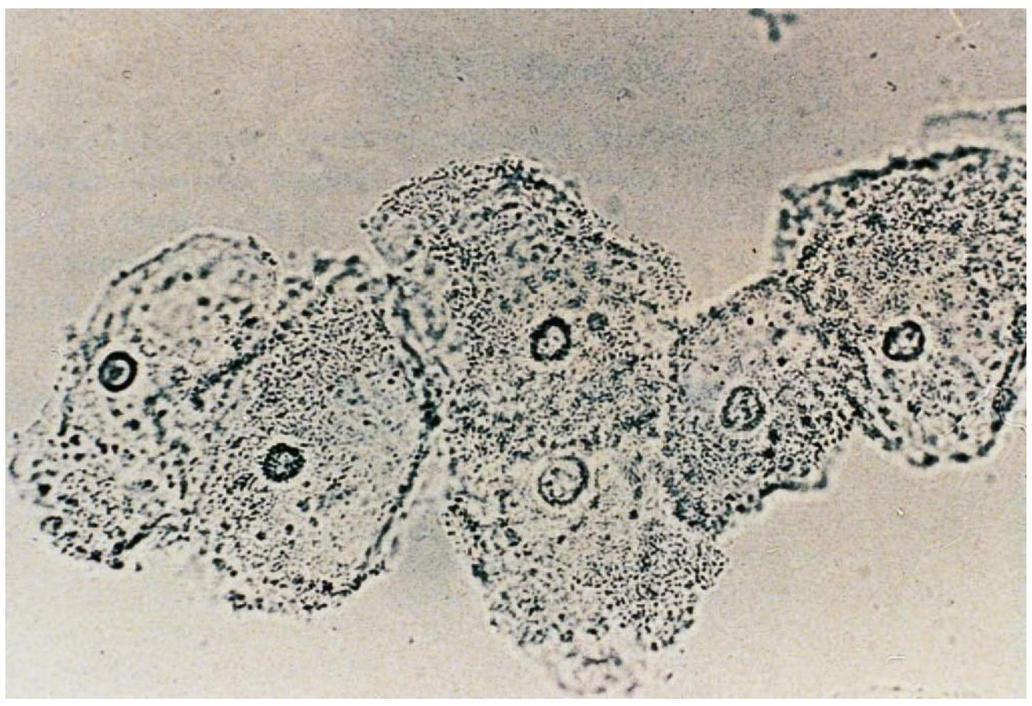


FIGURE 25.13 ■ **Clue Cells.** "Glitter cell" or "clue cell": Epithelial cell covered with adherent bacteria in a wet mount of a vaginal specimen from a patient with Gardnerella vaginalis (also known as nonspecific vaginitis or bacterial vaginosis). Note the refractile appearance, indistinct borders, and ragged edges of the epithelial clue cell. (Photo contributor: Curatek Pharmaceuticals.)

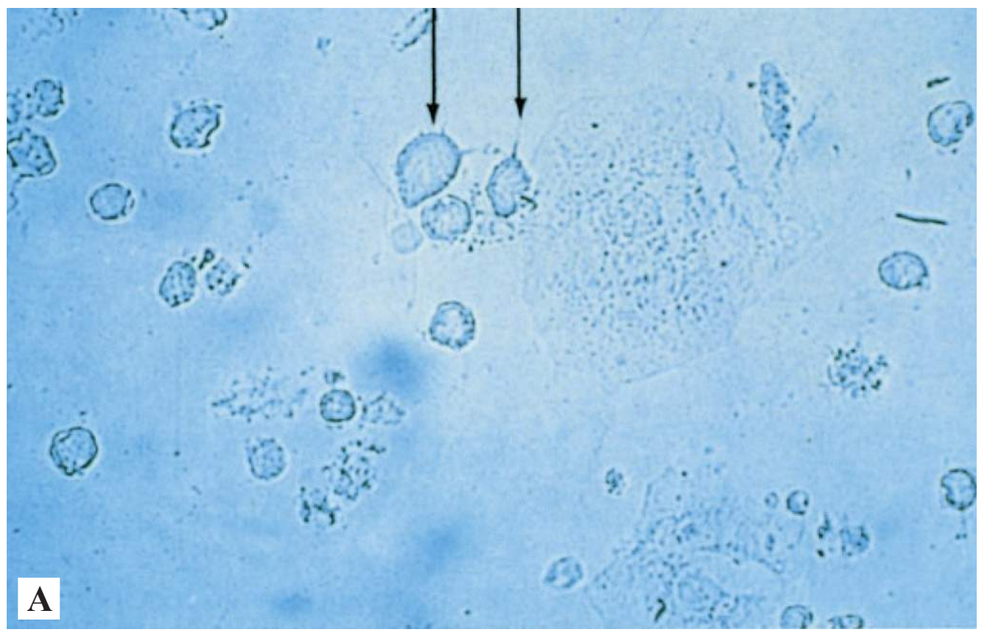


FIGURE 25.14 ■ Trichomonas. Saline wet mount demonstrating oval-bodied, flagellated trichomonads. They are similar in size to leukocytes and can be distinguished from them by their motility and presence of flagella. (Photo contributor: H. Hunter Handsfield, MD. From Handsfield HH (ed): Atlas of Sexually Transmitted Diseases, 3rd ed. New York: McGraw-Hill; 2011.)

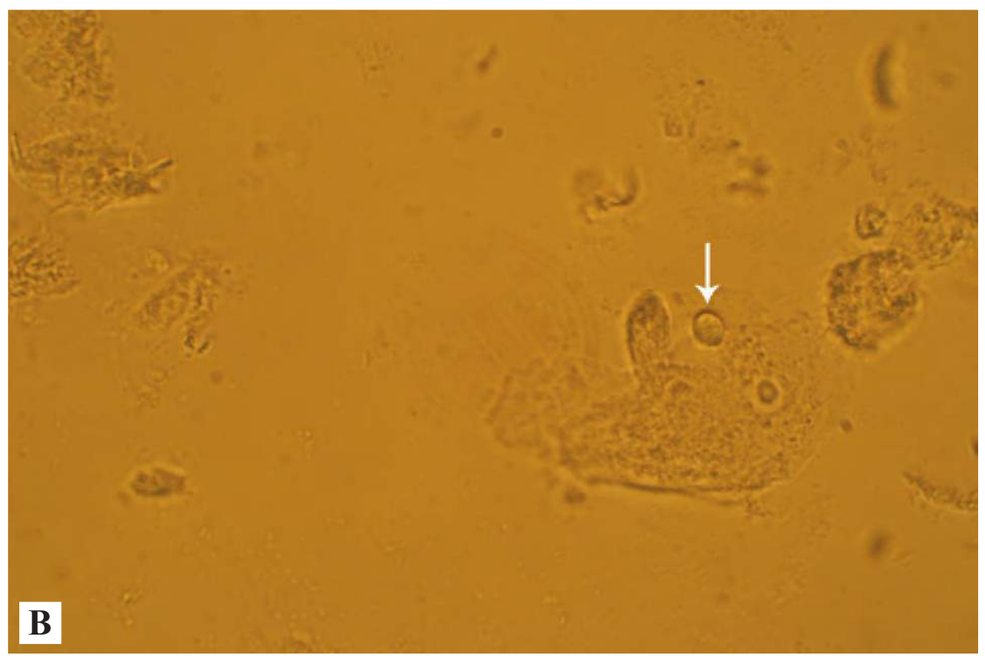


FIGURE 25.14 ■ Trichomonas. Saline wet mount demonstrating a single trichomonad. Its flagella are not easily seen in this photograph. (Photo contributor: Lawrence B. Stack, MD.)



FIGURE 25.15 ■ **Spermatozoa.** Spermatozoa may be motile or immotile. (Reproduced with permission from Strasinger SK, Di Lorenzo MS. Urinalysis and Body Fluids. 5th ed. Philadelphia: F.A. Davis Company; 2008, p. 102.)

POTASSIUM HYDROXIDE MOUNT

Uses

To examine for yeast and fungus.

Materials

Aqueous potassium hydroxide (KOH) 10%, glass microscope slide, and coverslip.

Method

1. Place a drop of KOH onto the middle of the glass slide.
2. Suspend a small amount of vaginal fluid into the drop of KOH.
3. Overlay a coverslip.
4. Let sit at room temperature for 30 minutes; as an alternative, gently heat the slide over a Bunsen burner but do not boil.
5. Examine under microscope for hyphae and spores.

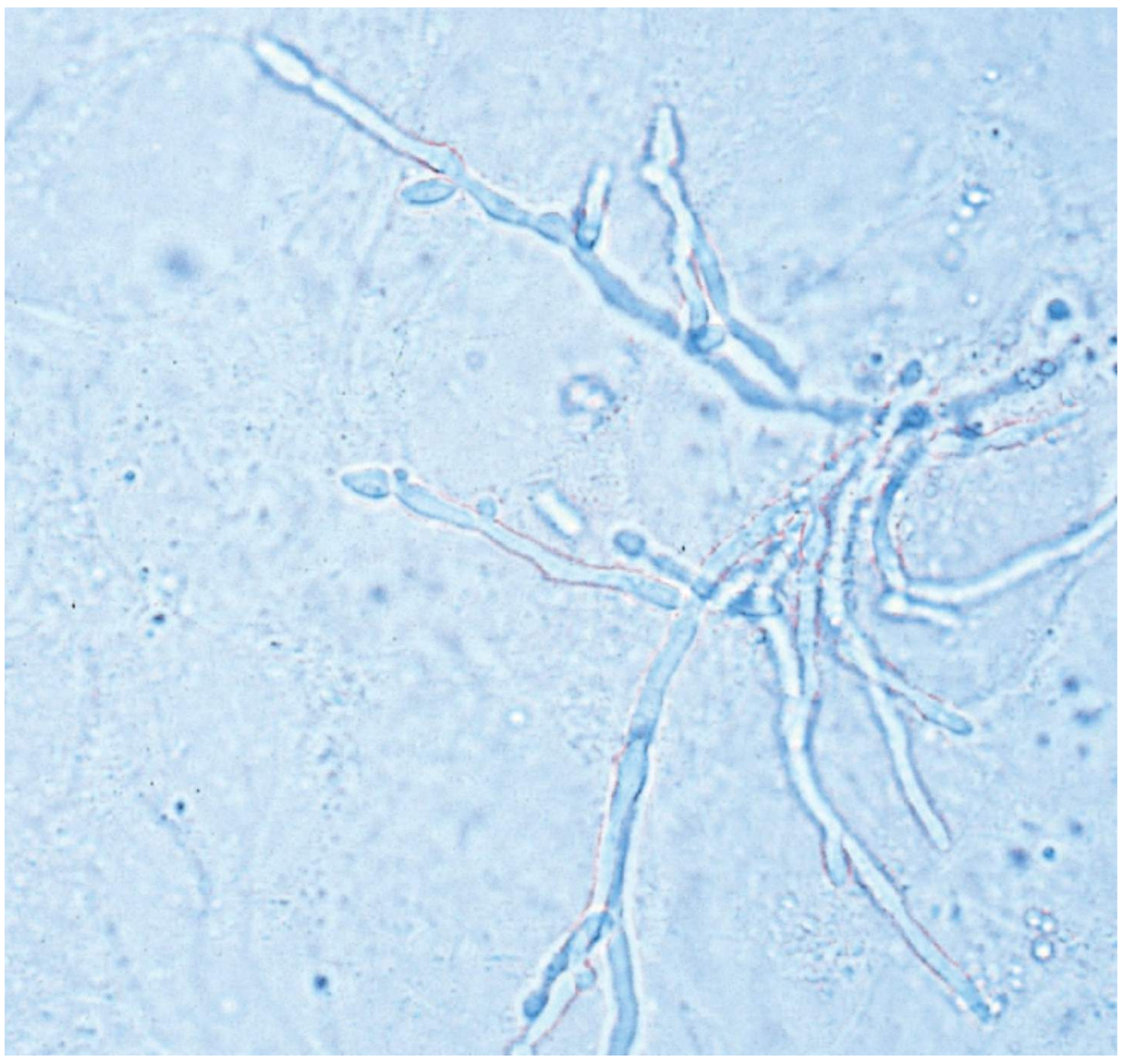


FIGURE 25.16 ■ Candida albicans. Potassium hydroxide preparation of vaginal secretions from a patient with vaginal candidiasis due to C albicans. Note the pseudohyphae characteristic of this organism. (Photo contributor: David A. Eschenbach, MD. From Handsfield HH (ed): Atlas of Sexually Transmitted Diseases, 3rd ed. New York: McGraw-Hill; 2011.)

STOOL EXAMINATION FOR FECAL LEUKOCYTES

Uses

To evaluate a patient's stool sample for the presence of fecal leukocytes.

Materials

Freshly collected liquid stool specimen, glass microscope slide, coverslip, and methylene blue.

Method

1. Place a drop of liquid stool onto the glass slide.
2. Add two drops of methylene blue to the stool specimen.
3. Mix thoroughly.
4. Overlay with a coverslip.
5. Place the edge of a piece of filter paper adjacent to the coverslip to absorb any excess methylene blue.
6. Examine using ×10 objective to scan specimen and ×40 and ×100 to identify specific leukocytes.

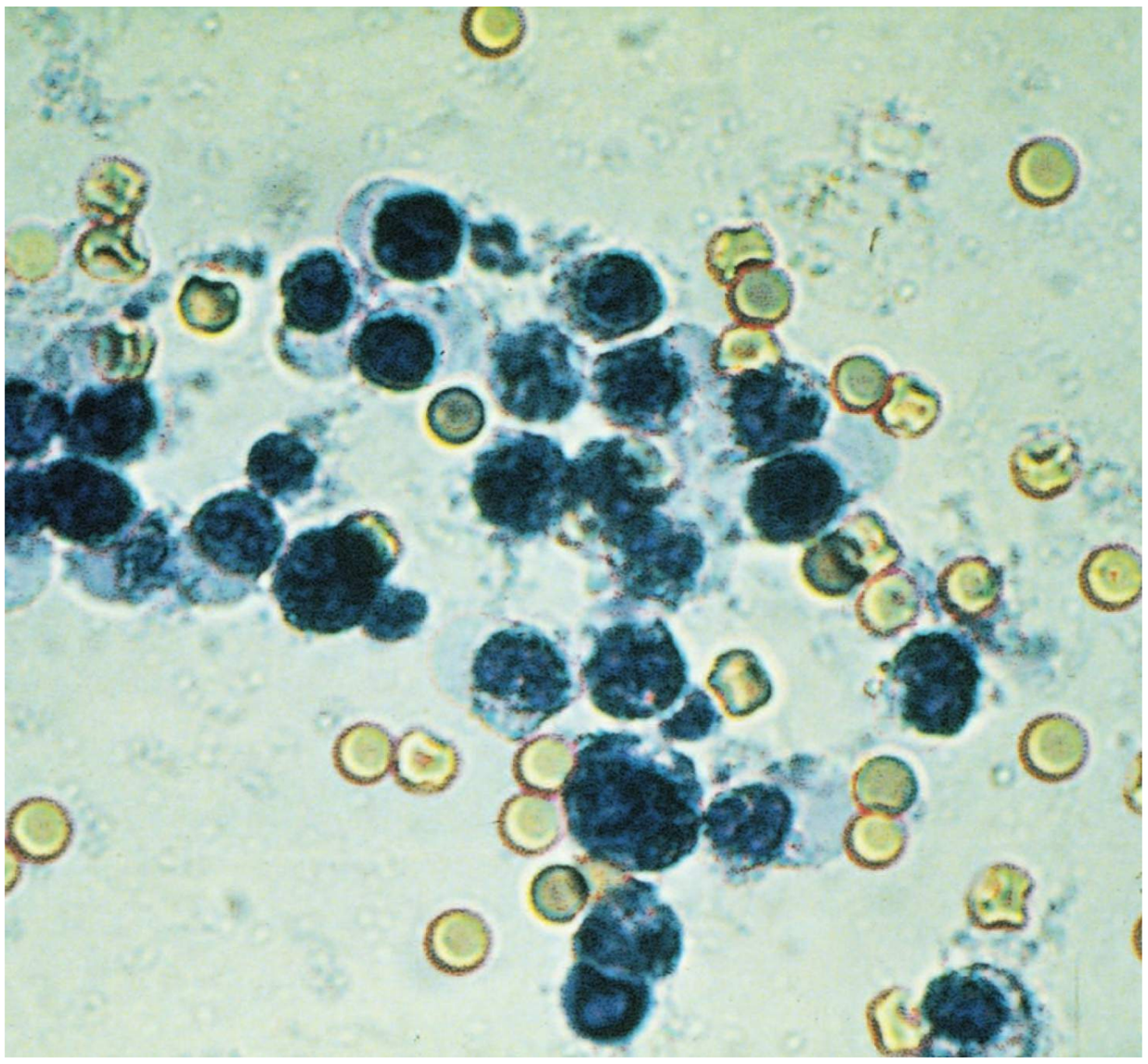


FIGURE 25.17 ■ **Fecal Leukocytes.** Multiple white cells in the stool specimen from a patient with bacterial diarrhea. (Photo contributor: Herbert L. DuPont, MD.)

SKIN SCRAPING FOR DERMATOSES AND INFESTATIONS

Uses

To determine fungal dermatoses or skin infestations.

Materials

Fresh skin scraping, glass microscope slide, coverslip, and 10% potassium hydroxide or mineral oil.

Method

1. Specimen collection:
   a. Gently scrape skin lesion with edge of a number 15 scalpel.
2. Slide preparation:
   a. Pediculosis may be seen grossly clinging to individual hair or under low power. Live nits may fluoresce with a Wood lamp.
   b. For scabies, place a drop of KOH or mineral oil onto the slide.
   c. Suspend a small amount of the scraping onto the drop.
   d. Overlay a coverslip.
   e. Let sit at room temperature for 30 minutes; as an alternative, gently heat the slide over a Bunsen burner but do not boil.
   f. Examine under microscope for hyphae, spores, or infestations.

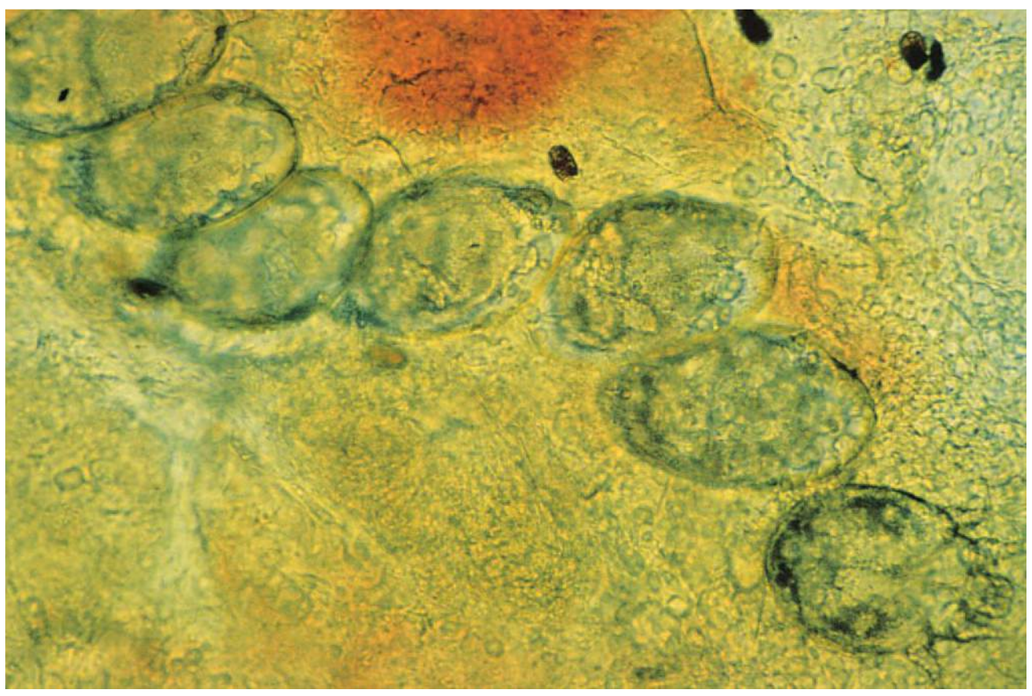


FIGURE 25.18 ■ **Scabies.** Skin scraping from a patient with scabies. Note the intact mite at the lower right of the photograph, and the ova and fecal pellets. (Photo contributor: Department of Dermatology, Naval Medical Center, Portsmouth, VA.)

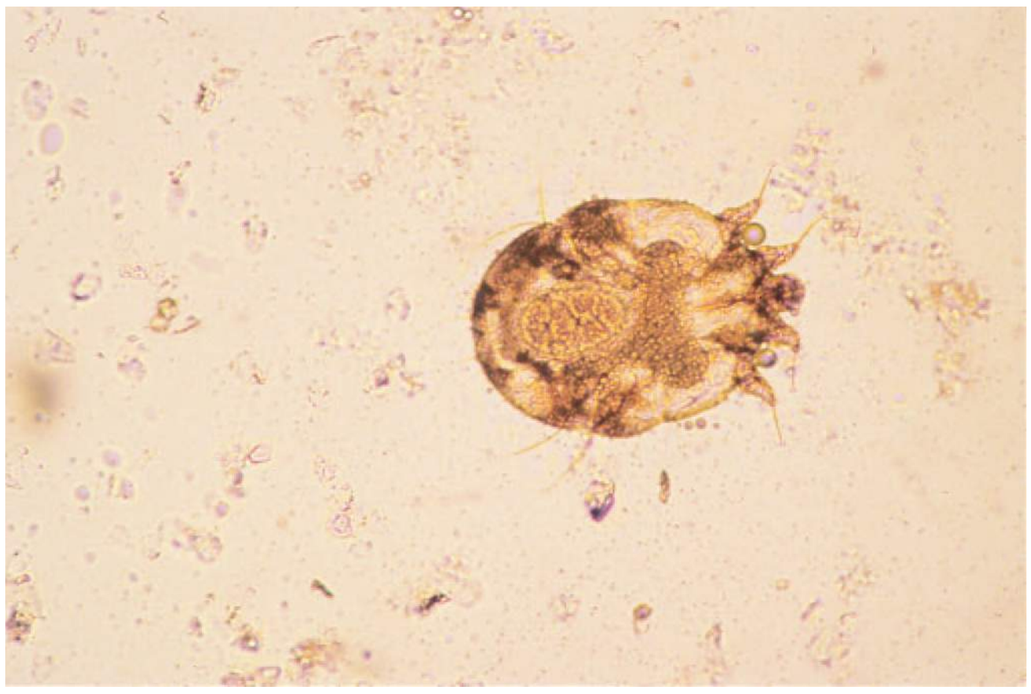


FIGURE 25.19 ■ **Scabies.** Adult female scabies mite. (Reproduced, with permission, from Morse SA, Ballard RC, Holmes KK, Moreland AA (eds). Atlas of Sexually Transmitted Diseases and AIDS. 3rd ed. Philadelphia: Mosby; 2003. Copyright Elsevier.)

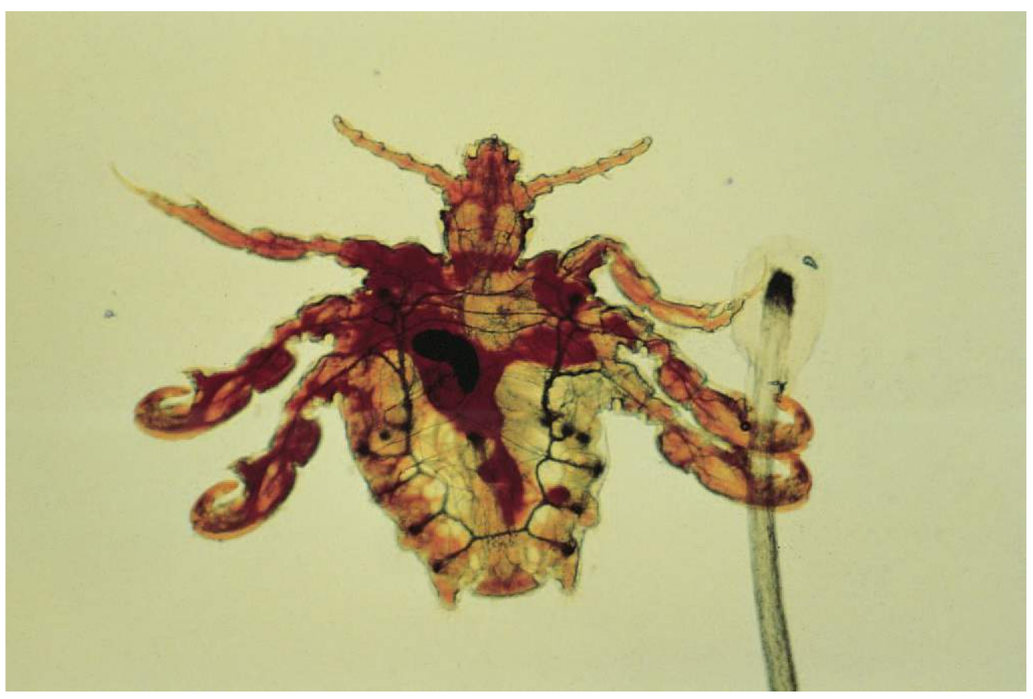


FIGURE 25.20 ■ **Pediculosis.** Phthirus pubis, the crab louse. Note the short body and claw-like legs, which are ideally suited for clinging to the hair shaft. (Photo contributor: Department of Dermatology, Naval Medical Center, Portsmouth, VA.)



FIGURE 25.21 ■ **Pediculosis.** Phthirus corporis, the body louse. Note the elongated body. (Photo contributor: Department of Dermatology, Naval Medical Center, Portsmouth, VA.)

CEREBROSPINAL FLUID EXAMINATION: INDIA INK PREPARATION

Uses

To examine cerebrospinal fluid for organisms with capsules, particularly Cryptococcus neoformans.

Materials

India Ink, glass microscope slide, and coverslip.

Method

1. Lightly centrifuge cerebrospinal fluid to concentrate cells at bottom of tube (1-2 minutes).
2. Pour off excess fluid (retain if further testing may be necessary).
3. Take a drop from the bottom of the centrifuge tube and place it in the middle of a glass microscope slide.
4. Place a drop of India Ink into the specimen drop; gently mix.
5. Overlay a coverslip.
6. Examine at ×10 to screen specimen, use ×40 objective to confirm findings.

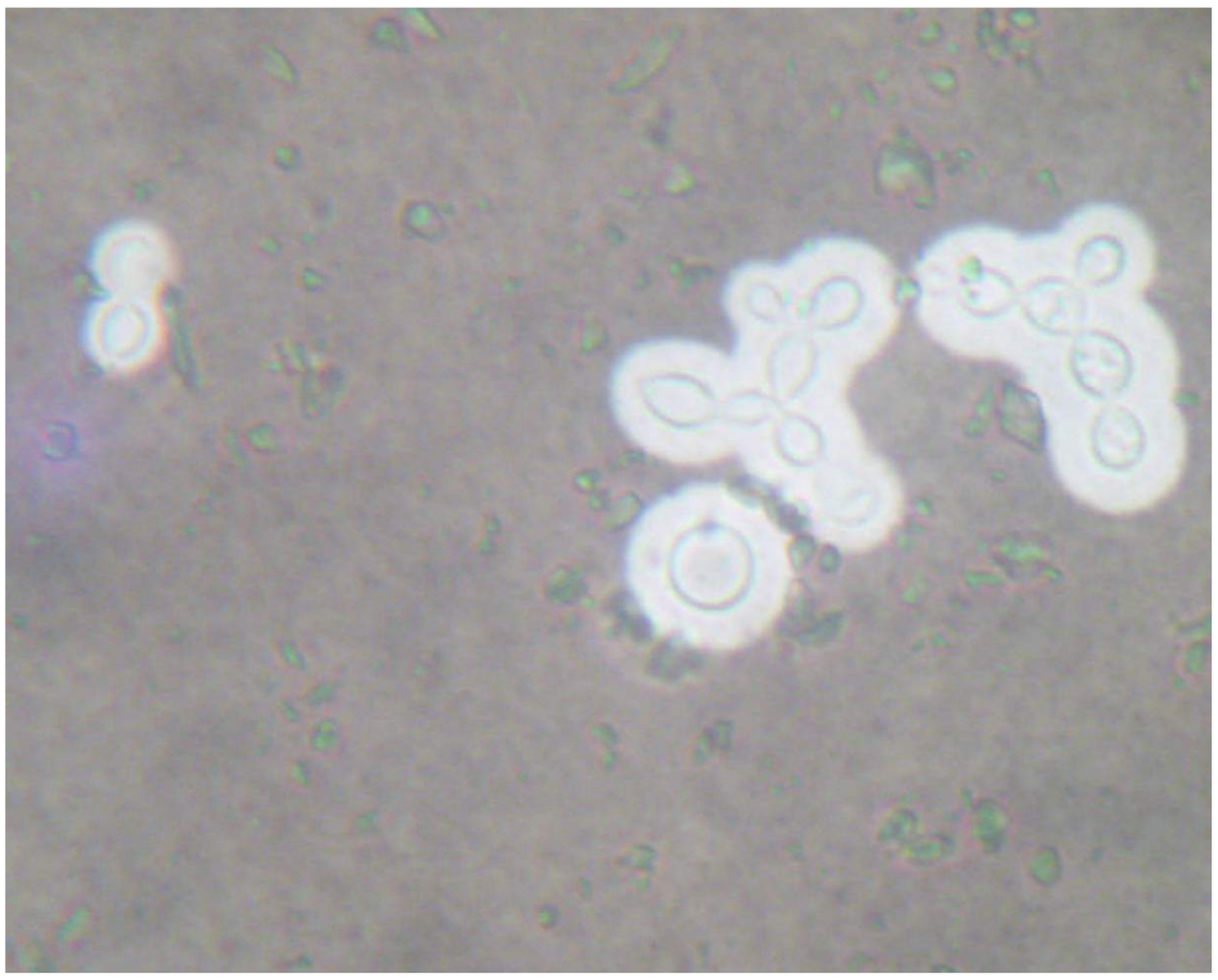


FIGURE 25.22 ■ **India Ink Preparation.** Budding yeast with prominent capsule on India Ink preparation from a patient with C neoformans meningitis. (Photo contributor: Seth Wright, MD.)

WRIGHT STAIN—THIN SMEAR FOR MALARIA

Uses

To evaluate for the presence of ring trophozoites.

Materials

Air-dried blood smear, Coplin jar of Wright stain, slide rack, pH 7.2 buffer, and blotting paper.

Method

1. Place a drop of blood on the middle of a slide.
2. Hold another slide evenly on top of the slide at a 45-degree angle and drag the slide over the drop of blood to the opposite edge to spread the blood evenly.
3. Allow the blood to dry for 5 to 10 minutes.
4. Stain air-dried smears in a closed Coplin jar of Wright stain for 5 minutes.
5. Place the slide on a rack.
6. Rinse and treat with pH 7.2 buffer primed with 1 mL Wright stain per 400 mL for 3 minutes.
7. Rinse in pH 7.2 buffer for 20 seconds.
8. Blot dry and mount on microscope at ×100 (oil immersion).

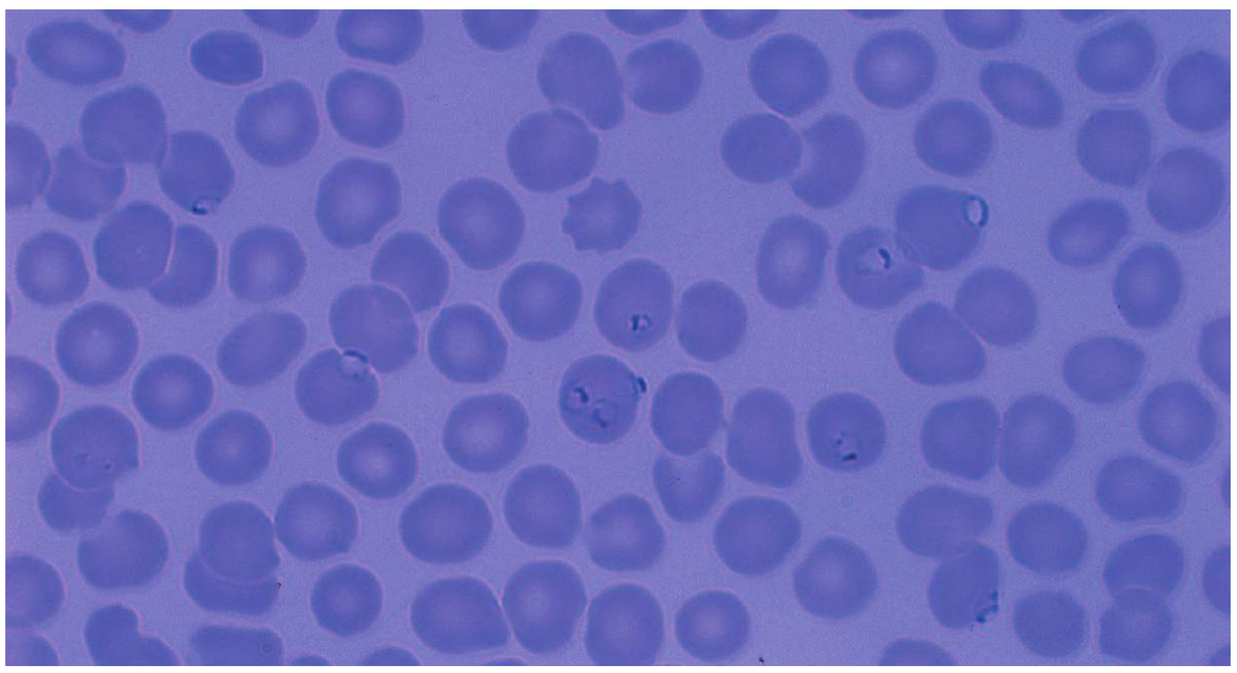


FIGURE 25.23 ■ Plasmodium falciparum **Thin Film.** Ring forms (trophozoites) of P falciparum are seen on the Wright stain thin film in a patient with intermittent fever who had recently traveled to Africa. (Photo contributor: James P. Elrod, MD, PhD.)

FERNING PATTERN FOR AMNIOTIC FLUID

Uses

To distinguish between amniotic fluid due to membrane rupture and normal vaginal secretions in patients beyond the 20th week in preganacy who present with spontaneous vaginal fluid passage. Characteristic arborization, or typical ferning pattern, confirms amniotic fluid and spontaneous membrane rupture.

Materials

Sterile speculum, vaginal fluid, microscope slide, microscope, and serile swab or pipette.

Method

1. Place patient in dorsal lithotomy position.
2. Do not use lubricants or cleaning agents.
3. Place sterile speculum into vaginal vault.
4. Obtain sample of vaginal secretions from posterior vaginal pool using a pipette or steril swab.
5. Place a drop of vaginal fluid on a microscope slide.
6. Spread the specimen evenly so that a thin smear is formed.
7. Allow the fluid to air dry for 5 to 10 minutes. Do not apply heat.
8. Examine slide under low power (10×) for ferning pattern.

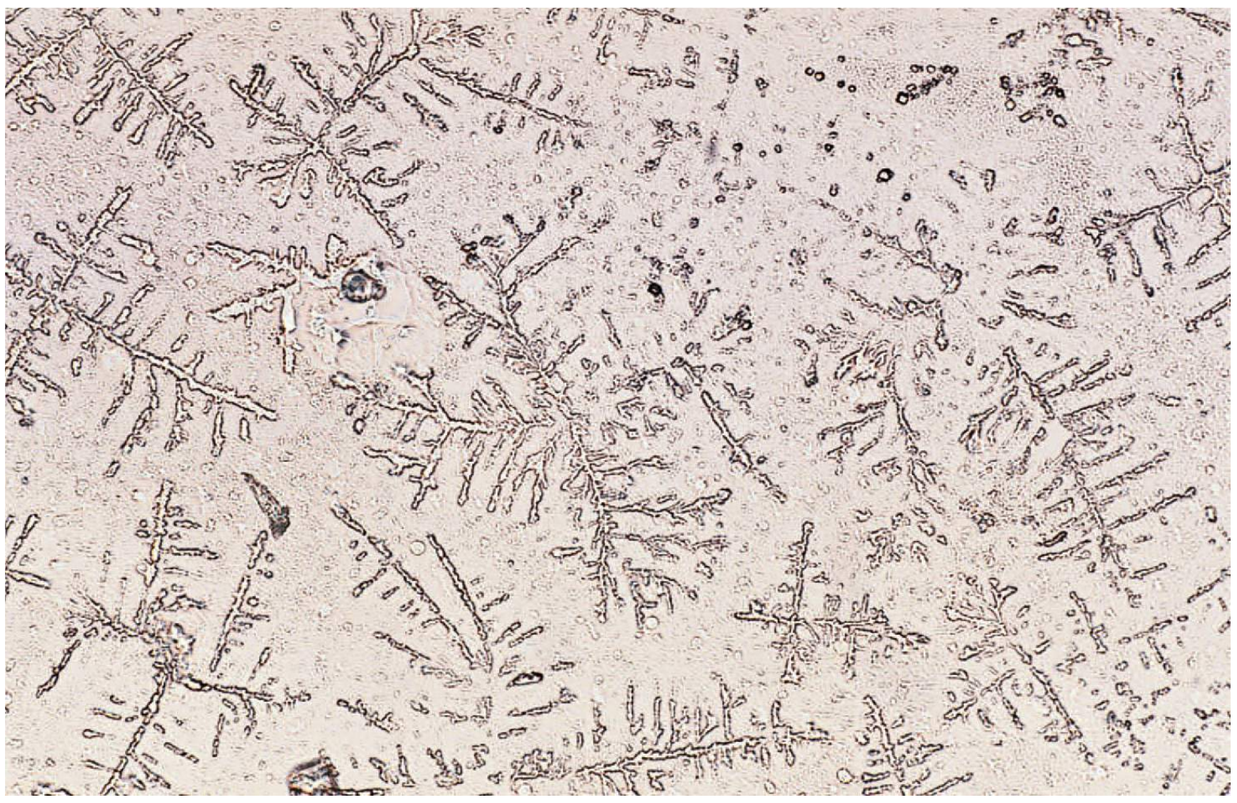


FIGURE 25.24 ■ **Ferning Pattern.** The arborization pattern found when a drop of amniotic fluid is allowed to air dry on a microscope slide, known as ferning. (Photo contributor: Robert Buckley, MD.)

PERIPHERAL BLOOD SMEAR FOR SCHISTOCYTES AND BLAST CELLS

Uses

To detect the presence of schistocytes or blast cells in a peripheral blood smear. Schistocytes are fragmented red blood cells due to shearing forces in microarterioles lined or meshed with fibrin strands. They are found in patients with disseminated intravascular coagulation, throbotic thrombocytopenic purpura/haemolytic uremic syndrome, microangiopathic haemolytic anemia, uremia, and carcinoma. Turbulent blood flow due to congestive heart failure, artificial heart valves, or vavlular stenosis may cause schistocyte formation. Greater than 1% of forms or greater than two schistocytes per high-powered field suggest schistocytosis.

Peripheral blood smear blasts are circulating immature white blood cells. When elevated white blood cell count and circulating blasts are seen in addition to anemia and thrombocytopenia, acute leukemia is suspected. As leukemia blast cells build up in the bone marrow, they allow less room for production of healthy white blood cells, red blood cells, and platelets. Although the diagnosis of leukemia can usually be made from the peripheral smear, bone marrow examination (aspiration or needle biopsy) is routinely done for definitive diagnosis.

Materials

Two glass microscope slides, drop of blood, pipette, Wright stain, Giemsa stain, immersion oil, and microscope.

Method

Smear Preparation

1. Agitate sample well, by inversion of tube or mechanical rocker.
2. Place a 2- to 3-mm drop of whole blood ¼ in from the right edge of a 1 × 3 in slide using a wooden applicator stick.
3. Place the slide on a flat surface and hold securely.
4. Grasp a second slide (spreader slide) in the right hand between thumb and forefinger.
5. Place the spreader slide onto the lower slide in front of the blood drop, and pull the slide back until it touches the drop.
6. Allow the blood to spread by capillary action almost to the edges to fht lower slide.
7. Push the spreader slide forward at a 30-degree angle, using a rapid even motion. The weight of the spreader slide should be the only weight applied. The drop of blood must be spread within seconds or the cell distribution will be uneven.
8. Allow to air dry.

Wright Stain of Peripheral Smear

1. Completely cover peripheral smear with Wright stain using a pipette. The stain layer should be ⅛ in thick.
2. Wait for 2 minutes.
3. Cover with equal amount of Giemsa solution.
4. Blow gently on the slide to mix solutions.
5. Allow to stand for 4 minutes.
6. Wash slide for 30 seconds with distilled water.
7. Allow to air dry
8. View smear with oil emersion at high (100×) power.

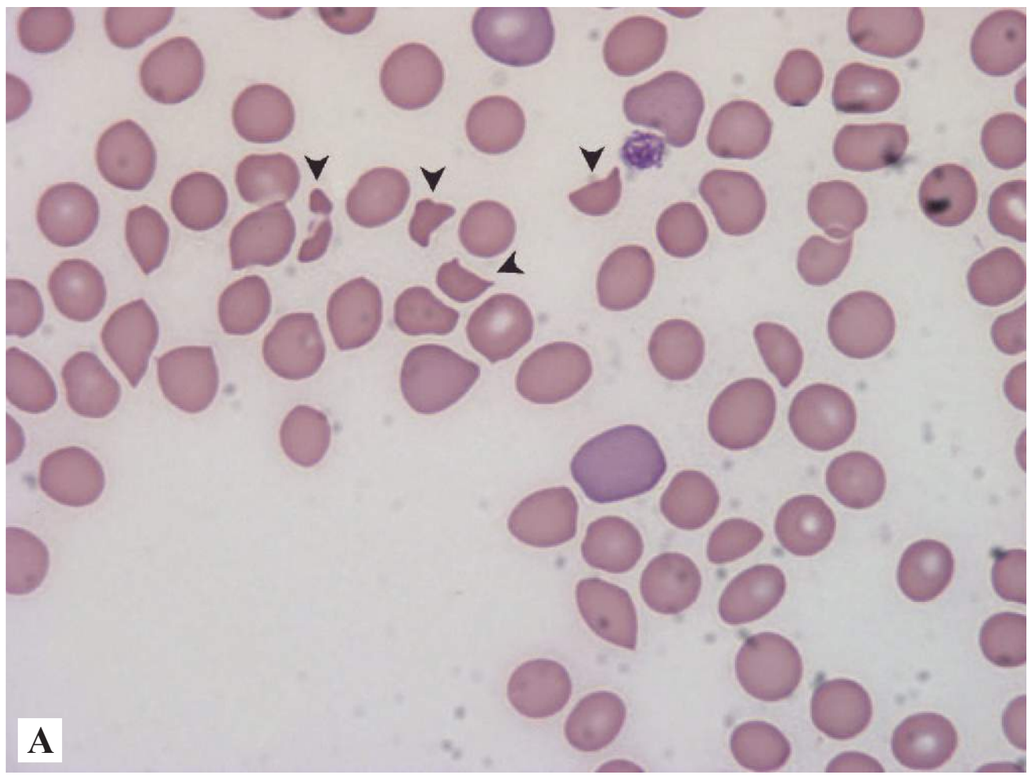


FIGURE 25.25A ■ **Schistocytes.** Multiple fragmented red blood cells (black arrowheads) seen on the peripheral smear at ×100 in a patient with microangiopathic hemolytic anemia. (Photo contributor: James P. Elrod, MD, PhD.)

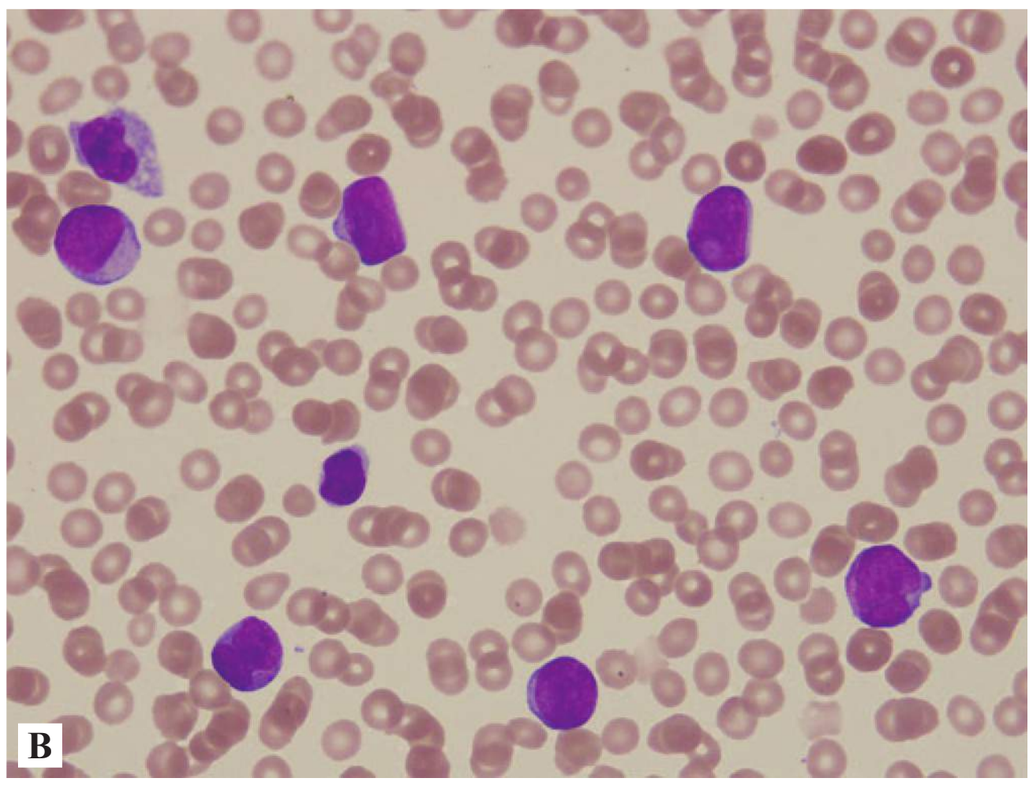


FIGURE 25.25B ■ **AML Blast Cells.** Wright stained peripheral blood smear, ×1000. Multiple blast cells seen in this smear from a patient with shortness of breath and a WBC of 105 K/uL, 80% blasts, HCT 20%, and PLT 75 K/uL. Flow cytometry confirms these are myeloid blasts conforming AML. (Photo contributor: Mary Ann Thompson Arildsen, MD, PhD.)

TAPE TEST FOR ENTEROBIAS VERMICULARIS EGGS

Uses

To detect the presence of eggs of Enterobias vermicularis in patients who present with nocturnal perianal pruritis or concern for pinworm infestation.

Materials

Microscope slide, clear transparent tape, tongue blade, microscope, or a cellulose-tape slide preparation.

Method

1. Affix ½ in of a 4-in piece of tape to the underside of a microscope slide.
2. Hold the slide against the tongue depressor 1 in from the end and lift the long end of the tape from the slide.
3. Loop tape over end of the depressor to expose gummed surface.
4. Hold tape and slide firmly against the tongue depressor.
5. Press gummed surfaces of the tape against several areas of the perianal area.
6. Affix long portion of the tape to the slide.
7. Smooth tape with cotton gauze.
8. View specimen under low (10×) power.

Note: Specimens are best obtained several hours after going to sleep or upon waking before a bowel movement or bath.

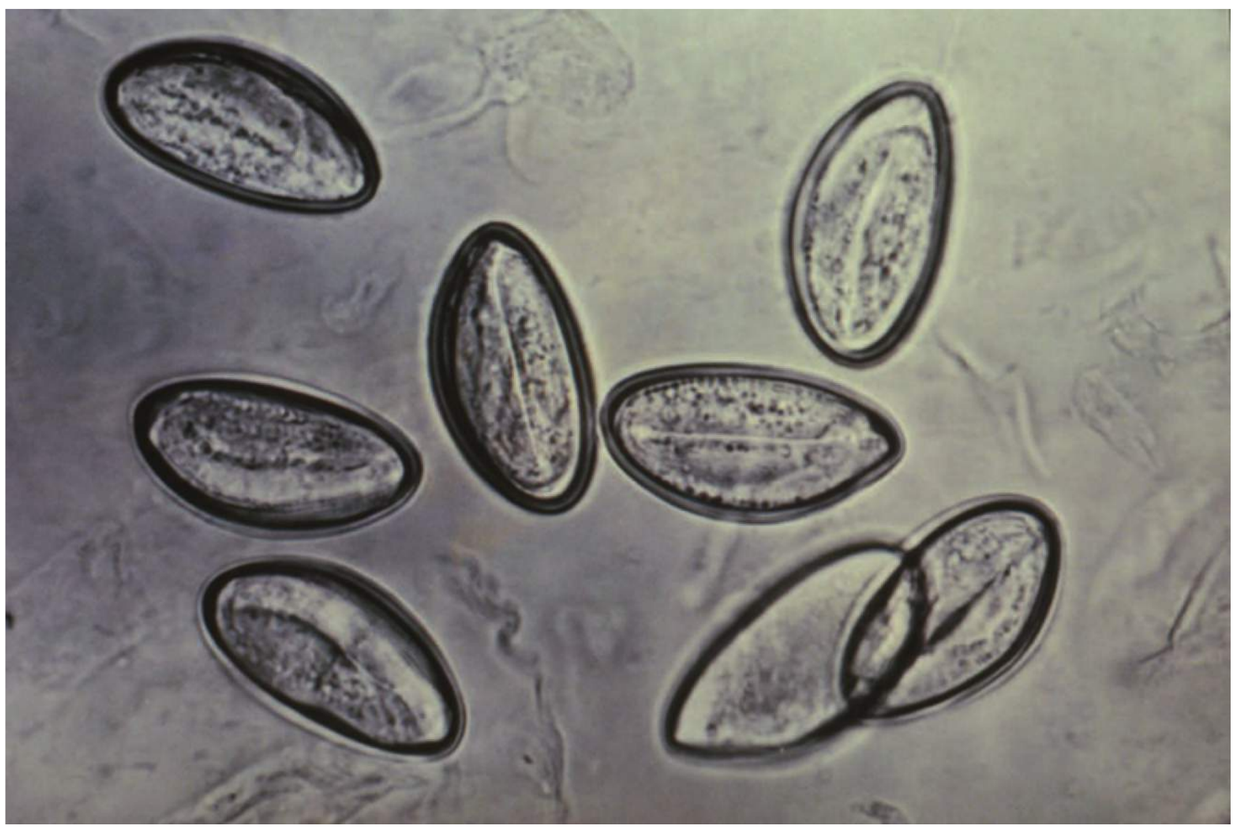


FIGURE 25.26 ■ Enterobius vermicularis. Characteristic appearance of the E vermicularis egg with contained larval form.

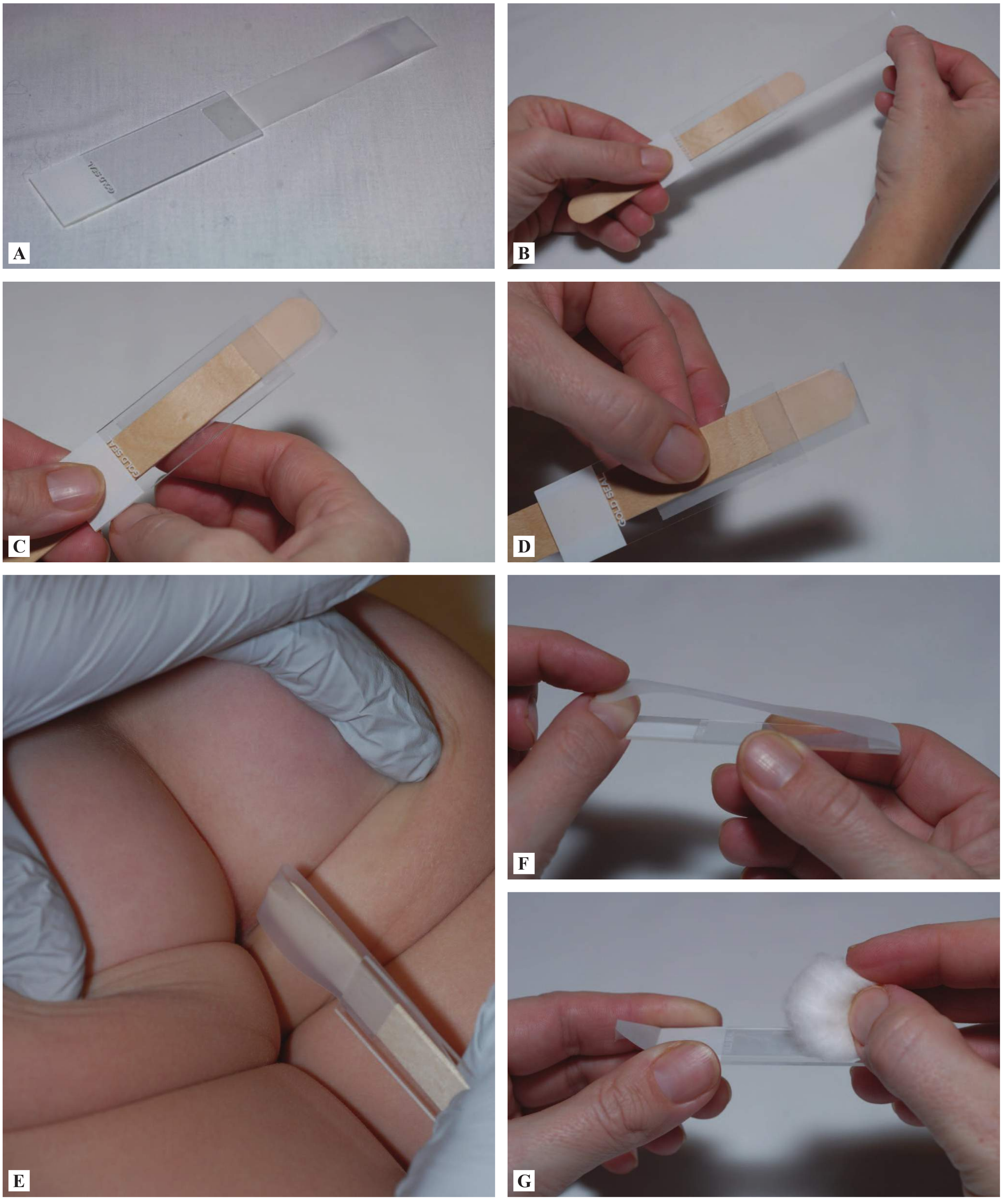


FIGURE 25.27 ■ **Cellophane Tape Preparation.** Steps for obtaining the eggs of E vermicularis. See text for details. (Photo contributor: Lawrence B. Stack, MD.)

TZANCK PREPARATION

Uses

To detect the multinucleated giant cells confirming the presence of a herpes infection.

Materials

Microscope, Bunsen burner, glass microscope slide, 5% methylene blue or Wright stain, or Geimsa stain, immersion oil, and sterile scalpel or hypdermic needle.

Method

1. Unroof a fresh, uncrusted vesicle with a sterile hypodermic needle or scalpel.
2. Scrape the floor of the vescicle with the scalpel and smear scrapings of the lesion onto a glass microscope slide.
3. Let air dry.
4. Fix specimen with absolute alcohol or gentle heat.
5. Stain with blue stain (5% methylene blue, Wright stain, Geimsa stain) for 5 seconds, rinse, and air dry.
6. View preparation through immersion oil at high power (40-50×).

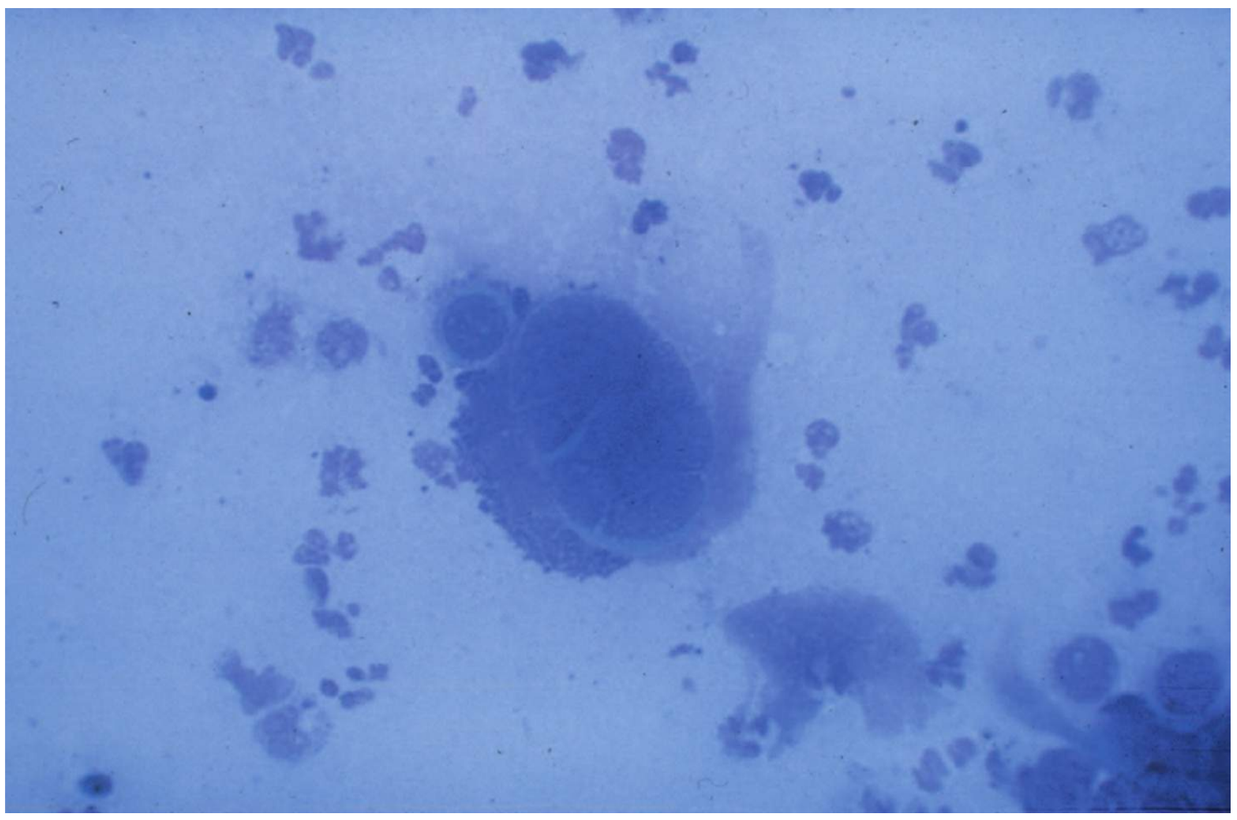


FIGURE 25.28 ■ **Tzanck Preparation.** A Tzanck preparation of both the roof and floor of a herpetic vesicle demonstrating a multinucleated giant cell. (Photo contributor: Department of Dermatology, Wilford Hall USAF Medical Center and Brook Army Medical Center, San Antonio, TX.)

INDEX

Note: Page numbers followed by "f" indicate figures; those followed by "t" indicate tables.

A
abdomen, anatomic boundaries of, 176
abdominal aorta, ultrasound of. See ultrasound, of abdominal aorta
abdominal aortic aneurysms, leaking, abdominal distention due to, 190f
abdominal bruises, 517f
abdominal conditions. See chest/abdominal conditions; specific conditions
abdominal distention, 189–191, 189f, 190f, 191f
abdominal evisceration, 176, 176f
abdominal hernias
  incisional, 184, 185f
  inguinal, 184, 184f, 185f
    pediatric, 490–491, 490f
  traumatic, 177, 177f
  umbilical, 186, 186f
abdominal injuries, due to child abuse, 517, 517f–518f
abdominal striae, 422, 422f
abdominal trauma, during third trimester of pregnancy, 271, 271f, 272f
abdominal ultrasound. See ultrasound, abdominal
abdominal wall hematoma, 192, 192f, 193f
abortion
  completed, 254
  incomplete, 254
  inevitable, 254
  septic, 254
  spontaneous, 254, 254f
  threatened, 254
abrasion, corneal, 82–83, 82f, 83f
abscesses
  anorectal, 238, 238f, 239f
  Bartholin gland, 253, 253f
  cold, 749, 749f, 762f
  odontogenic. See odontogenic infections
  peritonsillar, 123, 123f, 124f
  pilonidal, 244, 244f
  retropharyngeal, pediatric, 475–476, 475f, 476f
  scrotal, 210, 210f
  septal, 119, 119f
  soft tissue ultrasound for, 921f
abuse. See child abuse
abusive head trauma (AHT), 508
Acanthamoeba sp., free-living ameba infection due to, 716
acetaminophen poisoning, 599, 599f
acetazolamide, for acute angle-closure glaucoma, 43, 79
Achilles tendonitis, 366, 366f
Achilles tendon rupture, 327, 327f
acid exposures
  burns due to, 618, 620, 620f, 621f
  ocular, 98, 98f
acid tooth erosion, 155, 155f
acquired immunodeficiency syndrome. See HIV infection
acrocyanosis, 368, 368f
acromioclavicular joint separation, 286, 286f, 287f
acute myocardial infarction, electrocardiogram abnormalities in, 806–809, 806f–809f
  in anterior infarction, 806, 806f
  in inferior infarction, 807, 807f
  in posterior infarction, 809, 809f
  in right ventricular infarction, 808, 808f
  Sgarbossa criteria in setting of left bundle branch block and, 811, 811f
acute necrotizing ulcerative gingivitis, 154, 154f
  in HIV patients, 714, 714f
acute otitis media, 100, 100f, 102f
acyclovir
  for conjunctivitis, neonatal, 30, 31t
  for herpes simplex infections
    genital, 230–231
    ocular, 51
  for herpes zoster infections
    in HIV patients, 704
    in pediatric shingles, 451
  for herpetic gingivostomatitis, 454
  for varicella (chickenpox), 450
adhesives, for wound closure, 659, 660f
Adie pupil, anisocoria with, 48
adrenocorticotropic hormone, for crystal-induced synovitis, 348
afferent pupillary defect, 55, 55f, 56f
AIDS. See HIV infection
airway interventions, 765–803. See also cricothyrotomy; intubation
  alternatives to intubation, 766, 766f
  alternatives to rapid-sequence induction, 769, 769f
  bag-valve-mask ventilation, 770–771, 770f, 771f
  ear to sternal notch alignment and, 770, 770f
  laryngeal mask airway, 787–788, 787f, 788f
  nasal airways and, 771f
  oral airways and, 771f
  patient positioning for, 768, 768f
  requesting assistance for, 767
albendazole
  for cutaneous larva migrans, 724
  for elephantiasis, 730
  for pinworm infection (enterobiasis), 492

alcohol toxicity, 602–603, 602f, 603f
alkali burns, 618, 618f, 619f
  ocular, 98, 98f
allergic conjunctivitis, 35, 35f
allergic contact dermatitis, 402, 402f
altitude injuries
  cerebral edema, 538, 538f
  pulmonary edema, 537, 537f
  retinal hemorrhage, 536, 536f
alveolar ridge fractures, 141, 141f
amatoxin poisoning, 629, 629f, 630f
Amblyomma americanum, 562, 563f
ameba infection, 716, 716f
American dog tick, 562, 563f
American trypanosomiasis, 721, 721f
amikacin, for mycetoma, 745
amniotic fluid, fern pattern of, 936, 936f
amoxicillin
  for impetigo, 448
  for nursing bottle caries, 485
  for scarlet fever, 459
amoxicillin-clavulanate
  for dacryoadenitis, 37
  for mandibular fractures, 16
  for mastoiditis, 108
  for nasal cellulitis, 120
amoxicillin-sulbactam, for nasal cellulitis, 120
amphetamine toxicity, 588, 588f, 589f
ampicillin, for epidemic meningitis, 732
ampicillin-sulbactam
  for bite wounds, 654
  for dacryoadenitis, 37
amputations, traumatic, 338, 338f, 339f
anal fissures, 237, 237f
Ancylostoma braziliense, cutaneous larva migrans due to, 724
Ancylostoma caninum, cutaneous larva migrans due to, 724
Androctonus envenomation, 560
anemia, 717, 717f
aneurysms
  aortic, abdominal, leaking, abdominal distention due to, 190f
  coronary artery, in Kawasaki disease, 497, 499f
  left ventricular, electrocardiogram abnormalities in, 817, 817f
angioedema, 115, 115f, 116f, 767, 767f
  of uvula, 127, 127f
angiotensin-converting enzyme inhibitors, angioedema induced by, 115, 115f, 116f
angle-closure glaucoma, acute, 43, 43f, 44f
animal attacks, 549, 549f
animal bites, 654–655, 654f, 655f
anisocoria, 48, 48f
ankles
  dislocation of, 328, 328f, 329f
  fracture, 330, 330f, 331f
  sprain of, 332, 332f
anorectal abscesses, 238, 238f, 239f
anorectal conditions, 237–247. See also rectal conditions; specific conditions
  abscesses, 238, 238f, 239f
  anal fissure, 237, 237f
  hemorrhoids, 240–241, 240f, 241f, 242f
  melena, 247, 247f
  pilonidal abscess, 244, 244f
antacids, for hydrofluoric acid burns, 620, 621f
ant envenomation, 564, 564f, 565f
anterior ischemic optic neuropathy, funduscopic findings in, 78, 78f
anthozoan envenomation, 574
anthrax, cutaneous, 718, 718f, 719f
anticholinergic toxidrome, 594, 594f
anticoagulants poisoning, 616–617, 616f, 617f
antidepressants, tricyclic
  electrocardiogram abnormalities and, 856, 856f
  poisoning by, 604, 604f, 605f
antifreeze toxicity, 602–603, 602f, 603f
antimuscarinic toxidrome, 594, 594f
antivenins
  for coral snake envenomations, 550
  for pit viper envenomations, 551
  for scorpion envenomations, 561
  for stone fish envenomation, 578
  for tropical snake envenomation, 554–555
antral nipple sign in hypertrophic pyloric stenosis, 443f
aortic aneurysms, abdominal, leaking, abdominal distention due to, 190f
aphthous ulcers, oral, 158, 158f
apraclonidine, for acute angle-closure glaucoma, 43, 79
arrhythmias. See also electrocardiogram abnormalities, rhythm disturbances
  digitalis toxicity and, 631
arsenic poisoning, 622, 622f, 623f
arterial embolization, 369, 369f
artesunate, for malaria, 742
ascariasis, 720, 720f
Ascaris lumbricoides, ascariasis due to, 720
ascites, abdominal distention due to, 189, 189f
Ashman phenomenon, 829, 829f
Aspergillus niger, otitis externa due to, 106, 106f
asphyxia, traumatic, 166, 166f
aspirin
  for allergic conjunctivitis, 35
  bezoar, 600f
  poisoning, 600–601, 601f

assault, injuries due to, 679–687. See also specific injuries
blunt-force pattern of, 682–683, 682f
sharp-force pattern of, 679, 679f, 680f
strangulation pattern of, 684–685, 684f, 685f
thermal pattern of, 686, 686f, 687f
assessment of wounds, 642, 642f, 643f
atopic dermatitis, 388, 388f, 389f
atrial fibrillation, 833, 833f
atrial flutter, 834, 834f
atrial tachycardia, multifocal, 835, 835f
atrioventricular block. See AV block
atropine therapy, for cholinergic toxicity, 595, 596f
auricular perichondritis, 109, 109f, 110f
AV block
first-degree, electrocardiogram abnormalities in, 819, 819f
Mobitz I second-degree, electrocardiogram abnormalities in, 820, 820f
Mobitz II second-degree, electrocardiogram abnormalities in, 821, 821f
third-degree, electrocardiogram abnormalities in, 822, 822f
avulsion injuries
of ear, 20, 20f
partial, 646, 647f
of teeth, 138, 138f
azithromycin
for bacterial conjunctivitis, 32
for cat-scratch disease, 472
for chancroid, 233
for chlamydia infection, 229
for conjunctivitis, neonatal, 31t
for lymphogranuloma venereum, 232
for trachoma, 757

B

babesiosis, 562
Bacillus anthracis (anthrax), 718, 718f, 719f
bacitracin, for bacterial conjunctivitis, 32
background diabetic retinopathy, funduscopic appearance in, 71, 71f
background fundus, normal, funduscopic appearance of, 62, 62f
bacteria. See also specific bacteria
in urine, 925f
bacterial conjunctivitis, 30–32, 30f–31f, 31t, 32f, 227–228, 228f, 229f
bacterial keratitis, 52, 52f–54f
bacterial tracheitis, pediatric, 478, 478f
bacterial vaginitis, 250, 251, 251f
Bacteroides sp.
Ludwig angina due to, 152
necrotizing fasciitis due to, 346
bagging, 608, 609f
bag-valve-mask ventilation, 770–771, 770f, 771f
Baker cyst, ruptured, 363, 364f
Balamuthia mandrillaris, free-living ameba infection due to, 716
balanitis, 219, 219f, 220f
balanoposthitis, 219, 219f, 220f
barotitis media, 568, 568f
Bartholin abscess, 253, 253f
in gonorrhea, 228f
Bartonella henselae, cat-scratch disease due to, 472, 472f
Barton fractures, 296, 297f
basal cell carcinoma, 430, 430f
basilar skull fractures, 4, 4f–6f, 5
basophilic stippling, 625f
bath salts, 590, 590f
batteries, pediatric ingestion of, 477, 477f
Battle sign, 4, 4f
bear, attack by, 549, 549f
beau lines, in Kawasaki disease, 497, 498f
Beck Airway Airflow Monitor, 798, 798f
bee envenomation, 564, 565f
bell-clapper deformity, 202, 202f, 203f
Bell palsy, 113–114, 113f, 114f
Bennett fractures, 305, 305f, 306f
benzodiazepines
for amphetamine toxicity, 588
for cocaine toxicity, 591
for tetanus, 755
biceps tendon rupture, 293, 293f
Bi-Level Positive Airway Pressure (BiPAP), 766, 766f
bites. See also envenomations
animal, 549, 549f, 654–655, 654f, 655f
human, 502, 503f, 504f, 654
leech, 735, 735f, 736f
black hairy tongue, 162, 162f
black widow spider envenomation, 557, 557f
bladder
cystocele and, 263, 263f
ultrasound, 903–905, 903f–906f
blade insertion, in laryngoscopic intubation, 775
blast cells, testing for, 937, 937f
blepharitis, 39, 40f
herpes simplex, 50
blind nasotracheal intubation, 796–798, 796f–798f
blister(s), fractures, 326, 326f
blistering distal dactylitis, pediatric, 461, 461f
b-Blocker toxicity, 606, 606f
blood smear test, for schistocytes or blast cells, 937, 937f
bloody chemosis, 84, 84f
blowout fractures, orbital, 13, 13f–15f
blue-dot sign, 204, 204f

blue-gray sclera, 515f
blue-ringed octopus envenomation, 581, 581f
blunt-force-pattern injuries, 682–683, 682f
body lice, pediculosis due to, 236, 236f
body surface area, estimation of percentage burned, 666
Borrelia burgdorferi, 562
  erythema chronicum migrans due to, 407, 407f
botulism, 615, 615f
bougie-assisted intubation, 781–783, 781f–783f
boutonnière deformity, 307, 307f
"boxcar" appearance, in central retinal artery occlusion, 67, 67f
boxer's fractures, 303, 303f
brain. See also central nervous system
  herniation syndromes of, 26, 26f, 27f
  injury, in shaken baby syndrome, 508, 508f
branchial cleft cyst, pediatric, 469, 469f
"breakbone fever," 727–728, 727f, 728f
breech delivery, 277, 277f
brimonidine, for acute angle-closure glaucoma, 43
brown recluse spider envenomation, 558–559, 558f, 559f
Brugada syndrome, electrocardiogram abnormalities in, 847, 847f
Brugia sp., elephantiasis due to, 730
bruises
  due to child abuse, 502–503, 503f, 504f, 505f, 511
  hymenal, 524f
  ligature, 504f, 505f
  pinna, 511f
  scrotal, 524f
buccal space, abscess of, 150, 150f
bucket-handle fractures, 515f
bulimia, acid tooth erosion in, 155, 155f
"bullet wipe," 675, 675f
bullous diseases, autoimmune, 425, 425f
bullous impetigo, pediatric, 448, 448f
bullous myringitis, 103, 103f
bullous pemphigoid, 425, 425f
burns
  acid, 618, 620, 621f
  due to abuse, 502–503, 502f, 506f, 507f, 686, 686f, 687f
  estimating body surface area of, 666
  lightning-induced, 547–548, 547f, 548f
  oropharyngeal, due to caustic ingestion, 618, 618f
bursitis, 353, 353f
Buruli ulcer, 749, 749f
button batteries (disk), pediatric ingestion of, 477, 477f

C

calcaneal fractures, 333, 333f
calcium carbonate, for hydrofluoric acid burns, 620
calcium-channel blocker poisoning, 606, 607f
calcium gluconate, for hydrofluoric acid burns, 620
calcium oxalate crystals
  in ethylene glycol poisoning, 603f
  houseplants with, 633, 633f
  in urine, 924, 924f
calcium pyrophosphate crystals, in synovial fluid, 926, 927f
Candida sp.
  epiglottitis (supraglottitis) due to, 473
  erythema toxicum due to, 436
  in HIV patients, 693
  oral infection by, 156, 156f
  thrush due to, 117
  vaginitis due to, 250, 250f, 251, 251f
Candida albicans
  in HIV patients, 693
  otitis externa due to, 106
  potassium hydroxide mount demonstrating, 931, 931f
  uvulitis due to, 127
canine space abscesses, 151, 151f
canker sores, 158, 158f
cannabinoids, toxicological conditions due to, 590, 590f
capitellum fractures, 290, 291
Capnocytophaga canimorsus, in dog bite wounds, 654, 655
caput medusae, 183, 183f
carbon monoxide poisoning, 612–613, 612f
cardiac glycoside plant ingestion, 631, 631f, 632f
cardiac tamponade, 172, 172f
cardiac ultrasound. See ultrasound, of cardiac
carpal dislocations, 298, 298f–300f
carpometacarpal dislocations, 298, 298f–300f
castor bean ingestion, 636, 636f
cataracts, posttraumatic, 95, 95f
cat bites, 654, 655f
caterpillar envenomation, 566, 567f
Catha edulis (khat), 590, 590f
cat-scratch disease, pediatric, 472, 472f
cauliflower ear, 19, 19f, 650
caustic exposures, ocular, 98, 98f
caustic ingestion, 618, 618f, 619f
cefixime, for gonorrhea, 227
cefotaxime
  for gonorrhea, 227
  for mastitis, 440
cefoxitin, for bite wounds, 654
cefpodoxime, for gonorrhea, 227
ceftizoxime, for gonorrhea, 227
ceftriaxone
  for bite wounds, 654
  for chancroid, 233
  for conjunctivitis, neonatal, 31t
  for gonorrhea, 227
  for leptospirosis, 741
  for meningococcemia, 455

cellular asphyxiants, 612–613, 612f, 613f
cellulitis, 342, 342f, 921f
  nasal, 120, 120f, 121f
  orbital and periorbital, pediatric, 466, 466f, 467f, 468f
  of scalp, dissecting, 378, 378f
centipede envenomation, 566, 567f
central nervous system. See also brain
  Toxoplasma gondii infection of, in HIV patients, 697, 697f, 698f
central retinal artery
  normal, funduscopic appearance of, 62, 62f
  occlusion of, funduscopic findings in, 67, 67f–68f
central retinal vein
  normal, funduscopic appearance of, 62, 62f
  occlusion of, funduscopic findings in, 69, 69f
Centruroides envenomation, 560–561, 560f, 561f
cephalopod envenomation, 581, 581f
cephalosporin
  for dacryocystitis, 36
  for epidemic meningitis, 732
  for impetigo, 448
  for mastitis, 440
  for mastoiditis, 108
cercarial dermatitis, 577, 577f, 753, 754f
"cerebral" T waves, 815, 815f
cerebrospinal fluid
  leakage of, 4, 6f
  microscopic examination of, 934, 934f
cervical polyps, 252, 252f
cervical radiculopathy, 360, 360f
cervical trauma, 255–256, 256f
cervicitis
  chlamydial, 229, 229f
  in gonorrhea, 227, 228f
Chagas disease, 721, 721f
chalazion, 39, 40f
chancre, syphilitic, 224, 224f
chancroid, sexually transmitted, 233, 233f
chauffeur's fractures, 296, 297f
"cheese and ketchup" appearance, in cytomegalovirus retinitis, 75, 75f
chemical exposure, of eye, 98, 98f
chemosis, bloody, 84, 84f
cherry-red spot, in central retinal artery occlusion, 67, 67f
chest/abdominal conditions, 165–199. See also specific conditions
  abdominal distention, 189–191, 189f, 190f, 191f
  abdominal evisceration, 177, 177f
  abdominal wall hematoma, 193, 193f, 194f
  asphyxia, traumatic, 166, 166f
  caput medusae, 184, 184f
  cardiac tamponade, 172, 172f
  clavicle fractures, 169
  Cullen sign and, 175, 175f
  Eagle-Barrett syndrome, 198, 198f, 199f
  flail chest, 167, 167f
  gravid abdomen, 190, 191f
  Grey Turner sign and, 175, 175f
  hernias. See hernias
  impaled foreign bodies, 176, 176f
  jugulovenous distention, 183, 183f
  lung masses, apical, 182, 182f
  patent urachal duct, 188, 188f
  pectus carinatum, 195, 196f
  pectus deformities, 195, 195f, 196f
  pectus excavatum, 195, 195f
  pelvic fractures, 179, 179f
  pneumothorax, tension, 170, 170f
  prune belly syndrome, 198, 198f, 199f
  respiratory retractions, 180, 180f
  seat belt injury, 174, 174f
  Sister Mary Joseph node (nodular umbilicus), 189, 189f
  sternoclavicular dislocation, 168, 168f, 169f
  stoma prolapse, 197, 197f
  superior vena cava syndrome, 181, 181f
  supraclavicular adenopathy, 182, 182f
  thoracotomy and, 172, 172f
chewing tobacco, tooth erosion due to, 155, 155f
chickenpox, pediatric, 450, 450f
chigger envenomation, 566, 567f
chilblains, 542, 542f
child(ren). See child abuse; pediatric conditions
child abuse, 502–533
  facial findings, physical, 511–512
  oropharyngeal injuries in, 511, 512f
  pattern injuries in. See pattern injuries, in abuse
  physical, 502–518
    bruises. See bruises, due to child abuse
    burns. See burns, due to abuse
    fractures suggestive of, 514, 514f, 515f, 516f
    shaken baby syndrome and, 508, 508f, 509f
  sexual. See sexual abuse, of children
Chironex fleckeri envenomation, 575
Chlamydia trachomatis infection
  in Bartholin gland abscess, 253
  cervicitis due to, 229, 229f
  conjunctivitis due to, 30–31, 31t
  lymphogranuloma venereum due to, 232
  sexually transmitted, 229, 229f, 232, 524
  trachoma due to, 757
"chocolate blood," 611f
cholera, 722–723, 722f, 723f
cholesteatomas, of ear, 104, 104f
cholesterol emboli, 66, 66f

cholinergic toxidrome, 595, 595f, 596f
chronic plaque, 384, 384f, 385f
cigarette burns, due to child abuse, 507f
"ciliary flush" in anterior uveitis, 45, 46f
ciprofloxacin
  for anthrax, cutaneous, 718
  for bite wounds, 654
  for chancroid, 233
  for meningococcemia, 455
circinate rings, 64
  in diabetic retinopathy, 71f
classic metaphyseal lesion, due to child abuse, 514f–515f
clavicle fractures, 169
clavulanic acid, for impetigo, 448
claw hand deformity, 304, 304f
cleaning of wounds, 640–641, 640f, 641f
clenched fist injuries, 302, 302f
clindamycin
  for bite wounds, 654
  for blistering distal dactylitis, 461
  for dacryocystitis, 36
  for impetigo, 448
  for mandibular fractures, 16
  for nasal cellulitis, 120
  for retropharyngeal abscess, 475
  for scarlet fever, 459
clofazimine, for leprosy, 739
Clostridium perfringens myonecrosis, 345, 345f
Clostridium tetani, tetanus due to, 755
clotrimazole, for vaginitis, 251
clubbing, 361, 361f
clue cells, vaginal fluid wet mount demonstrating, 930, 930f
"cobblestones" in allergic conjunctivitis, 35, 35f
cocaine toxicity, 591, 591f–593f, 593
codeine, for acute sickle dactylitis in, 482
coelenterate envenomation, 574–575, 574f, 576f
coining (coin rubbing), marks left by, 505f, 758f
colchicine, for crystal-induced synovitis, 348
cold abscesses, 749, 749f, 762f
cold injuries, 539–543
  chilblains, 542, 542f
  frostbite, 540–541, 540f, 541f
  hypothermia, 539, 539f
  immersion injury (trench foot), 543, 543f
  pernio (perniosis or chilblains), 542, 542f
cold panniculitis, pediatric, 453, 453f
cold sores, herpetic, 157, 157f
cold urticaria, 400, 401f
Colles fractures, 296, 296f, 297f
coloboma, of iris, 94, 94f
Colorado tick fever, 562
comet tail abrasion collar, 672, 672f
compartment syndrome, of upper extremity, 314, 314f–315f
complete AV block, electrocardiogram abnormalities in, 822, 822f
completed abortion, 254
compression injuries, 646, 649f
  vertebral fractures, 516f
computed tomography, of peritonsillar abscess, 124f
conduction disturbances. See electrocardiogram abnormalities, conduction disturbances
condyloma acuminata, 234–235, 234f, 235f
  pediatric, due to sexual abuse, 524, 525f, 526f
condyloma lata
  pediatric, due to sexual abuse, 526f
  in secondary syphilis, 224–226, 226f
conjunctivitis
  allergic, 35, 35f
  bacterial. See bacterial conjunctivitis
  chlamydial, 229f
  epidemic keratoconjunctivitis, 33, 34f
  in gonorrhea, 227–228, 228f
  in Kawasaki disease, 497, 497f
  neonatal (ophthalmia neonatorum), 30–31, 30f–31f, 31t
  vernal, 35, 35f
  viral, 33, 33f
    herpes simplex, 50
contact burns, due to child abuse, 502, 506f
contact dermatitis, allergic, 402, 402f
contusions
  muzzle, 674f, 675
  pattern, 682–683, 682f, 683f
  of pinna, 19f
convex array ultrasound transducer, 861t
copperhead envenomation, 551, 552f
coral envenomation, 574–575, 574f
coral snake envenomation, 550, 550f
corneal abrasion, 82–83, 82f, 83f
corneal foreign bodies, 85, 85f
corneal-scleral rupture, 94, 94f
corneal ulcers, 52, 52f–54f
corner stitch, 661, 662f, 663f
Corynebacterium diphtheriae
  diphtheria due to, 122, 122f
  pharyngitis due to, 117
cottonmouth envenomation, 551, 552f
cotton swab technique, 522f
cotton wool spots, 64, 64f
  in central retinal vein occlusion, 69, 69f
cougar, mauling by, 549, 549f
coxsackievirus A16, for hand, foot, and mouth disease, 452, 452f
crab lice, pediculosis due to, 236, 236f
crack pipe, 591f
"cradle cap," in seborrheic dermatitis, 383, 383f

cranial findings, physical child abuse, 508
"creeping eruption," 724
cricothyrotomy
  open, 803, 803f
  Seldinger technique for, 801–802, 801f, 802f
"Crigler" massage, for dacryocystitis, 36
Crocq disease, acrocyanosis, 368, 368f
cromolyn, for allergic conjunctivitis, 35
crush injuries, 646, 649f
cryptococcal meningitis, in HIV patients, 700, 700f, 701f
Cryptococcus neoformans
  cerebrospinal fluid examination, 934
  infection in HIV patients, 700, 700f, 701f
crystal-induced synovitis, 348, 348f, 349f
Cullen sign, 175, 175f
cupping, marks left by, 505f
curvilinear array ultrasound transducer, 861t
Cushing syndrome, 422, 422f
cutaneous conditions, 371–432. See also specific conditions
  abdominal striae, 422, 422f
  angioedema, 115, 115f, 116f
  basal cell carcinoma, 430, 430f
  blistering distal dactylitis, pediatric, 461, 461f
  branchial cleft cyst, pediatric, 469, 469f
  bullous diseases, autoimmune, 425, 425f
  cat-scratch disease, pediatric, 472, 472f
  cold panniculitis (popsicle panniculitis), pediatric, 453, 453f
  Cryptococcus neoformans infection, in HIV patients, 700, 700f, 701f
  cystic hygroma, pediatric, 471, 471f
  in dengue fever, 727–728, 727f, 728f
  dermatitis. See dermatitis
  dermatographism, 400, 401f
  dermatophyte (tinea) infections, in HIV patients, 711, 711f
  discoid lesions, 426, 426f
  dissecting cellulitis of the scalp, 378, 378f
  disseminated gonococcus, 409, 409f
  in dracunculiasis, 729, 729f
  drug eruptions. See drug eruptions
  ecthyma gangrenosum, 411, 411f
  eczema. See eczema
  eosinophilic folliculitis, in HIV patients, 706, 706f
  erysipelas, 404, 404f
  erysipeloid ("fish handler's disease"), 582, 582f
  erythema chronicum migrans, 407, 407f
  erythema infectiosum (fifth disease), pediatric, 446, 446f
  erythema multiforme, 374, 374f, 375f
  erythema nodosum, 403, 403f
  febrile neutrophilic dermatosis, 427, 427f
  folliculitis, hot tub, 410, 410f
  hand, foot, and mouth disease, pediatric, 452, 452f
  herpes gladiatorum, 412, 412f
  herpes zoster infection, 413, 413f. See also herpes zoster infection
  herpetic gingivostomatitis, pediatric, 454, 454f
  herpetic whitlow, 412, 412f
  hidradenitis suppurativa, 379, 379f
  hyperpigmentation, arsenic poisoning and, 622, 622f
  id reaction. See id reaction
  impetigo, pediatric, 448, 448f
  in infective endocarditis, 405, 405f, 406f
  intertrigo, 418, 418f
  jaundice, 420, 420f
  Kaposi sarcoma, in HIV patients, 695, 695f, 696f
  kerion, 414, 415f
  larva migrans, 724, 724f
  in leishmaniasis, 737–738, 737f
  in leprosy, 739–740, 739f, 740f
  leukocytoclastic vasculitis, 396, 396f, 397f
  lip licker dermatitis, 432, 432f
  livedo reticularis, 394, 394f, 395f
  maculopapular rash, in HIV patients, 690, 690f
  measles, pediatric, 449, 449f
  melanoma, 431, 431f
  melasma, 421, 421f
  meningococcemia, pediatric, 455, 455f
  in mycetoma, 745, 745f, 746f
  nongenital herpes simplex infection, 412, 412f
  onychomycosis, 417, 417f
  orbital and periorbital cellulitis, pediatric, 466, 466f, 467f, 468f
  paraneoplastic pemphigus, 425, 425f
  pediatric, Henoch-Schönlein purpura, 462, 462f
  pemphigus vulgaris, 425, 425f
  physical child abuse, 502–503
  pityriasis rosea, 386, 386f, 387
  pityriasis versicolor, 416, 416f
  porphyria cutanea tarda, 428, 428f
  psoriasis, 384, 384f, 385f
  purpura. See purpura
  pyoderma gangrenosum, 382, 382f
  pyogenic granuloma, 380, 380f, 381f
  Rocky Mountain spotted fever, 408, 408f
  roseola infantum (exanthem subitum), pediatric, 447, 447f
  scabies, 419, 419f
    in HIV patients, 708–709, 708f, 709f
  scarlet fever, pediatric, 459, 459f, 460f
  in secondary syphilis, 224–226, 225f, 226f
  serum sickness-like reaction, pediatric, 463, 463f
  sporotrichosis, 760, 760f
  squamous cell carcinoma, 429, 429f
  staphylococcal scalded skin syndrome, pediatric, 458, 458f
  Stevens-Johnson syndrome, 372, 372f, 373f

cutaneous conditions (Cont.):
strawberry hemangioma, pediatric, 464, 464f, 465f
summer penile syndrome, pediatric, 457, 457f
Sweet syndrome, 427, 427f
systemic lupus erythematosus, 426, 426f
lip licker, 432, 432f
thyroglossal duct cyst, pediatric, 470, 470f
tinea capitis, 414, 415f
tinea corporis, 414, 414f
tinea cruris, 414, 415f
tinea faciale, 414, 414f
tinea pedis, 414, 415f
tinea unguium, 414
tinea versicolor, 416, 416f
toxic epidermal necrolysis, 372, 372f, 373f
uremic frost, 424, 424f
urticaria. See urticaria
of uvula, 127, 127f
varicella (chickenpox), pediatric, 450, 450f
vitiligo, 423, 423f
cutaneous larva migrans, 724, 724f
cyanide poisoning, 612–613, 613f
cyanosis, methemoglobinemia and, 610f
cyclosporin, for allergic conjunctivitis, 35
cyst(s), hydatid, 734, 734f
cysticercosis, 725–726, 725f, 726f
cystic hygroma, pediatric, 471, 471f
cystocele, 263, 263f
cytomegalovirus infections, in HIV patients, 703, 703f

D

dacryoadenitis, 37, 37f
dacryocystitis, 36, 36f
dactylitis
blistering distal, pediatric, 461, 461f
sickle (hand-foot syndrome), acute, pediatric, 482, 482f
dancer's fracture, 334, 334f, 335f
dapsone
for leprosy, 739
for mycetoma, 745
dark-field microscopy, 929, 929f
decubitus ulcers, treatment of, 670, 670f
deep venous thrombosis, 363, 363f, 364f
ultrasound of, 894–896, 894f–896f
deer ticks, 562, 562f
deferoxamine, for iron poisoning, 624
degloving injuries, 648f
dehydration, in cholera, 722f
delirium, anticholinergic, 594f
delivery, emergency, 273–285
breech, 277, 277f
crowning and, 273, 273f
normal sequence of, 274–276, 274f–276f
shoulder dystocia in, 285, 285f
umbilical cord prolapse and, 278, 278f
dengue fever, 727–728, 727f, 728f
dental conditions. See teeth
dental injury due to child abuse, 513f
depressed skull fractures, 7–8, 7f
de Quervain tenosynovitis, 355f
Dermacentor andersoni, 562, 562f
Dermacentor variabilis, 562, 563f
dermal closures, 658
dermal melanosis, 505f
dermatitis
atopic, 388, 388f, 389f
cercarial ("swimmer's itch"), 577, 577f
contact, allergic, 402, 402f
due to mango, 583, 585f
marine ("sea bather's eruption"), 577, 577f
seborrheic, 383, 383f
stasis, 393, 393f
Toxicodendron, 583, 583f, 584f, 585f
Dermatobia hominis, myiasis due to, 747, 747f, 748f
dermatographism, 400, 401f
dermatophyte infections, in HIV patients, 711, 711f
dermatoses, skin scraping for, 933, 933f
desquamating rash, in scarlet fever, pediatric, 459, 460f
dextrocardia, structural abnormalities in, 841, 841f
diabetic retinopathy, funduscopic findings in, 71, 71f, 72f
diarrhea, in cholera, 722–723, 723f
dicloxacillin, for impetigo, 448
dieffenbachia ingestion, 633, 633f
diethylcarbamazine, for elephantiasis, 730
digit(s)
clubbing of, 361, 361f, 762f
felon in, 343, 343f
frostbite of, 540–541, 540f, 541f
hair tourniquet, 483, 483f
ingrown toenail and, 350, 350f
tendon injuries of, 652, 652f, 653f
digital intubation, 792, 792f
digitalis toxicity, 631, 631f
digoxin
for arrhythmias, 631
for cardiac glycoside plant ingestion, 631
electrocardiogram abnormalities in toxicity of, 855, 855f
dimercaprol
for arsenic poisoning, 622
for lead poisoning, 625
for mercury poisoning, 627–628
diphenhydramine
for oculogyric crisis, 495
for vancomycin-induced red man syndrome, 614

diphtheria, 122, 122f
diplopia, with orbital blowout fractures, 13
discoid lesions, 426, 426f
dislocations. See specific sites
dissecting cellulitis of the scalp, 378, 378f
disseminated eczema, 392, 392f
disseminated histoplasmosis, 702, 702f
diving injuries
  mask squeeze, 569, 569f
  middle ear squeeze (barotitis media), 568, 568f
dogbane ingestion, 631
dog bites, 549, 549f, 654, 654f, 655
domestic violence, pattern injuries in. See pattern injuries, in abuse
dorzolamide, for acute angle-closure glaucoma, 43
doxycycline
  for anthrax, cutaneous, 718
  for chlamydia infection, 229
  for leptospirosis, 741
  for lymphogranuloma venereum, 232
  for syphilis, 244
Dracunculus medinensis, dracunculiasis due to, 729, 729f
drug eruptions
  exanthematous, 376, 376f
  fixed, 377, 377f
drug reactions, in HIV patients, 713, 713f
drusen, in age-related macular degeneration, funduscopic appearance of, 63, 63f
dry gangrene, 344, 344f
dumb cane ingestion, 633, 633f
Dupuytren contracture, 365, 365f
dyshidrotic eczema, 391, 391f

E

Eagle-Barrett syndrome, 198, 198f, 199f
ear injuries
  external, 19–20, 19f, 20f
  lacerations, 650, 650f
early repolarization, electrocardiogram abnormalities in, 816, 816f
ear, nose, and throat conditions, 99–134. See also specific conditions
  angioedema, 115, 115f, 116f
  auricular perichondritis, 109, 109f, 110f
  bullous myringitis, 103, 103f
  cholesteatoma, 104, 104f
  diphtheria, 122, 122f
  epiglottitis, 125–126, 125f, 126f
  facial nerve palsy, 113–114, 113f, 114f
  herpes zoster oticus (Ramsay Hunt syndrome), 111, 111f, 112f
  mastoiditis, 108, 108f
  mucocele (ranula), 128, 128f
  nasal cellulitis, 120, 120f, 121f
  otitis externa, 106, 106f
  otitis media, 100, 100f, 102f
  peritonsillar abscess, 123, 123f, 124f
  pharyngitis, 117, 117f, 118f
  preauricular sinus abscess, 107, 107f
  septal hematoma, 119, 119f
  sialoadenitis, 129–130, 129f, 130f
  sinusitis, 132, 132f, 133f
  tympanic membrane perforation, 105, 105f
  uvulitis, 127, 127f
ecchymosis
  on gravid abdomen, 271, 272f
  in raccoon eyes, 5f
  in sublingual hemorrhage, 17f
  with traumatic exophthalmus, 23, 23f
Echinococcus granulosus, hydatid cyst due to, 734
ecstasy (MDMA; 3,4-methylenedioxymethamphetamine), 588, 588f
ecthyma gangrenosum, 411, 411f
ectopic pregnancy, 266–268, 266f, 267f, 268f
eczema
  disseminated, 392, 392f
  dyshidrotic (pompholyx), 391, 391f
  nummular, 390, 390f
  xerotic, 390, 390f
edema
  angioedema, 115, 115f, 116f
    of uvula, 127, 127f
  cerebral, 538, 538f
  lymphedema, 352, 352f
  papilledema, funduscopic findings with, 76, 76f
  pitting, 352, 352f
  pulmonary. See pulmonary edema
  retinal, in central retinal vein occlusion, 69, 69f
Edwardsiella lineata, 577
E-FAST (Extended Focused Assessment with Sonography for Trauma), 863–875, 863f
  left upper quadrant (perisplenic view), 868–869, 868f, 869f
  pericardial (subxiphoid view), 864–865, 864f, 865f
  right upper quadrant (Morison pouch) view, 866, 866f, 867f
  suprapubic view, 870–872, 870f, 871f, 872f
  thorax (right and left) views, 873–875, 873f, 874f, 875f
"Eight-Ball" hyphema, 86, 86f
Eikenella corrodens, in human bite wounds, 654
elastic wound closure strips, for wound closure, 659
elbow
  dislocation of, 292, 292f
  fractures of, 290–291, 290f, 291f
  nursemaid's (radial head subluxation), pediatric, 486, 486f, 487f

elder abuse, thermal pattern injuries due to, 686
electrical injuries
lightning-induced, 547–548, 547f, 548f
treatment of, 668, 668f, 669f
electrocardiogram abnormalities, 805–858
conduction disturbances, 819–829
Ashman phenomenon, 829, 829f
first-degree AV block, 819, 819f
left anterior fascicular block, 826, 826f
left bundle branch block, 825, 825f
left posterior fascicular block, 827, 827f
Mobitz I second-degree AV block, 820, 820f
Mobitz II second-degree AV block, 821, 821f
QT interval prolongation, 823, 823f
right bundle branch block, 824, 824f
third-degree AV block, 822, 822f
of non-cardiac origin, 849–858
in digoxin toxicity, 855, 855f
in hyperkalemia, 851–852, 851f, 852f
in hypokalemia, 850, 850f
in hypothermia, 849, 849f
limb lead reversal and, 857, 857f
low voltage and, 858, 858f
in pericardial effusion, 854, 854f
in pulmonary embolism, 853, 853f
tricyclic antidepressants and, 856, 856f
rhythm disturbances, 830–840
atrial fibrillation, 833, 833f
atrial flutter, 834, 834f
junctional rhythm, 830, 830f
multifocal atrial tachycardia, 835, 835f
paced rhythm, 832, 832f
supraventricular tachycardia, 836, 836f
supraventricular tachycardia with aberrancy, 837, 837f
torsades de pointes, 840, 840f
ventricular flutter, 839, 839f
ventricular rhythm, 831, 831f
ventricular tachycardia, 838, 838f
structural abnormalities and, 841–848
Brugada syndrome, 847, 847f
dextrocardia, 841, 841f
hypertrophic cardiomyopathy, 846, 846f
left atrial hypertrophy, 844, 844f
left ventricular hypertrophy, 842, 842f
right atrial hypertrophy, 845, 845f
right ventricular hypertrophy, 843, 843f
Wolff-Parkinson-White syndrome, 848, 848f
ST-T, 806–818
acute myocardial infarction and, 806–809, 806f–809f
“cerebral” T waves, 815, 815f
early repolarization and, 816, 816f
hyperacute T waves, 813, 813f
with left main lesion, 810, 810f
left ventricular aneurysm and, 817, 817f
pericarditis and, 818, 818f
Sgarbossa criteria in setting of left bundle branch block and, 811, 811f
subendocardial ischemia and, 812, 812f
Wellens waves, 814, 814f
elephantiasis, 730–731, 730f, 731f
emboli
cholesterol, funduscopic findings with, 66, 66f
mercurial, 627f
encephalitis, amebic, granulomatous, 716
endocarditis, 405, 405f, 406f
endocavitary probe ultrasound transducer, 861t
endotracheal tube, 778–779, 779f, 780f
Enterobacter sp., cellulitis due to, 342
enterobiasis, pediatric, 492, 492f
Enterobius vermicularis
pediatric, 492, 492f
tape test for eggs of, 938, 938f, 939f
envenomations. See also specific envenomations
anthozoan, 574
black widow spider, 557, 557f
brown recluse spider, 558–559, 558f, 559f
caterpillar, 566, 567f
centipede, 566, 567f
chigger, 566, 567f
coelenterate, 574–575, 574f, 576f
cone snail, 579, 579f
hymenoptera, 564, 564f, 565f
lion fish, 578, 578f
scorpion, 560–561, 560f, 561f
scorpion fish, 578, 578f
scyphozoan, 574, 575f
sea anemone, 574, 576f
sea urchin, 572–573, 572f, 573f
snake bites. See snake bites
stingray, 570–571, 570f, 571f
stone fish, 578, 578f
tick, 562
environmental conditions, 535–586
cold-induced. See cold injuries
envenomations. See envenomations; snake bites
high-altitude
cerebral edema, 538, 538f
pulmonary edema, 537, 537f
retinal hemorrhage, 536, 536f
intoxication. See toxicological conditions
large animal attacks, 549, 549f
lightning injuries, 547–548, 547f, 548f
mango dermatitis, 583, 585f
marine. See marine injuries/conditions

sporotrichosis, 586, 586f
sun exposure, 544, 544f, 545f, 546f
tick bites, 562, 562f, 563f
Toxicodendron exposure, 583, 583f, 584f, 585f
eosinophilic folliculitis, in HIV patients, 706, 706f
epidemic keratoconjunctivitis, 33, 34f
epidemic meningitis, 732, 732f
epididymal appendix, torsion of, 204, 204f
epididymitis, 205, 205f
epiglottis
camouflage, 776f
visualization during laryngoscopy, 776f
epiglottitis, 125–126, 125f, 126f
pediatric, 473, 473f, 474f
epiglottoscopy, 775
episcleritis, 42, 42f
epistaxis, 9
Epstein-Barr virus infection, in HIV patients, 692, 692f
erysipelas, 404, 404f
erysipeloid, 582, 582f
Erysipelothrix rhusiopathiae infections, 582, 582f
erythema chronicum migrans, 407, 407f
erythema infectiosum, pediatric, 446, 446f
erythema migrans rash, 563f
erythema multiforme, 374, 374f, 375f
erythema nodosum, 403, 403f
erythema toxicum, 436, 436f
erythromycin
for anthrax, cutaneous, 718
for bacterial conjunctivitis, 32
for chlamydia infection, 229
for conjunctivitis, neonatal, 30, 31t
for diphtheria, 122
for scarlet fever, 459
erythromycin base
for chancroid, 233
for lymphogranuloma venereum, 232
erythromycin ethylsuccinate, for chlamydia infection, 229
Escherichia coli
in Bartholin gland abscess, 253
cellulitis due to, 342
Eschmann stylet, 781–783, 781f–783f
esophageal candidiasis, in HIV patients, 693, 693f
esophagitis, herpes simplex virus, in HIV patients, 707
esophagus
caustic ingestion and, 618, 619f
visualization during laryngoscopy, 777f
ethmoid sinusitis, 132
ethylene glycol toxicity, 602–603, 602f, 603f
evil eye make-up, 758f
evisceration, abdominal, 176, 176f
exanthematous drug eruption, 376, 376f
exanthem subitum, pediatric, 447, 447f
exophthalmus, traumatic, 23, 23f, 24f
external ear injuries, 19–20, 19f, 20f
extremity conditions, 341–369. See also specific conditions
Achilles tendonitis, 366, 366f
arterial embolization, 369, 369f
Baker cyst, 363, 364f
bursitis, 353, 353f
cellulitis, 342, 342f
cervical radiculopathy, 360, 360f
crystal-induced synovitis, 348, 348f, 349f
deep vein thrombosis, 363, 363f, 364f
digital clubbing, 361, 361f
Dupuytren contracture, 365, 365f
felon, 343, 343f
ganglion cysts, 367, 367f
gangrene
dry, 344, 344f
gas (myonecrosis), 345, 345f
wet, 344, 344f
ingrown toenail (onychocryptosis), 350, 350f
injuries. See lower extremity trauma; upper extremity trauma
lymphangitis, 351, 351f
lymphedema, 352, 352f
necrotizing fasciitis, 346, 346f, 347f
palmar space infection, 354, 354f
paronychia, 358, 358f
phlegmasia dolens, 362, 362f
Raynaud disease, 368, 368f
subclavian vein thrombosis, 359, 359f
synovial cysts, 367, 367f
tenosynovitis, 355, 355f
thrombophlebitis, 357, 357f
exudates, funduscopic findings with, 64, 64f
eye conditions. See funduscopic findings; ocular conditions; specific conditions
eyelid anatomy, 40f
eyelid lacerations, 96–97, 96f, 97f

F

face. See also ear, nose, and throat conditions; funduscopic findings; lips; ocular conditions
melasma of, 421, 421f
tinea faciale of, 414, 414f
trauma to. See head and facial trauma
facial palsy, 113–114, 113f, 114f
in herpes zoster oticus, 111, 111f
with seventh nerve dysfunction, 113–114, 113f, 114f
facial trauma, penetrating, 25, 25f
failed intrauterine pregnancy, 270, 270f
failure to thrive, 484, 484f

famciclovir
for herpes simplex virus infections, genital, 230–231
for herpes zoster infections, 704
fascicular block
left anterior, electrocardiogram abnormalities in, 826, 826f
left posterior, electrocardiogram abnormalities in, 827, 827f
fasciitis, necrotizing, 346, 346f, 347f
FAST (Focused Assessment with Sonography for Trauma), 863
fat pad sign, 290, 290f
feathering burns, 547, 548f
febrile neutrophilic dermatosis, 427, 427f
feet. See foot conditions; lower extremity trauma
felon, 343, 343f
femoral fractures, 323, 323f
spiral, 516f
Fer-de-Lance envenomation, 554f
ferning burns, 547, 548f
ferning pattern of amniotic fluid, 936, 936f
fetal demise, intrauterine, 285, 285f
fetal heart rate decelerations, 272t
fibular fractures, 324, 324f, 325f
fifth disease, pediatric, 446, 446f
fifth metatarsal base fractures, 334, 334f, 335f
fight bite, 302, 302f
filariasis
lymphatic, 730, 730f, 731f
nonlymphatic, 731, 731f
finger(s). See digit(s); upper extremity trauma
finger ring tourniquets, 642f
fingertip pattern contusion, 682, 682f
Finkelstein test, 356f
fire ant envenomation, 564, 564f, 565f
fire coral envenomation, 574, 574f
fire cupping, 759f
first-degree AV block, electrocardiogram abnormalities in, 819, 819f
“fish handler’s disease,” 582, 582f
fishhook injuries, 656, 656f, 657f
fish tank granuloma, 749
fist, clenched, injury of, 302, 302f
fixed drug eruptions, 377, 377f
flail chest, 167, 167f
Flavivirus, dengue fever due to, 727
flexor tenosynovitis, 355f
fluconazole
for dermatophyte (tinea) infections, 711
for vaginitis, 251
fluoroquinolone, for bacterial conjunctivitis, 32
Focused Assessment with Sonography for Trauma (FAST), 863. See also ultrasound, for trauma
folliculitis
eosinophilic, in HIV patients, 706, 706f
hot tub, 410, 410f
foot conditions
acute sickle dactylitis in, 482
in hand, foot, and mouth disease, 452, 452f
id reaction, tinea pedis caused, 392, 392f
immersion injury of, 543, 543f
tinea pedis of, 414, 415f
traumatic. See lower extremity trauma
forearm fractures, 294, 294f, 295f
foreign bodies
corneal, 85, 85f
impaled, in abdomen or chest, 176, 176f
ingested, button batteries (disk), 477, 477f
intraocular, 88, 88f
rectal, 245, 245f, 246f
in wounds, 643f, 644, 644f–645f
forensic medicine, 671–687
gunshot wounds and, 672–678
entrance wounds, 672–675, 672f–675f
exit wounds, 675–676, 676f
graze wounds, 676, 677f
pattern injuries in. See pattern injuries, in abuse
fourchette, posterior
laceration, 524f
trauma to, 255–256, 255f
Fournier gangrene, 211, 211f, 212f
foxglove ingestion, 631, 631f
fracture(s). See also specific sites
due to child abuse, 514
nasal, 9, 9f, 10f
Salter-Harris classification of, 480, 480f
suggestive of child abuse, 514, 514f, 515f, 516f
of teeth, 139–140, 139f, 140f
toddler’s, 481, 481f
fracture blisters, 326, 326f
free-living ameba infection, 716, 716f
frontal sinus fractures, 21, 21f, 22f
frontal sinusitis, 132
frostbite, 540–541, 540f, 541f
funduscopic findings, 61–80
in anterior ischemic optic neuropathy, 78, 78f
in central retinal artery occlusion, 67, 67f
in central retinal vein occlusion, 69, 69f
with emboli, 66, 66f
with exudates, 64, 64f
in glaucoma, 79, 79f
in macular degeneration, age-related, 63, 63f
normal, 62, 62f
in optic neuritis, 77, 77f
with papilledema, 76, 76f

with retinal detachment, 74, 74f
in retinitis, cytomegalovirus, 75, 75f
in retinopathy. See retinopathy
with Roth spot, 65, 65f
with subhyaloid hemorrhage, in subarachnoid hemorrhage, 80, 80f
with vitreous hemorrhage, 73, 73f
Fusobacterium sp., acute necrotizing ulcerative gingivitis due to, 154

G
gag ligature, in child abuse, 512f
Galeazzi fractures, 294, 295f
gallbladder ultrasound. See ultrasound, of gallbladder
gamekeeper's thumb, 309, 309f
Gamow bag, 537, 537f, 538
ganciclovir, for ocular herpes simplex, 51
ganglion cysts, 367, 367f
gangrene
dry, 344, 344f
gas (myonecrosis), 345, 345f
wet, 344, 344f
Gardnerella vaginalis
vaginal fluid wet mount demonstrating, 930, 930f
vaginosis due to, 250f
gas gangrene, 345, 345f
genital herpes, 230–231, 230f, 231f
genital trauma, 255–256, 255f, 256f
genital warts, 234–235, 234f, 235f
pediatric, due to sexual abuse, 524, 525f, 526f
gentamicin, for conjunctivitis, neonatal, 31t
giant warts, 235f
Gila monster envenomation, 556, 556f
gingival hyperplasia, 161, 161f
gingivitis, ulcerative, necrotizing, 154, 154f
in HIV patients, 714, 714f
gingivostomatitis, herpetic, pediatric, 454, 454f
glaucoma
angle closure, acute, 43, 43f, 44f
funduscopic findings in, 79, 79f
Glidescope video-assisted intubation, 784, 784f
"glitter cells" vaginal fluid wet mount demonstrating, 930, 930f
globe, open, 92, 92f, 93f
glottic opening, visualization during laryngoscopy, 777f
glycerol, for acute angle-closure glaucoma, 43
goiter, 733, 733f
gonococcus, disseminated, 409, 409f
gonorrhea, 227–228, 227f, 228f. See also Neisseria gonorrhoeae
goose flesh, due to scarlet fever, 459, 459f
gout, crystal-induced, 348, 348f
gram-negative rods, Gram stain of, 928f
Gram stain, 928, 928f
granulomas
pyogenic, 380, 380f, 381f
at umbilical cord base, 441, 441f
granulomatous amebic encephalitis, 716
gravid abdomen, 189, 190f
Grey Turner sign, 175, 175f
griseofulvin, for dermatophyte (tinea) infections, 711
Guinea worm, dracunculiasis due to, 729, 729f
"gumboil," 147, 147f
gunshot wounds, 672–678
entrance wounds, 672–675, 672f–675f
exit wounds, 675–676, 676f
graze wounds, 676, 677f
guttate psoriasis, 384
gynecologic conditions, 250–265. See also specific conditions
abortion, spontaneous, 254, 254f
Bartholin gland abscess, 253, 253f
cervical polyps, 252, 252f
cervicitis
chlamydial, 229, 229f
in gonorrhea, 227, 228f
cystocele, 263, 263f
genital trauma and sexual assault, 255–256, 255f, 256f
imperforate hymen, 265, 265f
lichen planus, 259, 259f
lichen sclerosus, 257, 257f, 258f
pregnancy. See pregnancy
rectocele, 264, 264f
urethral prolapse, 261, 261f
uterine prolapse, 262, 262f
vaginitis, 250–251, 250f, 251f
vulvar tumor, 260, 260f

H
Haemophilus bacteria infection, conjunctivitis due to, 30–31
Haemophilus ducreyi, chancroid due to, 233, 233f
Haemophilus influenzae
cellulitis due to, orbital and periorbital, 466
conjunctivitis due to, 32
epiglottitis due to, 125, 473
membranous tracheitis due to, 478
otitis media due to, 100
septal abscesses due to, 119
sinusitis due to, 132
thyroglossal duct cyst, pediatric, due to, 470, 470f
uvulitis due to, 127
hair loss, arsenic poisoning and, 623f
hair tourniquet, pediatric, 483, 483f
hairy leukoplakia, oral, in HIV patients, 692, 692f

Halo sign, 6f
hand(s). See also digit(s); upper extremity trauma
acute sickle dactylitis in, 482, 482f
blistering distal dactylitis of, 461, 461f
digital clubbing and, 361, 361f
Dupuytren contracture in, 365, 365f
dyshidrotic eczema of, 391, 391f
in hand, foot, and mouth disease, 452, 452f
immersion injury of, 543, 543f
paronychia in, 358, 358f
porphyria cutanea tarda in, 428, 428f
tinea manuum of, 414, 415f
hand, foot, and mouth disease, pediatric, 452, 452f
hand-foot syndrome, acute, pediatric, 482, 482f
Hansen disease, 739–740, 739f, 740f
Hapalochlaena envenomation, 581, 581f
head and facial trauma, 3–27
exophthalmos due to, 23, 23f, 24f
external ear injuries, 19–20, 19f, 20f
frontal sinus fracture, 21, 21f, 22f
herniation syndromes, 26, 26f, 27f
LeFort facial fractures, 12, 12f
mandibular fractures, 16, 16f, 18f
nasal, 9, 9f, 10f
orbital blowout fractures, 13, 13f–15f
penetrating, 25, 25f
skull fracture
basilar, 4, 4f–6f, 5
depressed, 7–8, 7f
ping pong ball, 8, 8f
zygomatic arch fractures, 11, 11f
head, lice infestation, 493, 493f
heart. See also cardiac entries
cocaine toxicity and, 591, 592f, 593
infective endocarditis and, cutaneous manifestations of, 405, 405f, 406f
helium-oxygen mixture (HELIOX), 766
Heloderma suspectum envenomation, 556, 556f
hemangiomas, strawberry, pediatric, 464, 464f, 465f
hematomas
of abdominal wall, 192, 192f, 193f
forehead with eye bruising, 512f
of pinna, 19–20, 19f
retrobulbar, 23, 23f
septal, 9, 9f, 119, 119f
subungual, 316, 316f
a -Hemolytic streptococci, dacryocystitis due to, 36
b-Hemolytic streptococci, blistering distal dactylitis due to, 461
hemopericardium, 864, 865f
hemorrhage
in age-related macular degeneration, funduscopic appearance of, 63, 63f
retinal
altitude-induced, 536, 536f
funduscopic appearance of, 65, 65f
in shaken baby syndrome, 508, 509f, 510f
splinter, in infective endocarditis, 405, 406f
subarachnoid, subhyaloid hemorrhage in, funduscopic findings with, 80, 80f
subconjunctival, 84, 84f
subdural, in shaken baby syndrome, 508, 508f, 509f
subgaleal, in shaken baby syndrome, 510f
subhyaloid, in subarachnoid hemorrhage, funduscopic findings with, 80, 80f
sublingual, 17f
vitreous, funduscopic findings with, 73, 73f
hemorrhoids, 240–241, 240f, 241f, 242f
hemostasis, for wound exploration, 642, 642f, 643f
hemotympanum, 4, 5f
Henoch-Schönlein purpura, 396, 397f, 462, 462f, 505f
herald patches, in pityriasis rosea, 386, 386f, 387
hernias
incisional, 184, 185f
inguinal, 184, 184f, 185f
pediatric, 490–491, 490f
traumatic, abdominal, 177, 177f
umbilical, 186, 186f
herniation syndromes, brain, 26, 26f, 27f
heroin toxicity, 597–598, 597f, 598f
herpes gladiatorum, 412, 412f
herpes simplex infection
conjunctivitis due to, 30, 31t
erythema multiforme due to, 374
genital, 230–231, 230f, 231f
herpetic gingivostomatitis due to, 454, 454f
in HIV patients, 707, 707f
keratitis due to, 50, 50f, 51f
nongenital, 412, 412f
oral, 157, 157f
Tzanck preparation for detection, 940, 940f
herpes zoster infection
cutaneous manifestations of, 413, 413f
herpes zoster ophthalmicus due to, 49, 49f
herpes zoster oticus due to, 111, 111f, 112f
in HIV patients, 704, 704f, 705f
Hutchinson sign and, 49, 49f
pediatric, 451, 451f
Tzanck preparation for detection, 940, 940f
herpetic gingivostomatitis, pediatric, 454, 454f
herpetic whitlow, 412, 412f
hidradenitis suppurativa, 379, 379f
high-altitude injuries
cerebral edema, 538, 538f

pulmonary edema, 537, 537f
retinal hemorrhage, 536, 536f
high-pressure injection injuries, 308, 308f
hip
dislocations of, 317, 317f
fractures of, 318, 318f
Histoplasma capsulatum, erythema multiforme, 374
HIV infection, 689–714
acute necrotizing ulcerative gingivitis associated with, 714, 714f
anemia in, 717f
candidiasis associated with, 693, 693f, 694f
cryptococcal meningitis associated with, 700, 700f, 701f
cytomegalovirus infections associated with, 703, 703f
dermatophyte (tinea) infections associated with, 711, 711f
disseminated histoplasmosis with, 702, 702f
drug reactions in, 713, 713f
eosinophilic folliculitis associated with, 706, 706f
herpes simplex virus infection associated with, 707, 707f
herpes zoster infection associated with, 704, 704f, 705f
immune reconstitution inflammatory syndrome and, 691, 691f
Kaposi sarcoma associated with, 695, 695f, 696f
molluscum contagiosum associated with, 710, 710f
oral hairy leukoplakia associated with, 692, 692f
pneumocystis and, 699, 699f
Pneumocystis jirovecii infection associated with, 699, 699f
primary, 690, 690f
pulmonary histoplasmosis, 702, 702f
scabies associated with, 708–709, 708f, 709f
thrombocytopenia associated with, 712, 712f
Toxoplasma gondii infection associated with, 697, 697f, 698f
Hollenhorst plaques, 66
hollow viscus injuries, 518f
honeybee envenomation, 564, 565f
hordeolum, 39, 39f, 40f
horizontal mattress suture, 661, 662f
Horner syndrome, 48, 57, 57f
horse serum antivenin, for pit viper envenomations, 551
hot tub folliculitis, 410, 410f
huffing, 608, 608f, 609f
human bites, 654
human herpesvirus 6, roseola infantum, pediatric due to, 447
human immunodeficiency virus. See HIV infection
Hutchinson fractures, 296, 297f
Hutchinson sign, 413
herpes zoster infection and, 49, 49f
hydatid cyst, 734, 734f
hydatidiform mole, 269, 269f
hydrocele, 208, 208f
hydrofluoric acid burns, 620, 621f
hydromorphone, for acute sickle dactylitis in, 482
hydrozoan envenomation, 574, 574f, 575f
hymen
bruises, 524f
imperforate, 265, 265f
prepubertal
annular, 521f
crescentic, 521f
normal, 520f, 521f
septate, 520f, 523f
hymenoptera envenomation, 564, 564f, 565f
hyperacute T waves, 813, 813f
hyperbaric therapy
for high-altitude cerebral edema, 538
for high-altitude pulmonary edema, 537, 537f
hyperkalemia, electrocardiogram abnormalities in, 851–852, 851f, 852f
hyperpigmentation, arsenic poisoning and, 622, 622f
hyperplasia, gingival, 161, 161f
hypertensive retinopathy, funduscopic findings in, 70, 70f
hypertrophic cardiomyopathy, electrocardiogram abnormalities in, 846, 846f
hypertrophic pyloric stenosis, 442, 442f, 443f
hyphema, 86, 86f, 87f
hypokalemia, electrocardiogram abnormalities in, 850, 850f
hypopyon, 45, 46f
corneal ulcers and, 52, 53f–54f
hypothermia, 539, 539f
electrocardiogram abnormalities in, 849, 849f

I

idiopathic thrombocytopenic purpura, 398, 398f
idoxuridine, for ocular herpes simplex, 51
id reaction
with tinea capitis, 392
with tinea pedis, 392, 392f
ileocolic intussusception, pediatric, 488–489, 488f, 489f
imiquimod, for condyloma acuminata, 234
immersion burns, due to child abuse, 502, 506f
immersion injuries, 543, 543f
immersion lines, in thermal pattern injuries, 686, 687f
imminent vertex delivery, 273, 273f
immune reconstitution inflammatory syndrome, 691, 691f
impaled foreign bodies, in abdomen or chest, 176, 176f
imperforate hymen, 265, 265f
impetigo
pediatric, 448, 448f
resemblance to cigarette burns, 503, 507f
incised wounds, 679
incomplete abortion, 254
India ink preparation, for cerebrospinal fluid examination, 934, 934f

inevitable abortion, 254
infantile botulism, 615, 615f
infections. See also specific infections
with clenched fist injury, 302, 302f
due to animal bite wounds, 654–655
odontogenic. See odontogenic infections
of wounds, postoperative, 664f
infectious keratitis, 52, 52f–54f
infective endocarditis, cutaneous manifestations of, 405, 405f, 406f
inferior rectus muscle entrapment, 14f
infestations
pediatric, lice, 493, 493f
skin scraping for, 933, 933f
inflammatory flexor tenosynovitis, 355
ingrown toenail, 350, 350f
inhalant abuse, 608, 608f, 609f
insect bites and stings. See envenomations
interarytenoid notch, visualization during laryngoscopy, 776f
interrupted wound closure, simple, 658, 659f, 660f
intersection syndrome, 355, 356f
intertrigo, 418, 418f
intestinal malrotation with volvulus, 444, 444f, 445f
intraocular foreign bodies, 88, 88f
intrauterine fetal demise, 285, 285f
intrauterine pregnancy, failed, 270, 270f
intrusive luxation of teeth, 137, 137f
intubating laryngeal airway, 793–794, 793f, 794f
intubation
blind nasotracheal intubation, 796–798, 796f–798f
bougie-assisted, 781–783, 781f–783f
digital (tactile), 792, 792f
fiberoptic, flexible, nasal, 799–800, 799f
Glidescope video-assisted, 784, 784f
indications for, 770–771
intubating laryngeal airway for, 793–794, 793f, 794f
king laryngeal tube, 795, 795f
laryngoscopic, 772–780, 772f–780f
McGrath MAC video-assisted, 786, 786f
position for, 768, 768f, 772
Storz C-MAC video-assisted, 785, 785f
ipsilateral dilated pupil, 26, 26f
iridodialysis, traumatic, 89, 89f
iridodonesis, 90
iris bombé, 44f
iritis, 45, 45f, 46f
iron poisoning, 624, 624f
irrigation of wounds, 640–641, 640f, 641f
isopropanol toxicity, 602
isosorbide, for acute angle-closure glaucoma, 43
itraconazole
for dermatophyte (tinea) infections, 711
for tropical sporotrichosis, 760
ivermectin
for cutaneous larva migrans, 724
for elephantiasis, 730
for scabies, 708
Ixodes dammini, 562, 562f

J

Janeway lesions, in infective endocarditis, 405, 405f
Jaundice, 420, 420f
in malaria, 742f
in newborns, 438, 438f
jellyfish envenomation, 574, 575, 575f, 576f
jequirity pea ingestion, 636, 636f
Jersey finger, 312–313, 313f
jimsonweed ingestion, 534f, 634
“jock itch,”, 414, 415f
Jones fractures, 334, 334f, 335f
jugulovenous distention, 183, 183f
with apical lung masses, 181, 181f
junctional rhythm, 830, 830f

K

Kanavel, signs of, 355
Kaposi sarcoma, in HIV patients, 695, 695f, 696f
Kawasaki disease, pediatric, 497–499, 497f, 498f, 499f
keloids, 664f
keratitis
in epidemic keratoconjunctivitis, 33, 34f
herpes simplex, 50, 50f, 51f
infectious (bacterial; ulcerative), 52, 52f–54f
ultraviolet (photokeratitis; snow blindness), 544, 546f
keratoconjunctivitis
epidemic, 33, 34f
herpes simplex, 50
kerion, 414, 415f
ketorolac, for acute sickle dactylitis in, 482
kidneys, ultrasound of. See ultrasound, of renal
king laryngeal tube, 795, 795f
Klebsiella sp., cellulitis due to, 342
knee dislocation, 322, 322f
knee extensor injury, 319
Koplik spots, 449, 449f
kwashiorkor, 750–751, 750f, 751f

L

labial adhesions, pediatric, 531, 531f
labial frenulum tear, in child abuse, 512f
lacerations
abusive liver, 518f

of clenched fist, 302, 302f
of ear, 650, 650f
of eyelid, 96–97, 96f, 97f
linear, 646, 646f
of lip, 145, 145f, 651f
nail bed, 316f
over frontal sinuses, 21, 21f
perineal, postpartum, 285, 285f, 285f
posterior fourchette, 524f
splenic, 518f
of tendons, 652, 652f, 653f
of tongue, 144, 144f
vermilion border, 651f
b-Lactam antibiotics, for cellulitis, 342
larva migrans, cutaneous, 724, 724f
laryngeal airway, intubating, 793–794, 793f, 794f
laryngeal fracture, 767, 767f
laryngeal mask airway, 787–788, 787f, 788f
laryngoscopy
alternatives to rapid-sequence induction for, 769, 769f
bimanual, 775, 777f
biomechanics and, 773–774, 774f
grip for, 773–774, 774f
latanoprost, for acute angle-closure glaucoma, 43
Latrodectus mactans envenomation, 557, 557f
laxative burns, 506f
lead lines, 625, 626f
lead poisoning, 625, 625f, 626f
leech bites, 735, 735f, 736f
LeFort fractures, 12, 12f
left anterior fascicular block, electrocardiogram abnormalities in, 826, 826f
left atrial hypertrophy, electrocardiogram abnormalities in, 844, 844f
left bundle branch block, electrocardiogram abnormalities in, 825, 825f
left main lesion, electrocardiogram abnormalities in, 810, 810f
left posterior fascicular block, electrocardiogram abnormalities in, 827, 827f
left ventricular aneurysm, electrocardiogram abnormalities in, 817, 817f
left ventricular hypertrophy, electrocardiogram abnormalities in, 842, 842f
leg. See also lower extremity trauma
phlegmasia dolens and, 362, 362f
leishmaniasis, 737–738, 737f, 738f
Leiurus envenomation, 560
lens dislocation, 90, 90f, 91f
leprosy, 739–740, 739f, 740f
leptospirosis, 741, 741f
leukocoria, 95
leukocytes, stool examination for, 932, 932f
leukocytoclastic vasculitis, 396, 396f, 397f
leukoplakia, hairy, oral, in HIV patients, 692, 692f
levamisole-induced vasculitis, 637–638, 637f, 638f
levofloxacin, for chlamydia infection, 229
lice infestation, pediatric, 493, 493f
lice, pediculosis due to, 236, 236f
lichen planus, 259, 259f
lichen sclerosis, pediatric, 530, 530f
lichen sclerosus, 257, 257f, 258f
ligature bruise, 504f, 505f
lighted styles and wands, 789–790, 789f, 790f, 791t
lightning injuries, 547–548, 547f, 548f
lily of the valley ingestion, 631, 632f
limb lead reversal, electrocardiogram abnormalities and, 857, 857f
linear array ultrasound transducer, 861t
lion fish envenomation, 578, 578f
lion's mane penis, 457, 457f
lip licker dermatitis, 432, 432f
lips
injury due to child abuse, 513f
lacerations of, 145, 145f, 651f
piercing complications of, 163, 163f
retraction of, for intubation, 775–778, 778f
Lisfranc fracture-dislocations, 336–337, 336f, 337f
livedo reticularis, 394, 394f, 395f
liver, acetaminophen poisoning and, 599, 599f
lizard bites, Gila monster, 556, 556f
Lone Star tick, 562, 563f
lower extremity trauma, 319–339, 338f
Achilles tendon rupture, 327, 327f
amputations, 338f
ankle dislocation, 328, 328f, 329f
ankle fracture, 330, 330f, 331f
ankle sprain, 332, 332f
calcaneal fracture, 333, 333f
femoral fracture, 323, 323f
fifth metatarsal base fractures, 334, 334f, 335f
fracture blisters, 326, 326f
knee dislocation, 322, 322f
knee extensor injury, 319
Lisfranc fracture-dislocation, 336–337, 336f, 337f
patellar dislocation, 321, 321f
patellar fractures, 319, 320f
patellar tendon rupture, 319, 319f, 320f
quadriceps tendon rupture, 319, 319f
tibial-fibular fractures, 324, 324f, 325f
low voltage, electrocardiogram abnormalities and, 858, 858f
Loxosceles reclusa envenomation, 558–559, 558f, 559f
Ludwig angina, 152, 152f
lunate dislocations, 298, 298f, 299f

lung masses, apical, 181, 181f
lung, ultrasound of, 887–889, 887f–889f
luxatio erecta, 289, 289f
luxation, intrusive, of teeth, 137, 137f
Lyme disease, 562
  erythema chronicum migrans in, 407, 407f
lymphadenopathy
  in cat-scratch disease, 472, 472f
  in Kawasaki disease, 497, 498f
lymphangitis, 351, 351f
lymphatic filariasis, 730, 730f, 731f
lymphedema, 352, 352f
lymphogranuloma venereum, 232, 232f

M

macrolides, for mastoiditis, 108
macula, normal, funduscopic appearance of, 62, 62f
macular degeneration, age-related, funduscopic findings in, 63, 63f
Madura foot, 745
maggots, 747, 747f, 748f
Maisonneuve fractures, 324, 325f
malaria, 742–743, 742f, 743f
  Wright stain for, 935, 935f
mallet finger, 312–313, 312f, 313f
malnutrition, protein-energy, 750–751, 750f, 751f
mandible, tori on, 160, 160f
mandibular fractures, 16, 16f, 18f
mannitol, for acute angle-closure glaucoma, 43
marasmus, 750–751, 750f, 751f
Marcus Gunn pupil, 55, 55f, 56f
marine injuries/conditions, 568–582
  cephalopod envenomation, 581, 581f
  coelenterate envenomation, 574–575, 574f, 576f
  cone snail envenomation, 579, 579f
  marine dermatitis ("sea bather's eruption"), 577, 577f
  pressure-induced
    mask squeeze, 569, 569f
    middle ear squeeze (barotitis media), 568, 568f
  scorpion fish envenomation, 578, 578f
  sea snake envenomation, 580, 580f
  sea urchin envenomation, 572–573, 572f, 573f
  stingray envenomation, 570–571, 570f, 571f
mask of pregnancy, 421, 421f
mask squeeze, 569, 569f
mastitis, in newborns, 440, 440f
mastoiditis, 108, 108f
mattress suture
  horizontal, 661, 662f
  vertical, 661, 661f
maxillary sinusitis, 132, 132f
McGrath MAC video-assisted intubation, 786, 786f
McRobert maneuver, 285
MDMA (ecstasy; 3,4-methylenedioxymethamphetamine), 588, 588f
measles-mumps-rubella (MMR) vaccine, for measles, 449
measles, pediatric, 449, 449f
mebendazole, for cutaneous larva migrans, 724
median nerve injuries, 304, 304f
Mees lines, arsenic poisoning and, 623f
Megalopyge opercularis envenomation, 566, 567f
melanoma, 431, 431f
melasma, 421, 421f
melena, 247, 247f
membranous tracheitis, pediatric, 478, 478f
meningitis
  cryptococcal, in HIV patients, 700, 700f, 701f
  epidemic, 732, 732f
meningococcemia, pediatric, 455, 455f
mercury poisoning, 627–628, 627f, 628f
mercury salts, 628f
metacarpal fractures
  of base, 305, 305f, 306f
  of neck, 303, 303f
metatarsal fractures, of fifth metatarsal base, 334, 334f, 335f
methanol toxicity, 602
methazolamide, for acute angle-closure glaucoma, 43
methemoglobinemia, 610, 610f, 611f
methylene blue, for methemoglobinemia, 610, 611f
3,4-methylenedioxymethamphetamine (ecstasy; MDMA), 588, 588f
metronidazole
  for tetanus, 755
  for vaginitis, 251
microscopic examinations
  of amniotic fluid, fern pattern in, 936, 936f
  for blast cells, 937, 937f
  of cerebrospinal fluid, 934, 934f
  dark-field, 929, 929f
  for Enterobias vermicularis eggs, 938, 938f, 939f
  for fecal leukocytes, 932, 932f
  Gram stain, 928, 928f
  potassium hydroxide mount, 931, 931f
  for schistocytes, 937, 937f
  skin scraping for dermatoses and infestations, 933, 933f
  of synovial fluid, 926, 926f, 927f
  Tzanck preparation for herpes detection, 940, 940f
  urinalysis, 924, 924f, 925f
  vaginal fluid wet mount, 930, 930f
  Wright stain for malaria, 935, 935f
middle ear squeeze, 568, 568f
midpalmar space infections, 354
milk production, neonatal, 439, 439f
miosis, cholinergic, 595f

Mobitz I second-degree AV block, electrocardiogram abnormalities in, 820, 820f
Mobitz II second-degree AV block, electrocardiogram abnormalities in, 821, 821f
modified Ritgen maneuver, 274, 274f
molar pregnancy, 269, 269f
molluscum contagiosum, in HIV patients, 710, 710f
Mongolian spots, 505f
Monteggia fractures, 294, 295f
Moraxella catarrhalis
  membranous tracheitis due to, 478
  orbital and periorbital cellulitis due to, 466
  otitis media due to, 100
  sinusitis due to, 132
Morison pouch ultrasound view, 866, 866f, 867f
morphine, for acute sickle dactylitis in, 482
mouth. See odontogenic infections; oral conditions; oral trauma; teeth
mucocele, of floor of mouth, 128, 128f
mucocutaneous lymph node syndrome, pediatric, 497–499, 497f, 498f, 499f
mucosal necrosis, due to opioid toxicity, 598f
mucous patches, in secondary syphilis, 224–226, 226f
mumps, 744, 744f
mushroom ingestion, 629, 629f, 630f
muzzle contusions, 674f, 675
mycetoma, 745, 745f, 746f
mycobacteria infections
  erythema multiforme due to, 374
  leprosy, 739–740, 739f, 740f
  nontuberculous, 749, 749f
  tuberculosis, 761, 761f–763f
Mycoplasma pneumoniae
  erythema multiforme due to, 374
  pharyngitis due to, 117
mydriasis, anticholinergic, 594f
myiasis, 747, 747f, 748f
myonecrosis, 345, 345f
  clostridial, 345, 345f
  in compartment syndrome, 314
myringitis, bullous, 103, 103f

N

Naegleria fowleri, free-living ameba infection due to, 716
nails
  ingrown toenail, 350, 350f
  Mees lines on, arsenic poisoning and, 623f
naloxone, for opioid toxicity, 597–598
Naphcon A
  for allergic conjunctivitis, 35
  for pinguecula, 38
  for pterygium, 38
nasal airways, 771f
nasal conditions. See ear, nose, and throat conditions; specific conditions
nasal foreign-body insertion, pediatric, 479, 479f
nasopharyngeal intubation. See intubation
necrotizing fasciitis, 346, 346f, 347f
necrotizing ulcerative gingivitis, acute, 154, 154f
  in HIV patients, 714, 714f
Neisseria gonorrhoeae
  in Bartholin gland abscess, 253
  conjunctivitis due to, 30–31, 30f–31f, 31t, 32
  disseminated gonococcus due to, 409, 409f
  gonorrhea due to, 227–228
  Gram stain of, 928, 928f
  pharyngitis due to, 117
  sexually transmitted diseases, pediatric, 524, 525f
Neisseria meningitidis, meningococcemia due to, 455
neonatal conditions. See newborn conditions; specific conditions
neonatal conjunctivitis, 30–31, 30f–31f, 31t
neonatal milk production, 439, 439f
neovascularization, in diabetic retinopathy, 71, 72f
nevus simplex, 437, 437f
newborn conditions, 436–445. See also specific conditions
  conjunctivitis, 30–31, 30f–31f, 31t
  erythema toxicum, 436, 436f
  hypertrophic pyloric stenosis, 442, 442f, 443f
  intestinal malrotation with volvulus, 444, 444f, 445f
  jaundice, 438, 438f
  mastitis, 440, 440f
  neonatal milk production (witch's milk), 439, 439f
  salmon patches (nevus simplex), 437, 437f
  umbilical cord granuloma, 441, 441f
nightstick fractures, 294, 294f
nits, 493, 493f, 526f
nodular melanoma, 431f
nodular umbilicus, 188, 188f
nontuberculous mycobacteria infections, 749, 749f
normal cupping of optic nerve, in glaucoma, 79f
Norwegian scabies, in HIV patients, 708–709, 708f, 709f
nursemaid's elbow, pediatric, 486, 486f, 487f
nursing bottle caries, 485, 485f

O

obstetric conditions, 266–285. See also delivery, emergency
  ectopic pregnancy, 266–268, 266f, 267f, 268f
  failed intrauterine pregnancy, 270, 270f
  intrauterine fetal demise, 285, 285f
  molar pregnancy, 269, 269f
  postpartum perineal lacerations, 285, 285f, 285f
  third-trimester blunt abdominal trauma, 271, 271f, 272f

octopus, blue-ringed, envenomation, 581, 581f
ocular conditions, 29–60. See also specific conditions
  afferent pupillary defect (Marcus Gunn pupil), 55, 55f, 56f
  anisocoria, 48, 48f
  blepharitis, 39, 40f
  chalazion, 39, 40f
  conjunctivitis. See bacterial conjunctivitis; conjunctivitis
  corneal ulcers, 52, 52f–54f
  dacryoadenitis, 37, 37f
  dacryocystitis, 36, 36f
  epidemic keratoconjunctivitis, 33, 34f
  episcleritis, 42, 42f
  funduscopic findings in. See funduscopic findings
  glaucoma, acute angle closure, 43, 43f, 44f
  herpes simplex virus infections
    in HIV patients, 707
    keratitis due to, 50, 50f, 51f
  herpes zoster infections
    herpes zoster ophthalmicus, 49, 49f
    in HIV patients, 704, 704f, 705f
  hordeolum, 39, 39f, 40f
  Horner syndrome, 48, 57, 57f
  hypopyon, 52, 53f–54f
  pinguecula, 38, 38f
  pterygium, 38, 38f
  retinitis
    cytomegalovirus, in HIV patients, 703, 703f
    Toxoplasma gondii, in HIV patients, 697, 698f
  scleritis, 41, 41f
  sixth-nerve palsy, 60, 60f
  sympathetic ophthalmia, 47, 47f
  third-nerve palsy, 58, 59f
  trachoma, 757, 757f
  traumatic, 81–98
    cataract due to, 95, 95f
    chemical exposure, 98, 98f
    corneal abrasion, 82–83, 82f, 83f
    corneal foreign bodies, 85, 85f
    eyelid lacerations, 96–97, 96f, 97f
    hyphema, 86, 86f, 87f
    intraocular foreign bodies, 88, 88f
    iridodialysis, 89, 89f
    lens dislocation, 90, 90f, 91f
    open globe, 92, 92f, 93f
    penetrated globe, 88, 92, 92f, 93f
    scleral rupture, 94, 94f
    subconjunctival hemorrhage, 84, 84f
  uveitis, anterior, 45, 45f, 46f
  viral conjunctivitis, 33, 33f
ocular herpes simplex, 40f
ocular ultrasound, 916–918, 916f–919f
oculogyric crisis, pediatric, 495, 495f
odontogenic infections, 146–153
  buccal space abscess, 150, 150f
  canine space abscess, 151, 151f
  gingival abscess, 146, 146f
  Ludwig angina, 152, 152f
  parapharyngeal space abscess, 153, 153f
  parulis (“gumboil”), 147, 147f
  periapical abscess, 147, 147f, 148f
  pericoronal abscess, 149, 149f
  periodontal abscess, 146, 146f
ofloxacin, for chlamydia infection, 229
Ogilvie syndrome, abdominal distention due to, 191f
oil of wintergreen toxicity, 600f
oleander ingestion, 631, 631f
olecranon bursitis, 353, 353f
olopatadine, for allergic conjunctivitis, 35
omphalomesenteric duct, 441, 441f
onychocryptosis, 350, 350f
onychomycosis, 417, 417f
open cricothyrotomy, 803, 803f
open globe injuries, 92, 92f, 93f
ophthalmia neonatorum, 30–31, 30f–31f, 31t
opioid toxicity, 597–598, 597f, 598f
  mucosal necrosis, 598f
optic disk
  normal, funduscopic appearance of, 62, 62f
  pallor of, in optic neuritis, 77, 77f
  swelling of, in anterior ischemic optic neuropathy, 78, 78f
optic nerve, cupping of, in glaucoma, 79, 79f
optic neuritis, funduscopic findings in, 77, 77f
optic neuropathy, ischemic, anterior, funduscopic findings in, 78, 78f
oral airways, 771f
oral conditions, 154–163. See also specific conditions
  acid tooth erosion, 155, 155f
  aphthous ulcers (canker sores), 158, 158f
  black hairy tongue, 162, 162f
  candidiasis, in HIV patients, 639f, 640f, 693f
  gingival hyperplasia, 161, 161f
  hairy leukoplakia, in HIV patients, 692, 692f
  herpes simplex virus infection (cold sores), 157, 157f
  Kaposi sarcoma, in HIV patients, 696f
  piercing complications, 163, 163f
  strawberry tongue, 159, 159f, 459
  thrush (candidiasis), 156, 156f
  torus palatinus, 160, 160f
  trench mouth (acute necrotizing ulcerative gingivitis), 154, 154f
oral trauma, 135–144
  alveolar ridge fracture due to, 141, 141f
  lip laceration due to, 145, 145f

temporomandibular joint dislocation due to, 142–143, 142f, 143f
tongue laceration due to, 144, 144f
tooth avulsion due to, 138, 138f
tooth fractures due to, 139–140, 139f, 140f
tooth impaction due to, 137, 137f
tooth subluxation due to, 136, 136f
orbital blowout fractures, 13, 13f–15f
orbital cellulitis, pediatric, 466, 466f, 467f
orchitis, 206–207, 206f, 207f
oropharyngeal burns, caustic ingestion and, 618, 618f
oropharyngeal injuries, in child abuse, 511, 512f
oropharyngeal intubation. See intubation
Osler nodes, in infective endocarditis, 405, 406f
osmoglyn, for acute angle-closure glaucoma, 79
otitis externa, 106, 106f
otitis media, 100, 100f, 102f
in bullous myringitis, 103
serous, with effusion, 100, 102f
otohematoma, 19
ouabain ingestion, 631

P

paced rhythm, 832, 832f
Paget-von Schröetter syndrome, 359, 359f
palate, tori on, 160, 160f
palmar space infections, 354, 354f
pancreatitis, hemorrhagic, 174, 174f
panniculitis, cold (popsicle), pediatric, 453, 453f
panniculus, intertrigo of, 418, 418f
pansinusitis, 132, 133f
papilledema, funduscopic findings with, 76, 76f
paracentesis ultrasound, 914–915, 914f
paralysis, tick-induced, 562
paraneoplastic pemphigus, 425, 425f
parapharyngeal space abscess, 153, 153f
paraphimosis, 213, 213f
paronychia, 358, 358f
parulis, 147, 147f
parvovirus B19, erythema infectiosum due to, 446, 446f
passive oxygenation, 772, 773f
Pasteurella multocida, in bite wounds, 654, 655
Pastia lines, 459, 460f
patella
dislocation of, 321, 321f
fractures of, 319, 320f
patellar tendon rupture, 319, 319f, 320f
patent urachal duct, 187, 187f
pattern burns, due to child abuse, 506f
pattern contusions, 682–683, 682f
pattern injuries, in abuse, 679–687
blunt-force, 682–683, 682f
sharp-force, 679, 679f, 680f
strangulation, 684–685, 684f, 685f
thermal, 686, 686f, 687f
peace lily ingestion, 633, 633f
pectus carinatum, 194, 195f
pectus deformities, 194, 194f, 195f
pectus excavatum, 194, 194f
pediatric conditions
abuse-related. See child abuse
acute sickle dactylitis (hand-foot syndrome), 482, 482f
cutaneous, 446–472
blistering distal dactylitis, 461, 461f
branchial cleft cyst, 469, 469f
cat-scratch disease, 472, 472f
cold panniculitis (popsicle panniculitis), 453, 453f
cystic hygroma, 471, 471f
erythema infectiosum (fifth disease), 446, 446f
hand, foot, and mouth disease, 452, 452f
Henoch-Schönlein purpura, 462, 462f
herpes zoster infection (shingles), 451, 451f
herpetic gingivostomatitis, 454, 454f
impetigo, 448, 448f
measles, 449, 449f
meningococcemia, 455, 455f
orbital and periorbital cellulitis, 466, 466f, 467f, 468f
roseola infantum (exanthem subitum), 447, 447f
scarlet fever, 459, 459f, 460f
serum sickness-like reaction, 463, 463f
staphylococcal scalded skin syndrome, 458, 458f
strawberry hemangioma, 464, 464f, 465f
summer penile syndrome, 457, 457f
thyroglossal duct cyst, 470, 470f
urticaria, 456, 456f
varicella (chickenpox), 450, 450f
epiglottitis (supraglottitis), 473, 473f, 474f
failure to thrive, 484, 484f
hair tourniquet, 483, 483f
herpes zoster (shingles), 451, 451f
ileocolic intussusception, 488–489, 488f, 489f
ingested foreign bodies, button batteries, 477, 477f
inguinal hernia, 490–491, 490f
Kawasaki disease, 497–499, 497f, 498f, 499f
lice infestation, 493, 493f
membranous (bacterial) tracheitis, 478, 478f
nasal foreign-body insertion, 479, 479f
neonatal. See newborn conditions
nursemaid's elbow (radial head subluxation), 486, 486f, 487f
nursing bottle caries, 485, 485f
oculogyric crisis, 495, 495f
pinworm infection (enterobiasis), 492, 492f
rashes and lesions, 446–472

pediatric conditions (Cont.):
retropharyngeal abscess, 475–476, 475f, 476f
Salter-Harris classification of fractures, 480, 480f
setting-sun phenomenon (sundowning), 496, 496f
toddler's fractures, 481, 481f
trichotillomania, 494, 494f
pediculosis, 236, 236f, 933, 933f
Pediculus humanus capitis (head louse), 493
Pediculus humanus corporis (body louse), 493
Pelamis platurus envenomation, 580, 580f
pelvic
fractures, 178, 178f
ultrasound, 907–910
pemphigus, paraneoplastic, 425, 425f
pemphigus vulgaris, 425, 425f
penetrated globe injury, 88, 92, 92f, 93f
penetrating facial trauma, 25, 25f
penicillin
for anthrax, cutaneous, 718
for diphtheria, 122
for epidemic meningitis, 732
for leptospirosis, 741
for mastitis, 440
for retropharyngeal abscess, 475
for tetanus, 755
penicillinase-resistant penicillins, for mastoiditis, 108
penicillin G
for scarlet fever, 459
for syphilis, 224
penile conditions
abrasion, 525f
balanitis, 219, 219f, 220f
balanoposthitis, 219, 219f, 220f
hair tourniquet, 483
paraphimosis, 213, 213f
phimosis, 213, 213f
posthitis, 219
priapism, 214, 214f
tinea cruris, 414, 415f
tourniquet, 217, 217f
traumatic
fracture, 216, 216f
toilet seat injury, 528, 528f
zipper injury, 221, 221f
urethral rupture, 215, 215f
varicocele, 222, 222f
zipper injury, 221, 221f
penlight test, for acute angle-closure glaucoma, 44f
Peptostreptococcus sp., necrotizing fasciitis due to, 346
perianal abscesses, 238, 238f, 239f
perianal strep desquamation, in scarlet fever, pediatric, 459, 460f
perianal trauma, 255–256, 255f, 256f
periapical abscesses, 147, 147f, 148f
pericardial effusion
electrocardiogram abnormalities in, 854, 854f
subxiphoid-cardiac (subcostal) view for, 878f
pericardiocentesis, cardiac tamponade with, 172, 172f
pericarditis, electrocardiogram abnormalities in, 818, 818f
pericoronal abscesses, 149, 149f
perilunate dislocations, 298, 300f
perineum
Fournier gangrene of, 211, 211f
lacerations of, postpartum, 285, 285f, 285f
straddle injury of, 218, 218f
pediatric, 527, 527f
periodontal abscesses, 146, 146f
periorbital cellulitis, pediatric, 466, 467f, 468f
periorbital contusion pattern, 682f
peripheral nerve injuries, of upper extremities, 304, 304f
perirectal infections, herpes simplex virus, in HIV patients, 707, 707f
peritonsillar abscesses, 123, 123f, 124f
periungual desquamation, in Kawasaki disease, 497, 498f
pernio, 542, 542f
perniosis, 542, 542f
Perthes syndrome, 166
petechiae, dengue, 727f, 728
petechial hemorrhages, in strangulation injuries, 684–685, 684f
peyote ingestion, 635, 635f
phacodonesis, 90
phalangeal dislocations, 310, 310f, 311f
phalanx fractures, 516f
pharyngitis, 117, 117f, 118f
pharyngoconjunctival fever, 33
phased array ultrasound transducer, 861t
phenylephrine, for episcleritis, 42
phimosis, 213, 213f
phlegmasia dolens, 362, 362f
photoallergic reactions, 544, 545f, 546f
photodermatoses, 544
photokeratitis, 544, 546f
photosensitivity reactions, 544, 544f, 545f, 546f
phototoxic reactions, 544
Phthirus corporis, 933f
Phthirus pubis, 236, 236f, 933f
pediatric, due to sexual abuse, 526f
phytophotodermatitis, 544, 545f, 546f
piercing complications, tongue, 163, 163f
pilocarpine, for acute angle-closure glaucoma, 43, 79
piloerection, with opioid withdrawal, 598f
pilonidal abscesses, 244, 244f
ping pong ball skull fracture, 8, 8f

pinguecula, 38, 38f
pinna bruise in child abuse, 511f
pinna, hematoma of, 19–20, 19f
pinworm infection, pediatric, 492, 492f
pit bull, mauling by, 549, 549f
pitting edema, 352, 352f
pit viper envenomation, 551, 551f, 552f, 553f
pityriasis rosea, 386, 386f, 387
pityriasis versicolor, 416, 416f
placenta, delivery of, 275, 276f
Plasmodium sp., malaria due to, 742–743
Plasmodium falciparum, Wright stain for, 935f
pneumococci, membranous tracheitis due to, 478
Pneumocystis jirovecii infection, in HIV patients, 699, 699f
pneumothorax, tension, 170, 170f
podagra, 348, 348f
podofilox, for condyloma acuminata, 234
poisoning. See toxicological conditions
poison ivy, oak, and sumac, 583, 583f, 584f, 585f
polymyxin, for conjunctivitis, 32
pompholyx, 391, 391f
popsicle panniculitis, pediatric, 453, 453f
porphyria cutanea tarda, 428, 428f
posthitis, 219
potassium hydroxide mount, 931, 931f
potassium iodide, for tropical sporotrichosis, 760
Potts disease, 761, 762f
Pott puffy tumor, 132, 133f, 134f
poxvirus, molluscum contagiosum due to, 710, 710f
praziquantel, for schistosomiasis, 753
preauricular sinus abscess, 107, 107f
prednisone
  for relapsing polychondritis, 109
  for Sweet syndrome, 427
pregnancy
  abdomen in, 189, 190f
  amniotic fluid and, ferning pattern of, 936, 936f
  ectopic, 266–268, 266f, 267f, 268f
  failed intrauterine, 270, 270f
  mask of, 421, 421f
  molar (hydatidiform mole), 269, 269f
  spontaneous abortion and, 254, 254f
  third trimester of, abdominal trauma during, 271, 271f, 272f
  ultrasound, 907–910, 907f–910f
preoxygenation, 772, 773f
prepatellar bursitis, 353, 353f
pressure-induced conditions
  mask squeeze, 569, 569f
  middle ear squeeze (barotitis media), 568, 568f
priapism, 214, 214f
primary ameba meningoencephalitis, 716
prolapse of iris, 94f
proliferative diabetic retinopathy, funduscopic appearance in, 72f
protein-energy malnutrition, 750–751, 750f, 751f
prune belly syndrome, 198, 198f, 199f
pseudogout, 348
  crystal-induced, 349f
Pseudomonas sp., otitis externa due to, 106
Pseudomonas aeruginosa
  auricular perichondritis due to, 109
  cellulitis due to, 342
  folliculitis, hot tub, due to, 410, 410f
  otitis externa due to, 106
Pseudomonas hot-foot syndrome, 410
pseudoobstruction, abdominal distention due to, 191f
psoriasis, 384, 384f, 385f
pterygium, 38, 38f
Pthirus pubis (crab louse), 493
pulmonary edema
  altitude-induced, 537, 537f
  noncardiogenic
    heroin-related, 597f
    opioid-induced, 598f
pulmonary embolism, electrocardiogram abnormalities in, 853, 853f
pulmonary histoplasmosis, 702, 702f
pulmonary tuberculosis, 761, 763f
“punched out” appearance, in acute necrotizing ulcerative gingivitis, 154, 154f
puncture wounds, 646, 647f. See also stab wounds
pupillary defect, afferent, 55, 55f, 56f
“pupil-sparing” in third-nerve palsy, 58, 59f
pupil, teardrop, with scleral rupture, 94, 94f
purpura
  Henoch-Schönlein. See Henoch-Schönlein purpura
  idiopathic thrombocytopenic, 398, 398f
  thrombotic thrombocytopenic, 399, 399f
puss caterpillar envenomation, 566, 567f
pustular psoriasis, 384
pustules, in gonorrhea, 227, 228f
pyloric stenosis, hypertrophic, 442, 442f, 443f
pyoderma gangrenosum, 382, 382f
pyogenic granuloma, 380, 380f, 381f

Q

QT interval prolongation, 823, 823f
quadriceps tendon rupture, 319, 319f
Quincke disease, 127, 127f
quinidine, for malaria, 742
quinsy, 123, 123f, 124f

R
rabies, 752, 752f
raccoon eyes, 4, 5f
radial head fractures, 290–291
radial head subluxation, pediatric, 486, 486f, 487f
radial nerve injuries, 304
radiculopathy, cervical, 360, 360f
radius, fractures of, distal, 296, 296f, 297f
Ramsay Hunt syndrome, 111, 111f, 112f, 413
ranula, 128, 128f
rape, 255–256, 255f, 256f
rapid-sequence induction, alternatives to, 769, 769f
rashes and lesions. See cutaneous conditions
rattlesnake envenomation, 551, 551f, 552f, 553f
Raynaud disease, 368, 368f
rectal conditions. See also anorectal conditions
  rectal foreign bodies, 245, 245f, 246f
  rectal prolapse, 243, 243f
  trauma, pediatric, 525f, 526f
rectocele, 264, 264f
rectus sheath hematomas, 192, 192f, 193f
red blood cell casts, 925f
red man syndrome, vancomycin-induced, 614, 614f
relapsing polychondritis, 110f
renal ultrasound. See ultrasound, of renal
respiratory retractions, 179, 179f
resuscitation, ultrasound, 897–898, 897f
retinal detachment, funduscopic findings with, 74, 74f
retinal edema, in central retinal vein occlusion, 69, 69f
retinal hemorrhage
  altitude-induced, 536, 536f
  funduscopic appearance of, 65, 65f
  in shaken baby syndrome, 508, 509f, 510f
retinitis
  cytomegalovirus
    funduscopic findings in, 75, 75f
    in HIV patients, 703, 703f
  Toxoplasma gondii, in HIV patients, 697, 698f
retinopathy
  diabetic, funduscopic findings in, 71, 71f, 72f
  hypertensive, funduscopic findings in, 70, 70f
retrobulbar hematoma, 23, 23f
retropharyngeal abscesses, pediatric, 475–476, 475f, 476f
rhabdomyolysis, cocaine-induced, 591, 592f, 593f
rib fractures, 515f
Rickettsia rickettsii, 408, 408f, 562
rifampin
  for leprosy, 739
  for meningococcemia, 455
right atrial hypertrophy, electrocardiogram abnormalities in, 845, 845f
right bundle branch block, electrocardiogram abnormalities in, 824, 824f
right ventricular hypertrophy, electrocardiogram abnormalities in, 843, 843f
ring injuries, 648f
ring tourniquets, 642f
ringworm. See tinea entries
Rocky Mountain spotted fever, 408, 408f, 562
rodenticide poisoning, 616–617, 616f, 617f
Rolando fractures, 305, 306f
roseola infantum, pediatric, 447, 447f
Roth spot, funduscopic findings with, 65, 65f
roundworms, 720, 720f
running wound closure, 659
"rust ring" with corneal foreign bodies, 85, 85f

S
saddle-nose deformity, 9, 10f
sail sign, 290, 290f
salicylate poisoning, 600–601, 600f, 601f
salmon patches, 437, 437f
Salter-Harris fracture classification, 480, 480f
"Saturday night palsy," 304
Scabies, 419, 419f
  adult mite, 933f
  in HIV patients, 708–709, 708f, 709f
  skin scraping from patient with, 933f
scald burn with sparing, due to child abuse, 507f
scald injuries, due to abuse, 686, 687f
scaphoid fracture, 301, 301f
scapholunate dissociations, 298, 300f
scarlet fever, pediatric, 459, 459f, 460f
schistocytes, testing for, 937, 937f
schistosomiasis, 753, 753f, 754f
scleral rupture, 94, 94f
scleritis, 41, 41f
scorpion envenomation, 560–561, 560f, 561f
scorpion fish envenomation, 578, 578f
scrofula, 761
scrotal bruises, 524f
scrotum
  Fournier gangrene of, 211, 211f, 212f
  straddle injury of, 218, 218f
  tinea cruris of, 414, 415f
scuba diving injuries
  mask squeeze, 569, 569f
  middle ear squeeze (barotitis media), 568, 568f
scyphozoan envenomation, 574, 575f
sea anemone envenomation, 574, 576f
"sea bather's eruption," 577, 577f
"seashore sign," 874f
sea snake envenomation, 580, 580f

seat belt injuries, 174, 174f
sea urchin envenomation, 572–573, 572f, 573f
seborrheic dermatitis, 383, 383f
Seidel test, 88, 88f, 94
Seldinger technique, 801–802, 801f, 802f
septal abscesses, 119, 119f
septal conditions, nasal, 119, 119f
septal hematoma, 9, 9f, 119, 119f
septal perforation, 119, 119f
septic abortion, 254
septic prepatellar bursitis, 353, 353f
septic thrombophlebitis, 357, 357f
serum sickness-like reaction, pediatric, 463, 463f
setting-sun phenomenon (sundowning), pediatric, 496, 496f
seventh-nerve palsy, 113–114, 113f, 114f
sexual abuse, of children, 519–533
  examination for, 519–520, 519f–523f
  injuries and findings due to, 524, 524f–526f
sexual assault, 255–256, 255f, 256f
sexually transmitted diseases, 223–247
  chancroid, 233, 233f
  chlamydia infection, 229, 229f
  condyloma acuminata, 234–235, 234f, 235f
  genital herpes, 230–231, 230f, 231f
  gonorrhea, 227–228, 227f, 228f. See also Neisseria gonorrhoeae
  lymphogranuloma venereum, 232, 232f
  pediatric, due to sexual abuse, 524, 524f–526f, 525f, 526f
  pediculosis, 236, 236f
  syphilis
    primary, 224, 224f
    secondary, 224–226, 225f, 226f, 526f
Sgarbossa criteria, in setting of left bundle branch block, 811, 811f
shaken baby syndrome, 508, 508f, 509f, 510f
sharp-force-pattern injuries, 679, 679f, 680f
shearing injuries, 646, 646f
shoulder(s)
  delivery of, 274–275, 274f, 275f
  dislocation of, 288–289, 288f, 289f
shoulder dystocia, 285, 285f
sialoadenitis, 129–130, 129f, 130f
sialolithiasis, 129, 130f, 131f
sickle dactylitis, acute, pediatric, 482, 482f
sinecatechin, for condyloma acuminata, 234
sinusitis, 132, 132f, 133f
Sister Mary Joseph node, 189, 189f
sixth-nerve palsy, 60, 60f
skeletal findings, physical child abuse, 514
skier's thumb, 309, 309f
skin scraping, for dermatoses and infestations, 933, 933f
skull fractures
  basilar, 4, 4f–6f, 5
  depressed, 7–8, 7f
  ping pong ball, 8, 8f
  in shaken baby syndrome, 509f
slap-mark contusion pattern, 682, 682f
Smith fractures, 296, 297f
snake bites
  coral snake, 550, 550f
  Fer-de-Lance, 554f
  pit viper, 551, 551f, 552f, 553f
  rattlesnake, 551, 551f, 552f, 553f
  sea snake, 580, 580f
  tropical snake, 554–555, 554f, 555f
snow blindness, 544
soaking of wounds, 640, 640f
soft tissue ultrasound, 920–921, 920f–922f
Solenopsis invicta envenomation, 564, 564f, 565f
solvent abuse, 608, 608f, 609f
southern tick associated rash illness, 562
spermatozoa, vaginal fluid wet mount demonstrating, 930, 930f
sphenoid sinusitis, 132
spider bites
  black widow spider, 557, 557f
  brown recluse spider, 558–559, 558f, 559f
spirochetes, due to acute necrotizing ulcerative gingivitis, 154
splash burns, due to child abuse, 506f
splenomegaly, in malaria, 743f
splinter hemorrhages, in infective endocarditis, 405, 406f
Sporothrix schenckii, sporotrichosis due to, 586, 586f
  tropical, 760, 760f
sprains. See specific sites
squamous cell carcinoma, 260, 260f, 429, 429f
squill ingestion, 631
stab wounds, 679, 679f, 680f
  to chest or abdomen, 175, 175f
staphylococcal scalded skin syndrome, pediatric, 458, 458f
Staphylococcus sp.
  epiglottitis (supraglottitis) due to, 473
  Ludwig angina due to, 152
  staphylococcal scalded skin syndrome due to, 458, 458f
Staphylococcus aureus
  auricular perichondritis due to, 109
  blistering distal dactylitis due to, 461
  bursitis due to, 353
  cellulitis due to, 342
  conjunctivitis due to, 30–31, 32
  dacryocystitis due to, 36
  epiglottitis due to, 125
  Gram stain of, 928, 928f
  hordeolum due to, 39, 39f, 40f

Staphylococcus aureus (Cont.):
impetigo due to, 448
infective endocarditis due to, 405
mastitis due to, in newborns, 440
membranous tracheitis due to, 478
nasal cellulitis due to, 120
necrotizing fasciitis due to, 346
orbital and periorbital cellulitis due to, 466
otitis externa due to, 106
palmar space infections due to, 354, 354f
pilonidal abscess due to, 244
retropharyngeal abscess due to, 475
septal abscesses due to, 119
sinusitis due to, 132
tenosynovitis due to, 355
thyroglossal duct cyst, pediatric, due to, 470
Staphylococcus epidermidis
dacryocystitis due to, 36
thyroglossal duct cyst, pediatric, due to, 470
staples, for wound closure, 658, 659f
stasis dermatitis, 393, 393f
sternal retractions, 179, 179f
sternoclavicular dislocation, 168, 168f, 169f
stethoscope airflow monitor, 798f
Stevens-Johnson syndrome, 372, 372f, 373f
sting(s). See envenomations
stingray envenomation, 570–571, 570f, 571f
stoma prolapse, 196, 196f
stomatitis
herpes simplex virus, 157, 157f
herpetic gingivostomatitis, pediatric, 454, 454f
stone fish envenomation, 578, 578f
stool examination, for fecal leukocytes, 932, 932f
Storz C-MAC video-assisted intubation, 785, 785f
straddle injuries, 218, 218f
pediatric, 527, 527f
strangulation injuries, 684–685, 684f, 685f
strawberry hemangioma, pediatric, 464, 464f, 465f
strawberry tongue, 159, 159f, 459
streptococcal infections
b-Hemolytic, epiglottitis due to, 125
group A
cellulitis, 342, 466
impetigo, 448
lymphangitis, 351
pharyngitis, 117, 117f, 118f
strawberry tongue associated with, 159, 159f, 459
group B, mastitis, in newborns, 440
membranous tracheitis due to, 478
perianal, pediatric, 532, 532f
uvulitis due to, 127
Streptococcus sp.
epiglottitis (supraglottitis) due to, 473
Ludwig angina due to, 152
orbital and periorbital cellulitis due to, 466
pilonidal abscess due to, 244
Streptococcus mutans, nursing bottle caries due to, 485
Streptococcus pneumoniae
conjunctivitis due to, 30–31, 32
epiglottitis due to, 125
Gram stain of, 928, 928f
otitis media due to, 100
septal abscesses due to, 119
sinusitis due to, 132
Streptococcus pyogenes
auricular perichondritis due to, 109
impetigo due to, 448
myonecrosis due to, 345, 345f
nasal cellulitis due to, 120
otitis media due to, 100
sinusitis due to, 132
streptomycin, for mycetoma, 745
striae atrophicae, 422, 422f
ST-T abnormalities. See electrocardiogram abnormalities, ST-T
stylets for endotracheal intubation
Eschmann, 781–783, 781f–783f
lighted, 789–790, 789f, 790f, 791t
shape of, 772–773, 773f
subarachnoid hemorrhage, subhyaloid hemorrhage in, funduscopic findings with, 80, 80f
subclavian vein thrombosis, 359, 359f
subconjunctival hemorrhage, 84, 84f, 166f
subdural hemorrhage, in shaken baby syndrome, 508, 508f, 509f
subendocardial ischemia, electrocardiogram abnormalities in, 812, 812f
subgaleal hemorrhage, in shaken baby syndrome, 510f
subhyaloid hemorrhage, in subarachnoid hemorrhage, funduscopic findings with, 80, 80f
sublingual hemorrhage, 17f
subluxation. See specific sites
subungual hematoma, 316, 316f
succimer
for arsenic poisoning, 622
for lead poisoning, 625
for mercury poisoning, 627
sulfacetamide, for bacterial conjunctivitis, 32
sulfamethoxazole
for blistering distal dactylitis, 461
for cellulitis, 342
for mycetoma, 745

summer penile syndrome, pediatric, 457, 457f
sundowning, pediatric, 496, 496f
sun exposure, 544, 544f, 545f, 546f
"sun poisoning," 544
superior vena cava syndrome, 180, 180f
superwarfarin poisoning, 616–617, 616f
supraclavicular adenopathy, 181, 181f
supracondylar fractures, 290, 290f, 291, 291f
supraglottitis, 125–126, 125f, 126f
  pediatric, 473, 473f, 474f
suprasternal retractions, 179, 179f
supraventricular tachycardia, 836, 836f
  with aberrancy, 837, 837f
suture materials, 660t
swan-neck deformity, 307, 307f
Sweet syndrome, 427, 427f
"swimmers' ear," 106, 106f
"swimmer's itch," 577, 577f
symblepharon, with caustic burns, 98, 98f
sympathetic ophthalmia, 47, 47f
synovial cysts, 367, 367f
synovial fluid analysis, 349t, 926, 926f, 927f
synovitis, crystal-induced, 348, 348f, 349f
syphilis
  primary, 224, 224f
  secondary, 224–226, 225f, 226f
    pediatric, due to sexual abuse, 526f
systemic lupus erythematosus, cutaneous conditions, 426, 426f

T
tachycardia, digitalis toxicity and, 631
tactile intubation, 792, 792f
Taenia solium, cysticercosis due to, 725
tape test for Enterobias vermicularis eggs, 938, 938f, 939f
tapeworms, 725–726, 725f, 726f
  hydatid cysts and, 734, 734f
Tardieu spots, 166, 166f
target sign in hypertrophic pyloric stenosis, 443f
tattooing, with gunshot wounds, 672–673, 673f
teardrop pupil, with scleral rupture, 94, 94f
teeth
  acid erosion of, 155, 155f
  avulsion of, 138, 138f
  fractures of, 139–140, 139f, 140f
  hand injuries caused by, 302, 302f
  impaction (intrusive luxation) of, 137, 137f
  methamphetamine and, 588, 589f
  nursing bottle caries and, 485, 485f
  subluxation of, 136, 136f
temporomandibular joint dislocation, traumatic, 142–143, 142f, 143f
tendons. See also specific tendons
  lacerations of, 652, 652f, 653f
tenosynovitis, 355, 355f
tension injuries, 646, 647f
tension pneumothorax, 170, 170f
terbinafine
  for dermatophyte (tinea) infections, 711
  for tropical sporotrichosis, 760
testicular appendix, torsion of, 204, 204f
testicular conditions
  acute epididymitis, 205, 205f
  appendiceal torsion, 204, 204f
  hydrocele, 208, 208f
  orchitis, 206–207, 206f, 207f
  torsion, 202, 202f, 203f
  tumors, 209, 209f
tetanus, 755, 755f, 756f
  prophylaxis of, 646, 649t
tetracycline
  for syphilis, 224
  for trachoma, 757
thenar space infections, 354, 354f
thermal pattern injuries, 686, 686f, 687f
thiabendazole, for cutaneous larva migrans, 724
third-degree AV block, electrocardiogram abnormalities in, 822, 822f
third-nerve palsy, 58, 59f
Thompson test, 327, 327f
thoracentesis
  needle, tension pneumothorax with, 170, 170f
  ultrasound, 914–915, 914f
thoracotomy, 172, 172f
threatened abortion, 254
throat conditions. See ear, nose, and throat conditions; specific conditions
thrombocytopenia, in HIV patients, 712, 712f
thrombophlebitis, 357, 357f
thrombosis
  deep venous, 363, 363f, 364f
  of subclavian vein, 359, 359f
thrombotic thrombocytopenic purpura, 399, 399f
thrush, 156, 156f
"thumb" sign, in epiglottitis, 125, 125f
thyroglossal duct cyst, pediatric, 470, 470f
tibial fractures, 324, 324f, 325f
tick bites, 562, 562f, 563f
tick envenomation, 562
timolol maleate, for acute angle-closure glaucoma, 43, 79
tinea capitis, 392, 414, 415f, 711
tinea corporis, 414, 414f, 709, 711, 711f
tinea cruris, 414, 415f
tinea faciale, 414, 414f

tinea infections, in HIV patients, 711, 711f
tinea manuum, 414, 415f
tinea pedis, 392, 392f, 414, 415f, 711
tinea unguium, 414
tinea versicolor, 416, 416f
tire mark contusion pattern, 683f
Tityus envenomation, 560
tobacco use, tooth erosion due to, 155, 155f
tobramycin, for conjunctivitis, neonatal, 31t
toddler's fractures, 481, 481f, 514, 516f
toenails
  ingrown, 350, 350f
  tinea unguium of, 414
toilet seat injuries, 528, 528f
tongue
  black hairy, 162, 162f
  laceration of, 144, 144f
  piercing complications of, 163, 163f
  strawberry, 159, 159f, 459
tooth. See teeth
tophus, 348
torsades de pointes, 840, 840f
torus palatinus, 160, 160f
tourniquet, penile, 217, 217f
toxalbumin ingestion, 636, 636f
toxic epidermal necrolysis, 372, 372f, 373f
Toxicodendron exposure, 583, 583f, 584f, 585f
toxicological conditions, 587–638
  acetaminophen poisoning, 599, 599f
  amphetamine toxicity, 588, 588f, 589f
  anticholinergic (antimuscarinic) toxicity, 594, 594f
  anticoagulants, 616–617, 616f, 617f
  arsenic poisoning, 622, 622f, 623f
  bath salts, 590, 590f
  b-Blocker poisoning, 606, 606f
  botulism, 615, 615f
  calcium-channel blocker poisoning, 606, 607f
  cannabinoids, 590, 590f
  cardiac glycoside plant ingestion, 631, 631f, 632f
  Catha edulis (khat), 590, 590f
  caustic ingestion, 618, 618f, 619f
  cellular asphyxiants, 612–613, 612f, 613f
  cholinergic toxicity, 595, 595f, 596f
  cocaine toxicity, 591, 591f–593f, 593
  digitalis toxicity, 631, 631f
  houseplants with calcium oxalate crystals, 633, 633f
  hydrofluoric acid burns, 620, 621f
  inhalant abuse, 608, 608f, 609f
  iron poisoning, 624, 624f
  jimsonweed ingestion, 534f, 634
  lead poisoning, 625, 625f, 626f
  levamisole-induced vasculitis, 637–638, 637f, 638f
  mercury poisoning, 627–628, 627f, 628f
  methemoglobinemia, 610, 610f, 611f
  mushroom ingestion, 629, 629f, 630f
  oleander ingestion, 631, 631f
  peyote ingestion, 635, 635f
  salicylate poisoning, 600–601, 600f, 601f
  toxalbumin ingestion, 636, 636f
  toxic alcohol ingestion, 602–603, 602f, 603f
  tricyclic antidepressant toxicity, 604, 604f, 605f
  vancomycin-induced red man syndrome, 614, 614f
toxic shock syndrome, 159, 459, 460f
Toxoplasma gondii infection, in HIV patients, 697, 697f, 698f
tracheitis, membranous (bacterial), pediatric, 478, 478f
trachoma, 757, 757f
traditional tropical medicine, 758–759, 758f, 759f
transabdominal ultrasound, 907–910, 907f, 908f
transducers, for ultrasound, 860, 862f
  of abdominal aorta, 890
  of deep venous thrombosis, 894
  of lung, 887
  of ocular, 916
  pelvic, 907
  renal, 904
  for trauma, 863
transvaginal ultrasound, 908, 908f–910f
trauma. See also ultrasound, for trauma; specific types of trauma and specific injuries
  wounds caused by, 646–647. See also specific types of wounds
    crush or compression injuries, 646, 649f
    shearing injuries, 646, 646f
    tension or partial avulsion injuries, 646, 647f
    tetanus prophylaxis for, 646, 649t
traumatic enucleation, 24f
trench foot, 543, 543f
trench mouth, 154, 154f
Treponema pallidum, syphilis due to, 224–226
Trichomonas
  sexually transmitted diseases, pediatric, 524
  vaginal fluid wet mount demonstrating, 930, 930f
  vaginitis due to, 250, 250f, 251
trichotillomania, pediatric, 494, 494f
tricyclic antidepressants
  electrocardiogram abnormalities and, 856, 856f
  poisoning by, 604, 604f, 605f
trifluorothymidine, for ocular herpes simplex, 51
trifluridine
  for conjunctivitis, neonatal, 30, 31t
  for ocular herpes simplex, 51
trimethoprim
  for blistering distal dactylitis, 461
  for cellulitis, 342

for conjunctivitis, 32
for mycetoma, 745
trimethoprim-sulfamethoxazole, for nasal cellulitis, 120
Trinder reagent, 601f
tripod fractures, 11, 11f
tropical medicine, 715–764
anemia, 717, 717f
ascariasis, 720, 720f
bacillus anthracis (anthrax), 718, 718f, 719f
Chagas disease, 721, 721f
cholera, 722–723, 722f, 723f
cutaneous larva migrans, 724, 724f
cysticercosis, 725–726, 725f, 726f
dengue fever, 727–728, 727f, 728f
dracunculiasis, 729, 729f
elephantiasis, 730–731, 730f, 731f
epidemic meningitis, 732, 732f
free-living ameba infection, 716, 716f
goiter, 733, 733f
hydatid cyst, 734, 734f
leech bites, 735, 735f, 736f
leishmaniasis, 737–738, 737f, 738f
leprosy, 739–740, 739f, 740f
leptospirosis, 741, 741f
malaria, 742–743, 742f, 743f
mumps, 744, 744f
mycetoma, 745, 745f, 746f
myiasis, 747, 747f, 748f
nontuberculous mycobacteria infections, 749, 749f
protein-energy malnutrition, 750–751, 750f, 751f
rabies, 752, 752f
schistosomiasis, 753, 753f, 754f
tetanus, 755, 755f, 756f
trachoma, 757, 757f
traditional medicine and, 758–759, 758f, 759f
tropical sporotrichosis, 760, 760f
tuberculosis, 761, 761f–763f
tropical sporotrichosis, 760, 760f
Trypanosoma cruzi, Chagas disease due to, 721
tuberculosis, 761, 761f–763f
tumbu fly, myiasis due to, 747, 748f
tumors
apical, of lung, 181, 181f
basal cell carcinoma, 430, 430f
squamous cell carcinoma, 429, 429f
testicular/urological, 209, 209f
vulvar, 260, 260f
Tunga penetrans, tungiasis due to, 764, 764f
tympanic membrane
in bullous myringitis, 103f
cholesteatoma and, 104, 104f
middle ear squeeze and, 568, 568f
normal, 100, 100f
in otitis media, 100, 100f, 102f
perforation of, 105, 105f
tympanostomy tubes, 101, 102f

U

ulcer(s)
corneal, 52, 52f–54f
decubitus, treatment of, 670, 670f
oral, aphthous, 158, 158f
ulcerative gingivitis, necrotizing, acute, 154, 154f
in HIV patients, 714, 714f
ulcerative keratitis, 52, 52f–54f
ulnar nerve injuries, 304, 304f
ultrasound, 859–922
abdominal, 899f–902f
of abdominal aorta, 890–892
bladder, 903–905, 903f–906f
of gallbladder, 899–901, 899f–902f
pelvic, 907–910
renal, 903–905, 903f–906f
of abdominal aorta, 890–892
abnormal findings by, 892, 892f, 893f
clinical indications for, 890
required views for, 890
sagittal view for, 890, 891f
transducers for, 890
transverse view for, 890, 890f, 891f
of abdominal distention, 189f
antral nipple sign in hypertrophic pyloric stenosis, 443f
of bladder, 903–905, 903f–906f
of cardiac, 876–886, 876f
apical four-chamber view for, 883, 883f, 884f
indications for, 876
parasternal long-axis view for, 879–880, 879f, 880f
parasternal short-axis view for, 881, 881f, 882f
subxiphoid-cardiac (subcostal) view for, 877, 877f, 878f
subxiphoid long axis view for, 885, 885f, 886f
tamponade, 172f
views for, 876
of deep venous thrombosis, 894–896, 894f–896f
of ectopic pregnancy, 266–268, 266f, 267f, 268f
of gallbladder, 899–901
abnormal findings of, 900–901, 901f, 902f
sagittal view for, 899, 899f
transverse view for, 899, 900f
guided resuscitation, 897–898, 897f, 898t
key terms, 860
of lung, 887–889, 887f–889f
of ocular, 916–918, 916f–919f
paracentesis, 914–915
of peritonsillar abscess, 123f

ultrasound (Cont.):
of pregnancy, 907–910, 907f–910f
of renal, 903–905, 903f–906f
resuscitation, 897–898, 897f
soft tissue, 920–921, 920f–922f
target sign in hypertrophic pyloric stenosis, 443f
thoracentesis, 914–915
transabdominal, 907–910, 907f, 908f
transducers for. See transducers, for ultrasound
transvaginal, 908, 908f–910f
for trauma
E-FAST series for, 863–875, 863f
FAST series for, 863
indications for, 863
left upper quadrant (perisplenic view), 868–869, 868f, 869f
pericardial (subxiphoid view), 864–865, 864f, 865f
right upper quadrant (Morison pouch) view, 866, 866f, 867f
suprapubic view, 870–872, 870f, 871f, 872f
thorax (right and left) views, 873–875, 873f, 874f, 875f
transducers for, 863
views required for, 863
venous access, 911–913, 911f–913f
ultraviolet keratitis, 544, 546f
umbilical cord
clamping of, 274, 275f
granuloma, 441, 441f
prolapse of, 278, 278f
umbilicus, nodular, 188, 188f
uncal herniation syndrome, 26, 26f, 27f
upper extremity trauma, 285–316, 339f
acromioclavicular joint separation, 286, 286f, 287f
amputations, 339f
Bennett fracture, 305, 305f, 306f
biceps tendon rupture, 293, 293f
Boutonnière deformity, 307, 307f
boxer's fracture, 303, 303f
carpal and carpometacarpal dislocations, 298, 298f–300f
clenched fist injury, 302, 302f
compartment syndrome, 314, 314f–315f
elbow dislocation, 292, 292f
elbow fractures, 290–291, 290f, 291f
electrical injury, 668, 668f, 669f
forearm fractures, 294, 294f, 295f
gamekeeper's thumb, 309, 309f
high-pressure injection injuries, 308, 308f
Jersey finger, 312–313, 313f
mallet finger, 312–313, 312f, 313f
peripheral nerve injury, 304, 304f
phalangeal dislocations, 310, 310f, 311f
radial fractures, distal, 296, 296f, 297f
Rolando fracture, 305, 306f
shoulder dislocation, 288–289, 288f, 289f
skier's thumb, 309, 309f
subungual hematoma, 316, 316f
swan-neck deformity, 307, 307f
urachal duct, patent, 187, 187f
uremic frost, 424, 424f
urethra
prolapse of, 261, 261f
pediatric, 529, 529f
rupture of, 215, 215f
urethritis
chlamydial, 229, 229f
in gonorrhea, 227, 227f
uric acid crystals
in synovial fluid, 926, 926f
in urine, 924, 924f
urinalysis, microscopic, 924, 924f, 925f
urologic conditions, 201–222. See also specific conditions
Fournier gangrene, 211, 211f, 212f
hydrocele, 208, 208f
penile. See penile conditions
scrotal abscess, 210, 210f
straddle injury, 218, 218f
pediatric, 527, 527f
testicular. See testicular conditions
urticaria, 400, 400f, 401f
pediatric, 456, 456f
vasculitis, 396, 397f
uterine prolapse, 262, 262f
uveitis, anterior, 45, 45f, 46f
uvula, visualization during laryngoscopy, 776f
uvulitis, 127, 127f

V

vaginal discharge, pediatric, due to sexual abuse, 525f
vaginal fluid wet mount, 930, 930f
valacyclovir
for herpes simplex virus infections, genital, 230–231
for herpes zoster infections, 704
vancomycin
for dacryocystitis, 36
for nasal cellulitis, 120
red man syndrome induced by, 614, 614f
vapotherm, 766, 766f
varicella, pediatric, 450, 450f
varicocele, 222, 222f
vasculitis
leukocytoclastic, 396, 396f, 397f
levamisole-induced, 637–638, 637f, 638f
urticarial, 396, 397f
venous access ultrasound, 911–913, 911f–913f

venous pooling, pediatric, 533, 533f
ventricular flutter, 839, 839f
ventricular rhythm, 831, 831f
ventricular tachycardia, 838, 838f
vermilion border of lips, laceration of, 145, 145f
vernal conjunctivitis, 35, 35f
vertebral fractures, compression, 515f
vertical mattress suture, 661, 661f
Vibrio cholerae, 722
vidarabine
  for conjunctivitis, neonatal, 31t
  for ocular herpes simplex, 51
video-assisted intubation
  Glidescope, 784, 784f
  McGrath MAC, 786, 786f
  Storz C-MAC, 785, 785f
violence. See child abuse; domestic violence
viral conjunctivitis, 33, 33f
  herpes simplex virus, 50
visceral findings, physical child abuse, 517
vitiligo, 423, 423f
vitreous hemorrhage, funduscopic findings with, 73, 73f
vocal cords, visualization during laryngoscopy, 777f
volar plate injuries, 310, 311f
Volkmann ischemic contracture, in compartment syndrome, 314, 315f
volvulus, intestinal malrotation, 444, 444f
vomiting, in bulimia, acid tooth erosion in, 155, 155f
vulvar tumor, 260, 260f

W

wallflower ingestion, 631
warfarin poisoning, 616–617, 616f
warts
  genital, 234–235, 234f, 235f
  giant, 235f
wasp envenomation, 564, 564f
Wellens waves, 814, 814f
Wenckebach second-degree AV block, electrocardiogram abnormalities in, 820, 820f
wet gangrene, 344, 344f
white blood cell casts, in urine, 924, 924f
"widowmaker lesion," 810, 810f
witch's milk, 439, 439f
Wolff-Parkinson-White syndrome, electrocardiogram abnormalities in, 848, 848f
wood maneuver, 285
wood ticks, 562, 562f
woolly slug envenomation, 566, 567f
wound(s)
  assessment of, 642, 642f
  cleaning and irrigation of, 640–641, 640f, 641f
  closure of
    complex, 661, 661f–663f
    simple, 658–659, 658f–660f, 660t
  complications of, 664–665, 664f
  foreign bodies in, 643f, 644, 644f–645f
  traumatic, 646–647. See also specific types of wounds
    crush or compression injuries, 646, 649f
    shearing injuries, 646, 646f
    tension or partial avulsion injuries, 646, 647f
    tetanus prophylaxis for, 646, 649t
wound adhesives, 659, 660f
wound botulism, 615, 615f
wound dehiscence, 664f, 665
wound myiasis, 664
Wright stain for malaria, 935, 935f
Wuchereria bancrofti, elephantiasis due to, 730

X

xerostomia, jimsonweed-induced, 634

Z

zipper injury, penile, 221, 221f
zygoma fractures, 11, 11f